CHAPTER SIX

Dimensions of plant disease in tropical forests

GREGORY S. GILBERT
University of California, Santa Cruz, Smithsonian Tropical Research Institute, Balboa

Introduction

Pest pressure is the inevitable, ubiquitous factor in evolution which makes for an apparently pointless multiplicity of species in all areas in which it has time to operate.
(Gillett 1962)

At a symposium 44 years ago, J. B. Gillett proposed the Theory of Pest Pressure, whereby plant pathogens and pests were responsible for the genesis and maintenance of high plant diversity in tropical forests and other high-diversity systems. In his conclusion to the paper produced from that talk he hoped that this 'new theory may be useful in stimulating discussion and research' on the roles of pests and pathogens as a force in plant diversity. Apparently his theory has been useful, as much has happened in the last four decades to explore and expand on his idea. My aim is to review key research on the effects of plant pathogens in tropical forests since Gillett's seminal paper with emphasis on the special case of his Theory of Pest Pressure known as the Janzen–Connell Hypothesis. I will also suggest critical areas that need to be explored as we continue to discuss and research the role of pathogens in the maintenance of species diversity in tropical forests.

'*Parcere subiectis et debellare superbos*'

Gillett (1962) paraphrased Virgil's famous formula for the greatness of Rome, 'Spare the lowly and conquer the haughty' (*Aeneid* **VI**: 853), when he first introduced the idea that if plant pests have a greater impact on more common species than on rare species, the rare species should then increase in relative frequency, providing a mechanism for the maintenance of diversity in species-rich communities. Gillett's proposal is essentially that plant pests prevent competitive exclusion through density-dependent disease or herbivory. The competitive exclusion principle predicts that in a heterogeneous environment, one species will exclude all others (Gause 1934). Species can coexist by partitioning resources

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(Tilman 1982), but since plants generally have similar requirements for the same few limiting resources (e.g. light, water, N, P, K), Gillett wondered 'can it be seriously suggested that a rather uniform area of Amazonian rain forest provides, in 3.5 hectares of land, anything like 179 separate ecological niches for trees?' (Gillett 1962). Density-dependent mortality caused by host-specific pests and pathogens got the lead role in Gillett's 'Theory of Pest Pressure' for the maintenance of diversity in plant communities. A decade later D. H. Janzen and J. H. Connell (Janzen 1970; Connell 1971) further explored the spatial component of pest impacts on seedling shadows to popularize some of the ideas in Theory of Pest Pressure in what is now known as the Janzen–Connell Hypothesis.

The Janzen–Connell Hypothesis – a special case

> It will be seen that this invasiveness of taxa, due to pest pressure, applies as well to the spread of species from one plant association to another . . . When we see that by moving from one formation to the other it escapes to a great extent from its own pests, while entering into competition with plants which are still burdened down by theirs, the phenomenon is easy to understand. (Gillett 1962)

The Janzen–Connell Hypothesis (a special case of the Theory of Pest Pressure) begins by recognizing that the fruits of most tree species in tropical forests do not, on average, disperse very far, leading to a great density of conspecific seedlings close to the mother tree. Within the seedling shadow, the high density of that plant species may exclude the growth of other species. Pathogens or other pests that are either transmitted from the mother tree or that develop in a density-dependent way on the locally dominant species can make scarce water, light or nutrients available to rare, but pest-resistant plant species. Similarly, escaping pest pressure may provide a competitive advantage to those rare seeds that disperse out of their maternal neighbourhood and into the neighbourhood of a heterospecific mother tree where the dominant species of seedlings will be burdened down by its own pathogens. In this way, pathogens or other pests provide an advantage to the 'lowly' rare (and resistant) species over the 'haughty' (but susceptible) dominants, helping to maintain local tree species diversity.

Density-dependent mortality caused by host-specific pathogens and pests is at the heart of the Theory of Pest Pressure. There should be strong selection pressure for seeds to disperse out of their maternal neighbourhood (where they are disadvantaged by local pathogens) into the 'disease neighbourhood' of a heterospecific mother tree. The Janzen–Connell Hypothesis leads to several clear predictions of the Theory: (1) disease incidence should be greater at higher host density and/or among juveniles close to the mother tree, (2) through non-random mortality, the spatial distribution of the host species will become less clumped through time, (3) there should be greater recruitment of non-susceptible

species within the mother's disease neighbourhood, and (4) over time, plant diversity should be greater than expected compared with random survival of seeds.

From a collection of studies on Barro Colorado Island in Panama, we have significant empirical evidence to support the first of these predictions, that disease development should be greatest at high host density and/or among juveniles close to the maternal tree. Damping-off diseases are density- and/or distance-dependent for seedlings of a range of host species (Augspurger 1983a, b; Augspurger & Kelly 1984; Augspurger 1984, 1990; Davidson 2000). Similarly, canker diseases have density- or distance-dependent rates of infection on saplings of susceptible species (Gilbert *et al.* 1994; Gilbert & de Steven 1996). As a general principle for both agronomic and wild systems, nearly all fungal diseases except heteroecious rusts show density-dependent development (see reviews in Burdon & Chilvers 1982; Gilbert 2002). As such, density-dependent disease development, at least for fungal and oomycete-caused diseases, is probably the rule in both tropical and temperate systems, but there are exceptions. Davidson (2000) found that for two pathogens of *Anacardium* seedlings, *Phytophthora heveae* showed density-dependent effects on the seedling populations whereas the related *Pythium* spp. did not. Pathogens have as much life-history variation as any other group of organisms in a tropical forests; not all pathogens will cause density-dependent mortality on all hosts, although most will. Those that do not will be unlikely to contribute to Janzen–Connell effects.

Similarly, several studies have demonstrated that diseases can mediate shifts in the spatial pattern of host trees so that they become less clumped over time, as predicted by Janzen (1970) and Connell (1971). Once again, damping-off and canker diseases provide the classic examples. The median distance from seedlings of *Platypodium elegans* to the mother tree shifted from 15 m to 30 m, following non-random mortality caused by damping-off pathogens (Augspurger 1983a). Even among decades-old juveniles, Phytophthora canker of *Ocotea whitei* caused a significant shift in distribution of survivors away from conspecific adults (Gilbert *et al.* 1994).

To date, however, no studies in tropical forests have provided direct evidence for the last two predictions – that disease-related density-dependent mortality of one plant contributes to the greater survival of heterospecific, non-susceptible hosts, leading to greater local plant diversity than expected. The most complete demonstration of a disease fulfilling the diversity-maintaining Janzen–Connell predictions is that of damping-off caused by *Pythium* in a temperate forest in the mid-western United States Packer & Clay (2000). There, *Pythium* causes density- and distance-dependent mortality of wild cherry (*Prunus serotina*) seedlings around maternal trees, causing a reduction in clumping over time, and allowing a greater survival of rarer but less susceptible tree species in the area.

These case studies clearly show the potential for pathogens to reduce the local dominance of tree species and permit greater establishment of rare species (see also review of impacts of herbivores in Clark & Clark 1984). Collectively, they demonstrate that pathogens can (1) cause density- or distance-dependent mortality of the locally dominant tree species, (2) leading to a shift in density over time away from the maternal tree, while (3) allowing greater survival of less common, resistant species, and (4) ultimately enhancing local tree diversity, fulfilling the specific predictions of the Janzen–Connell version of the Theory of Pest Pressure. What is not yet clear, however, is whether such impacts are common enough to have a significant impact on diversity of tropical forests.

For plant diseases to be useful in explaining the maintenance of tree diversity in tropical forests through the mechanisms proposed in the Janzen–Connell Hypothesis and the Theory of Pest Pressure, several additional requirements must be met. For disease to be a driving force, (1) pathogens must be the 'inevitable, ubiquitous factors' suggested by Gillett (1962), (2) their impacts must be significant, but not devastating, (3) there must be adequate host specificity among the pathogens to create different selective pressures on different host species, (4) dominant plant species must be affected more strongly, and (5) their effects must be variable in space or time. I will discuss each of these in turn.

'The inevitable, ubiquitous factor'

For pathogens to be important drivers in tropical tree diversity, plant disease must be common and widespread in tropical forests. Clearly, if pathogens caused disease only rarely and on few plant species, they might be important components of the life history of individual species, but would be unlikely to be important factors in maintaining overall tree species diversity. Gillett's (1962) claim of the ubiquity of plant diseases came at a time when there were very few studies on plant pathogens in tropical forests beyond a handful of economically important diseases. In developing his theory he consciously followed Darwin's lead and relied on case studies and agricultural analogies to propose general principles. However, in assessing the relevance of the Theory of Pest Pressure to real tropical forest diversity we must recognize that real plant pathogens come in many forms. The most familiar are foliar pathogens that cause necrosis, chlorosis or deformation of leaves. Pathogens can also kill seeds and seedlings, plug the vascular system, cause cankers on stems and trunks, decay wood in roots and trunks, and attack flowers and developing fruits – often with potentially significant impacts on the host populations in natural ecosystems (see recent review in Gilbert 2002). The life histories, diversities and potential impacts of each of these groups of plant pathogens vary widely, and with potentially large consequences for their roles in maintenance of tropical tree species diversity. If we are to understand the role of pathogens, it is crucial that they not be lumped into a single black box. We would not blithely consider harpy eagles and jaguars

to be ecological equivalents in a tropical forest simply because they are both predators, and similarly, we stand to learn much more about the impacts of pathogens by considering the kinds of interactions particular pathogens have with their hosts.

Studies of individual, charismatic plant diseases can provide useful case studies of the potential effects of diseases, but to assess Gillett's claim of ubiquity of diseases we need to examine comparative studies across a range of plant species within a forest. Selecting study species because they have obvious diseases leads to a potentially misleading form of 'publication bias'. Much more useful are comparative studies across host species chosen either arbitrarily or for their ecological significance. Fortunately, there are now a few such quantitative, comparative studies for some kinds of plant pathogens in tropical forests, which allow us to begin to assess whether plant pathogens really are Gillett's inevitable and ubiquitous selective agents. I will review such studies grouped by broad life-history strategies of the pathogens.

Foliar diseases

Foliar diseases, which reduce the photosynthetic area available to plants, are the most widely studied diseases in tropical forests. In the most detailed comparative survey to date, García-Guzmán and Dirzo (2001) surveyed understorey plant species in the Los Tuxtlas tropical rain forest in Mexico and found that 69% of 57 understorey plant species, and 45% of all examined leaves, were damaged by fungal pathogens. Similarly, all 10 species examined on Barro Colorado Island (BCI) in Panama (Barone 1998), three species examined in Brazilian rain forest (Benitez-Malvido *et al.* 1999), all five species examined in a seasonal dry forest in Panama (Gilbert 1995), and all three species present in a Panamanian mangrove forest (Gilbert *et al.* 2001) suffered foliar diseases. However, disease incidence (proportion of leaves with at least some disease) varied widely across species (10% to 32%; Benitez-Malvido *et al.* 1999). From these studies, which included both high- and low-diversity tropical forests and both moist and dry sites, it is clear that most tropical tree species suffer from foliar diseases but that the incidence of disease varies widely across species.

Seed and seedling diseases

Diseases of seeds and seedlings have a major impact on individual plants, usually leading to a quick death. Fungal attack caused 47% and 39% annual mortality of seeds in the soil seed bank of *Miconia argentea* (Melastomataceae) and *Cecropia insignis* (Moraceae), two pioneer tree species in Panama (Dalling *et al.* 1998), and based on studies in temperate systems, I would expect pathogenic fungi to have widespread effects on other tropical species with seed banks. Seedlings are probably the most vulnerable stage in the life of a tropical tree, and a number of damping-off pathogens (often the fungus-like Oomycetes *Phytophthora*

and *Pythium*) cause dramatic losses of seedlings. In pioneering studies of the impacts of pathogens on the survival of tropical forest plants, Augspurger and coworkers, working on BCI, found that damping-off affected 80% of all tested species, and was the leading cause of death for seedlings of six of nine focal species (Augspurger 1983b; Augspurger & Kelly 1984; Augspurger 1984; Kitajima & Augspurger 1989).

Cankers, wilts and diebacks

Wilt and canker diseases affect the growth and survival of juvenile and adult woody plants, but have been little studied in tropical forests. The responsible pathogens use a variety of mechanisms to disrupt the plant's vascular system. On BCI, a canker associated with *Phytophthora* sp. affects 9 of 10 species of Lauraceae, including 73% of the individuals of *Ocotea whitei* (Gilbert *et al.* (1994), and G. S. Gilbert, unpublished data). Other case studies indicate that canker, wilt and dieback diseases can have significant effects on particular host species (Botryosphaeria canker on *Tetragastris panamensis* (Burseraceae), Gilbert and de Steven (1996); Fusarium wilt on the Hawaiian endemic *Acacia koa* (Fabaceae), Anderson *et al.* (2001)), but there have been no comparative studies across a diverse range of host species or in different forests to assess how widespread and common these pathogens are in tropical forests.

Wood-decay fungi

Wood-decay fungi, particularly the Basidiomycete polypore fungi, play a variety of roles in tropical forests. Some can be aggressive pathogens, destroying root systems or cambial tissue and killing trees directly, whereas others consume only dead wood, hollowing out the centres of living trees and making them more susceptible to damage from wind and rain. Still others may infect living trees, but really only colonize and decay extensively when the tree is dead or dying from other factors. Finally, some polypore fungi may play several of these roles sequentially. Recent studies provide some assessment of the ubiquity of wood-decay fungi not only on fallen logs but on standing trees in tropical forests, although the studies do not consistently differentiate among different ecological roles. For instance, in a survey of 10 focal tree species on BCI, the percentage of live trees with polypore fruiting bodies ranges from zero to 33% (with a mean of 7%), while 56% (ranging from 38% to 93%) of all dead trees had polypores (Gilbert *et al.* 2002). Comparable systematic study of macroscopic Ascomycete wood decay fungi has been rare in tropical systems, but for three tree species at the same site, ascomycete fruiting bodies were about two-thirds as common as polypore fungi (Ferrer & Gilbert 2003). In a Caribbean mangrove forest, 3% to 19% of all live and dead trunks of the three dominant tree species had fruiting bodies of polypore fungi (Gilbert & Sousa 2002). Most, if not all, tropical trees

have associated wood-decay polypore fungi, and for some tree species, a high proportion of live hosts may be attacked.

Flower and fruit diseases

Although any pathogens that affect the growth or survival of their host plants can reduce the host's reproductive output, a number of pathogens attack flowers and developing fruits directly. Very little work and no comparative studies have been done in tropical forests on such diseases. In one example, the rust *Aecidium farameae* attacks developing ovaries of the understorey treelet *Faramea occidentalis* (Rubiaceae) on BCI, but over the last decade has only appeared in outbreaks every several years (Travers *et al.* 1998). Of course, fungal colonization of seed may also be an important mechanism for vertical transmission of endophytes or pathogens (Bayman *et al.* 1998), so a clear picture of the frequency and ecological role of fruit- and seed-infecting fungi may be complex.

Missing diseases

García-Guzmán and Dirzo (2001) note specifically that they found no rusts, smuts or powdery mildews in their survey of plant diseases at Los Tuxtlas, and this corresponds well with my experiences elsewhere. Rusts have been known to cause problems in tropical tree plantations (Lee 1999), and the introduction of coffee rust (*Hemileia vastatrix*) has had devastating effects in tropical America (Wellman & Echandi 1981). The importance of rusts in many wild temperate systems (Bella & Navratil 1988; Burdon & Jarosz 1991; Davelos *et al.* 1996) leads us to wonder about their importance in the tropics. Nevertheless, there are few examples of rusts in tropical forests (Arthur & Cummings 1933; Gardner 1994; Chen *et al.* 1996). Smuts as well can have large impacts on natural plant populations in temperate systems (Alexander & Antonovics 1988; García-Guzmán *et al.* 1996; Carlsson-Graner 1997). However, although smuts have been recorded on tropical trees in the Sterculiaceae, Araliaceae, Tiliaceae and Piperaceae, only 11 (of 1450) smut species are known from woody plants and their overall importance to tropical trees is probably minimal (Vánky 2002). Powdery mildews cause serious problems in tropical agriculture including on fruit trees (Schoeman *et al.* 1995), but I am unaware of any studies of their incidence or importance in tropical forests. Other 'missing' plant pathogens such as viruses and phytoplasmas are of great importance in tropical agriculture, but remain essentially undocumented in tropical forests owing to the inherent technical difficulties in their detection and study. Whether the lack of records of rusts, smuts and powdery mildews reflects a similar blind spot among those working in tropical forests, the paucity of researchers in this area or a biological reality is not yet clear. However, all these missing pathogens are obligate biotrophs; that is, they complete their life cycles only on living hosts, for the most part cannot grow apart from a living plant, and in many cases show strong host specificity. It is possible that

conditions in diverse tropical forests are not supportive of such specialized pathogens, a possibility I will return to later.

Endophytic and epifoliar fungi

Two groups of plant-infecting fungi are often excluded from discussion of diseases, but they share so many life-history traits with other pathogens, and the potential for interaction with other pathogens is so great, that they must be included here. Leaf endophytes are fungi that invade living plant tissue and cause unapparent and asymptomatic infections within healthy plant tissues (Wilson 1995). Endophytes include mutualists and commensals, but many endophytes are latent pathogens that may cause disease at a later time depending on environmental conditions or plant stress. For instance, *Pestalotiopsis* (=*Pestalotia*) *subcuticularis* on *Hymenaea courbaril* (Fabaceae) stays as a benign endophyte unless leaves are wounded by cutting or scraping (Fail & Langenheim 1990). Recent studies clearly demonstrate the ubiquity of endophytes in tropical forests; generally 90–100% of all leaves are infected by endophytic fungi (Lodge *et al.* 1996; Bayman *et al.* 1998; Arnold *et al.* 2000; Bethancourt 2000; Arnold *et al.* 2001; Gilbert *et al.* 2001). On a finer scale, the area of individual leaves that is infected varies widely, from 98% of all 2-mm^2 leaf fragments from plants in nine families on BCI (Arnold *et al.* 2001) to only 25% of leaf discs of the Amazonian palm *Euterpe oleraceae* (Rodrigues 1994). Variation in infection rates may be due to different anti-fungal defenses across plant species (Gilbert *et al.* 2001) or to genotypic or environmental variation among individuals within a tree species (Bethancourt 2000). In fact, Langenheim and Stubblebine (1983) proposed that differences in chemical defences between adults trees and nearby offspring may reduce pressure on juvenile trees from pests of the adults, and offset the distance effects of the Janzen–Connell Hypothesis. This proposal has yet to be tested for tropical plant diseases, however.

On the surface of many leaves are epifoliar (or epiphyllous) fungi (e.g. sooty moulds) that may act entirely as superficial saprotrophs, consuming honeydew, leaf exudates and detritus. Some species receive nutritional support from the plant by penetrating the leaf cuticle and forming haustoria within host plant cells. Epifoliar fungi do not cause apparent disease symptoms, but may intercept light (but see Anthony *et al.* 2002), or interact synergistically or antagonistically with pathogens to affect disease development (Leben 1965). In a recent comparative study at sites in moist tropical forests in Cape Tribulation in Queensland, Australia and on the Caribbean slope of Panama, 36% (of 182) and 65% (of 81), respectively, of the surveyed understorey plant species were colonized by epifoliar fungi (D. Reynolds and G. S. Gilbert, unpublished data). Similarly, epifoliar fungi were found in 51% and 81% of 120 understorey sampling locations at the Australian and Panamanian sites, respectively. Their commonness, their

potential for direct effects, and the possibility for augmenting or inhibiting the effects of other foliar pathogens lend epifoliar fungi potential importance in tropical forests beyond what is usually recognized.

Asymptomatic fungal infection of leaves (as well as of epiphyte roots (Richardson & Currah 1995; Bayman *et al.* 1997), and bark (Suryanarayanan & Rajagopal 2000)) is clearly ubiquitous in tropical forests, but their importance to the plants is almost entirely unknown. In particular, understanding how many of these fungi are latent pathogens, how they affect diseases caused by other pathogens, or how many may provide other direct benefits to host plants may be key to understanding many aspects of plant diseases.

The importance of plant diseases in tropical forests has only entered the mainstream of thought in plant ecology in the last two decades, and few plant pathologists have ventured into the forest with Petri plate or PCR machine in hand. There is of course a rich history, too vast to review here, of collecting and describing fungi from tropical forests (see e.g. Stevens 1927; Weston 1933), but the collecting has traditionally been haphazard and qualitative, and because the focus was on the fungi, often lacked adequate information about host plants. Despite our patchwork understanding of plant pathogens in tropical forests, a growing body of literature clearly indicates that a plant without disease is a biological anomaly, but that the types of pathogens and that the appearance of disease may vary greatly among hosts and across sites. Pathogens are ubiquitous, but particular plant–pathogen interactions are not. Overall, however, plant pathogens clearly conform to Gillett's vision of an 'inevitable, ubiquitous factor'. The question remains whether they have the impact on plants necessary for them to really affect species diversity in tropical forests.

Significant but not devastating impacts

It is inevitable that each tropical tree will suffer at least some disease over the course of its life, and being diseased is probably the normal state for most trees. However, what is not clear is how large an impact disease has on host survival, growth and reproduction in tropical forests, nor how the impact on individual plants translates into effects on populations or species. For disease to be an important driver of tropical diversity, pathogens must be capable of causing diseases severe enough to limit (but not extinguish) populations of susceptible plant species either directly or by placing them at a competitive disadvantage to other plant species. However, although a highly virulent pathogen with a catholic appetite may have a dramatic impact on forest ecosystems (e.g. *Phytophthora cinnamomi* in temperate Western Australian forests (Weste & Marks 1987)), it is unlikely that such a pathogen will help maintain local plant species diversity. It is the subtle, not the sledgehammer, that is most likely to be important in tropical diversity.

Foliar diseases

Although foliar diseases affect the majority of species studied and may be responsible for between 2% and 75% of all identifiable leaf damage (mean 34%) (Barone 1998), the proportion of leaf tissue actually damaged is often low. García-Guzmán and Dirzo (2001) found that although 45% of leaves suffered damage from fungal pathogens, damage was generally less than 6% of the leaf area. Similarly, Benitez-Malvido *et al*. (1999) found that average damage from fungal pathogens on three host plants in Amazonian forest never exceeded 1.5% of the leaf area. The authors concluded that 'fungal infection is so rare that it is unlikely to affect seedling performance'. However, even very small reductions in leaf area of understorey seedlings may have large impacts on seedling survival (Clark & Clark 1985), and foliar pathogens can cause a large reduction in host growth (Esquivel & Carranza 1996). Clearly, even low levels of foliar disease have the potential for large impacts on plants, but the realized impacts will vary greatly among hosts, pathogen species and sites.

Seed and seedling diseases

By definition, damping-off diseases have a large impact (death) on individual seedlings. On BCI, Augspurger (1984) found that the proportion of seedlings that died from damping-off exceeded 70% for two species of Bombacaceae, but that mortality across eight focal species varied greatly, averaging 35% in the shade. Similarly, Infante (1999) applied fungicide drenches to control damping-off in tree seedlings in the rainforest interior. Five of nine species showed significantly greater survival with fungicide treatment, with an overall 31% increase in mean seedling survival time. However, damping-off was negligible in high light environments such as light gaps (Augspurger 1984) or the edges of forest fragments (Infante 1999). Susceptible host species in conducive environments may suffer dramatic mortality from damping-off pathogens, but like foliar diseases, the impacts will vary among host species and sites.

Cankers, wilts and diebacks

Canker, wilt and dieback pathogens may have large impacts on the growth and survival of both juvenile and mature tropical trees, but there are only a limited number of studies with direct measurements of impact. *Fusarium* dieback of *Acacia koa* in Hawaii (a wilt disease) quickly reduces stomatal conductance and foliar growth, and usually leads to death of the tree (Anderson *et al.* 2001). Inoculation of juveniles of *Tetragastris panamensis* with the canker pathogen *Botryosphaeria dothidea* led to a three-fold increase in mortality, and among survivors, limited growth to 59% of controls (Gilbert & De Steven 1996). Studying the impacts of canker, wilt and dieback pathogens on host growth and mortality is much more difficult than for foliar or seedling diseases because there is often a long latent period between infection and symptom development, and because large, woody

trees have inherently slow rates of growth. Long-term and broadly based studies of canker diseases in tropical forests are needed to make more than an educated guess as to their potential impact on species diversity.

Wood-decay fungi

Wood-decay fungi, particularly those that cause root- and butt-rots of large trees, have been shown to play key roles in the population dynamics and community structure of temperate forests (Hansen & Goheen 2000) and to reduce the growth rate of infected trees (Alexander *et al.* 1981; Bloomberg & Morrison 1989). For tropical forests, although we know that polypore and other wood-decay fungi are common in standing trees (Meza 1992; Gilbert *et al.* 2002; Gilbert & Sousa 2002; Ferrer & Gilbert 2003), we have few data on their importance in either killing trees directly, or making trees susceptible to breaking or falling from excessive wind and water. Although in temperate forests such disease is often an important precursor to trees falling or breaking (e.g. Worrall & Harrington 1988), only 13% of snapped trees on BCI had heart rot; incidence of root rot was not determined for uprooted trees (Putz *et al.* 1983). Recent studies of the diversity and incidence of wood-decay fungi have highlighted their potential importance in tropical forests, but there is much need for work on their ecological effects.

Flower and fruit diseases

A number of plant venereal diseases have been shown to have large impacts in temperate systems (see review in Gilbert 2002), and we might expect similar effects in tropical forests. The potential for large effects of flower and fruit diseases can be illustrated by the ovary-attacking rust *Aecidium farameae*, which reduced fruit set by 75% for infected individuals of the understorey treelet *Faramea occidentalis* (Rubiaceae) on BCI (Travers *et al.* 1998). Similarly, 85% of fertilized ovules of *Anacardium excelsum* (Anacardiaceae) were killed by the fungus *Cladosporium* (Sánchez Garduño *et al.* 1995). Based on these examples, and the high rate of tropical agricultural fruit loss to pathogens (Opoku *et al.* 2002), it would be surprising if a significant proportion of fruits and developing seeds of tropical forest trees were not lost to diseases. However, not enough work has been done on flower and fruit diseases in tropical forests to make an informed guess about their overall importance.

We have good evidence that plant pathogens – and plant-microbe interactions in general – are extremely common in tropical forests. It is also clear that some can have significant impacts on individual plants. However, the effects of pathogens on plants will only be important in the dynamics of tree populations or in maintaining species diversity in the forest if disease reduces the contribution of that population to the next generation. Compensatory responses may prevent even strong effects of disease on individual fitness from affecting numerical population responses (see reviews in Alexander and Holt (1998); Gilbert (2002)).

Integrating effects of disease into plant population dynamics across multiple generations is the next key step in understanding the real impact of diseases on individual plant populations, and will ultimately be central to understanding the importance of disease in tree diversity.

Host specificity

The diversity of fungi (including pathogens) in tropical forests is staggering, and efforts to estimate the true number of fungal species have involved much deliberation over the scale of host specificity (Hawksworth 2001). May (1991) suggested that the most successful plant-infecting fungi in species-rich tropical forests should be non-specialists, because specialists would have difficulty with transmission among individuals of rare host species. However, despite its importance, our estimates of fungal specificity in tropical forests are currently little more than guesses. As for most tropical taxa, fungal diversity takes the form of many rare species and just a few common species (Bills & Polishook 1994; Polishook *et al.* 1996; Huhndorf & Lodge 1997; Arnold *et al.* 2000; Bethancourt 2000; Lindblad 2000; Arnold *et al.* 2001; Gilbert *et al.* 2001; Gilbert *et al.* 2002). The rarity of most species makes determining the scale of host specificity extremely difficult. Most existing literature is only minimally helpful in determining host ranges, since phytopathological and mycological investigation has focused strongly on plants of economic value (Cannon & Hawksworth 1995; Clay 1995), and traditional fungal collecting in forests has been haphazard and non-quantitative. Recently, however, a few systematic, comparative studies (aided by developments in molecular characterization of pathogens) are beginning to provide useful data on the scale of host specificity among plant-infecting fungi in the tropics.

Arnold and coworkers (Arnold *et al.* 2000; Arnold *et al.* 2001) found evidence for host specialization by endophytes from the leaves of two co-occurring but unrelated understorey shrubs, *Heisteria concinna* (Olacaceae) and *Ouratea lucens* (Ochnaceae). Of the 140 morphospecies found more than once, almost two-thirds were found on one host or the other, but not both. Although suggestive, the power of this study is limited because a small number of hosts were studied, the species accumulation curves had not yet saturated, and there was difficulty in differentiating fungal species in largely undescribed groups. Focusing instead on a single fungal genus, Laesse and Lodge found that more than half of 13 species of *Xylaria* isolated from leaves and fruits in Puerto Rico were restricted to a single host-plant genus or family (Laesse & Lodge 1994; Lodge 1997). These studies suggest the potential for ecologically significant specialization among leaf-inhabiting fungi, but much more extensive sampling is needed together with additional use of molecular classification to distinguish among morphologically similar species and to recognize the unity of phenotypically plastic ones.

Recent studies of tropical wood-decay fungi, particularly polypores, also provide a glimpse into the scale of host specificity for another group of fungi. Polypore fungi are taxonomically among the best known of tropical fungi, are largely confined to woody substrates, and produce persistent, showy fruiting bodies convenient for broad systematic surveys. In dry tropical forest in Costa Rica, Lindblad (2000) found that only 3 of 32 common species of polypore fungi showed host specificity, and that there was a strong correlation between the number of hosts a fungus was found on and the commonness of the fungus. In a similar study in seasonal moist tropical forest on BCI, Gilbert *et al.* (2002) found no statistical evidence for host specialization among the 17 species found multiple times. For the five most common fungi, each was found on multiple host species from multiple families (Fig. 6.1a). Although the most common fungi are clearly non-specialists, the assemblages of polypore and ascomycete wood-decay fungi on three host species were readily distinguishable and persistent through time, suggesting that there is sufficient host specialization for host composition to influence the types of fungi present in the forest (Ferrer & Gilbert 2003).

In trying to understand host specificity in species-rich tropical forests, it may be most useful to consider what happens when tree diversity is naturally low. In a Caribbean mangrove forest about 25 km from BCI, only three tree species make up the entire community. In sharp contrast to the high diversity and low dominance of polypore species found on BCI (Gilbert *et al.* 2002; Ferrer & Gilbert 2003), 88% of all polypore collections (nine species total) were comprised of just three fungal species (Gilbert & Sousa 2002). In fact, of the five polypore species found multiple times, all showed high host specificity (Fig. 7.1b). The combination of these polypore studies provides support for May's (1991) prediction of low host specificity in species-rich forests. It may be that in a low-diversity forest where the density of individual host species is high, reliable transmission among hosts allows for the success of host-specialized fungi. In contrast, in high-diversity forests with a low density of individual host species, host non-specialists will be more successful. Similar tests with other groups of fungi should be a high priority for study.

The mangrove polypore study also highlights the importance of scale in the discussion of host specificity. Many herbivorous insects have generalized diets over the entire range of the species, but have much more restricted diets in a local context (Fox & Morrow 1981). Similar behaviour might be expected from plant pathogens. In fact, all the fungi described from the mangroves are described in the literature as attacking non-mangrove hosts (Gilbert & Sousa 2002). Within the local context of a three-species mangrove forest, the fungi are specialists, but globally they are not. It is important to determine the appropriate geographic scale when investigating host specialization.

Although the studies of endophytes and polypores provide a glimpse into host specificity of tropical plant-infecting fungi, only a portion of the included species

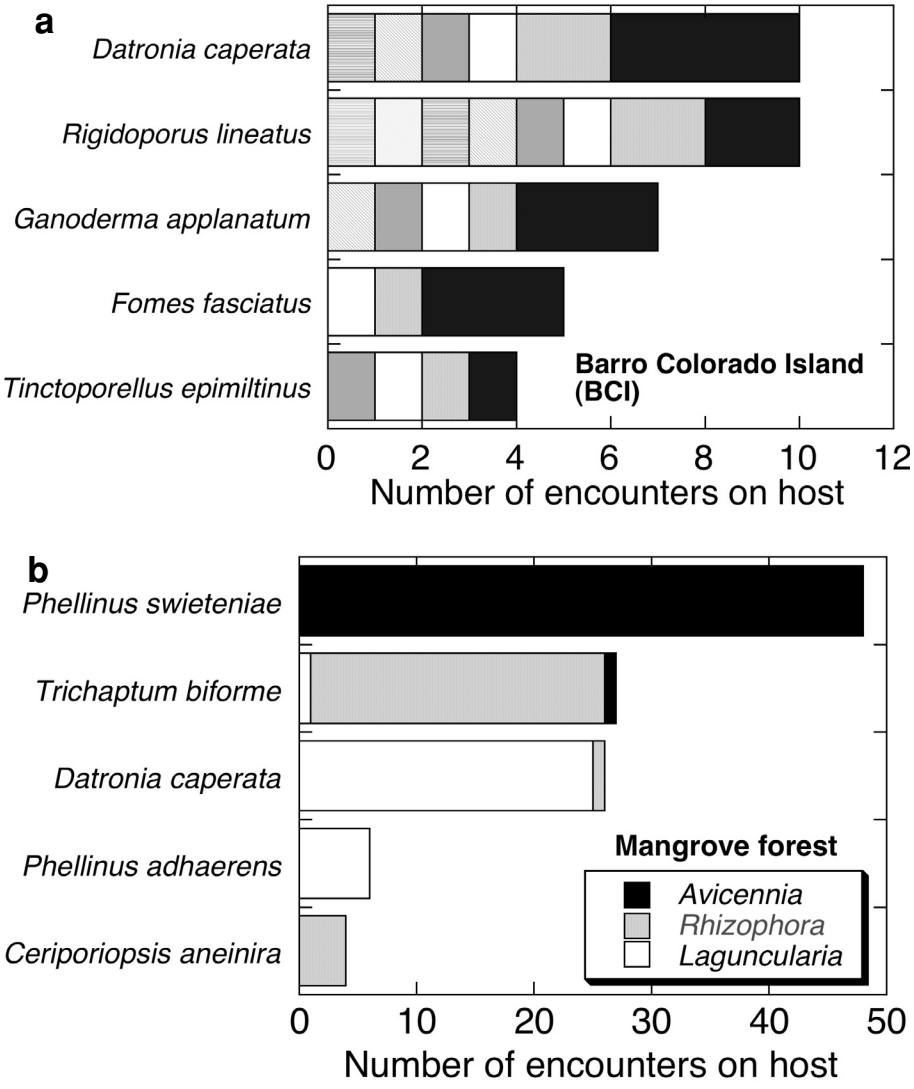

Figure 6.1 Host specificity of the five most common polypore fungi in (a) the seasonal moist tropical forest of Barro Colorado Island and (b) the Caribbean mangrove forest at Punta Galeta, Republic of Panama. For BCI, different shadings within a bar indicate a different host species, but colours are not consistent across fungi. Polypore fungi in the species-rich forest on BCI show no host specificity, whereas in the low-diversity mangrove forest host specificity is high. Data are from Gilbert *et al*. (2002), and Gilbert and Sousa (2002).

are plant pathogens. Systematic studies of host range for pathogens in tropical forests are rare, but one interesting study of a pathogen with a broad host range comes from the study of an unusual luminescent mushroom. The agaric *Mycena citricolor* causes American leaf spot disease of introduced coffee (*Coffea* spp, Rubiaceae), a devastating disease through much of tropical America. It also has an exceptionally large host range. Sequiera (1958) found it causing leaf spots on 150 host plants from 45 families in Costa Rica, including many forest tree species. Although it can cause disease on many hosts, *M. citricolor* may reproduce readily on only a small number of hosts (Sequiera 1958). Lodge and Cantrell (1995) noted that *M. citricolor* became rare in the Luquillo Mountains of Puerto Rico when coffee production was abandoned, but that outbreaks of *M. citricolor* followed the sharp increase in density of native Rubiaceae after hurricane damage to the forest. It may be important to consider source–sink dynamics when pathogens can infect and cause disease on a range of hosts, but are able to reproduce readily on only some subset of those hosts. In a recent temperate example, the spread of sudden oak death (caused by *Phytophthora ramorum*) through California forests has had devastating impacts on oaks and tanoak (*Quercus* spp. and *Lithocarpus densiflorus*, Fagaceae), but much of the source of inoculum appears to come from infections on the sympatric *Umbellularia californica* (Lauraceae), a host that suffers only minimal damage but supports luxuriant reproduction of the pathogen (Rizzo & Garbelotto 2003). Such asymmetrical disease interactions may lead to apparent competition (Alexander & Holt 1998) and must be considered when looking at the importance of host range in the role of diseases in maintaining plant diversity. Our poor understanding of the extent of host specificity, and the potential for linking host population dynamics through shared pathogens, is probably the most important gap in our ability to assess the importance of pathogens in tropical tree diversity.

Effects on dominant plant species

Host specificity is not sufficient for virulent pathogens to prevent competitive exclusion, as envisaged by Gillett. The pathogens must also have greater effects on the locally dominant plant species. There are currently no clear studies from tropical forests of the relative impacts of either individual pathogens across a range of susceptible species or the overall pathogen load of a suite of species across a range of abundance. The ubiquity of density-dependent disease development discussed above suggests that whatever tree species is locally abundant at a particular time is likely to suffer significantly more disease pressure than more rare species. Since density effects are nearly always examined within a single host species, there is little empirical support for this expectation. However, in a broad test across 10 tree species on BCI, the more common tree species were more often colonized by wood-decay fungi (Gilbert *et al.* 2002), suggesting the strong possibility that density-dependent disease development may be observable

even across host species with very different phylogenetic, physiological and ecological backgrounds.

Variability in space or time

Plant diseases meet the requirements of inevitability and potential impact, but for diseases to be important factors in explaining the 'pointless multiplicity of species' in tropical forests, there must be variation in how pathogens affect different tree species. Host specificity may be the most fundamental dimension of variation, but two other dimensions may be equally important ecologically: variation across space due to environmental factors or dispersal limitation, and change over time.

Environmental heterogeneity

> It will be noted, however, that pest pressure is not uniform in all areas; it will be less where there is a cold winter, or a long dry season, than in constantly warm and humid areas where the pests can live and breed all the year round. (Gillett 1962)

Gillett considered that warm temperatures, abundant moisture and aseasonality would favour populations of pests and pathogens, increasing their impacts in areas such as aseasonal moist tropical forests. This could in part explain latitudinal gradients in plant diversity, as well as diversity differences along moisture gradients within tropical regions. Givnish (1999) expanded on this idea, and proposed that increased rainfall, increased soil fertility and decreased seasonality would not only favour pests and pathogens, but should decrease plant investment in anti-herbivore (or anti-pathogen) defenses. This should in turn lead to greater pathogen/pest-driven density-dependent mortality, supporting a greater diversity of tree species in moist tropical communities than elsewhere. He suggests that plant competition should play a greater role in determining plant diversity at high latitudes, and that pest pressure should play a relatively more important role at low latitudes. There is growing evidence for the importance in environmental heterogeneity in causing differential disease impacts at scales from very local to geographical.

The predictions of Gillett and Givnish depend on differential disease development along environmental gradients, with generally greater disease pressure under wetter and more constant climatic conditions. There is a large literature from agricultural systems and temperate-zone natural ecosystems showing greater disease pressure with increased moisture (e.g. Bradley *et al.* (2003) and references therein). In tropical forests, damping-off disease is reduced under low relative humidity associated with light gaps and forest fragment edges (Augspurger & Kelly 1984; Augspurger 1984; Infante 1999). Rodrigues (1994) found greater rates of endophyte infection in understorey saplings than in canopy adults of

the palm *Euterpe oleraceae*, as well as differences in the types of fungi present. She speculates that the differences may be driven by different microclimates experienced by understorey saplings and taller palms. Environmental variation can affect not only the abundance or activity of particular pathogens, but the kinds of fungi present. Different kinds of diseases developed on leaves in the subcanopy shade than in the sun for *Anacardium excelsum* (Gilbert 1995). Finally, Lindblad (2001) found little overlap in polypore species among dry, moist and wet tropical forests in Costa Rica. Across different scales, environmental variation is associated with different kinds and intensity of disease development.

Latitudinal gradient

For the Theory of Pest Pressure to be an effective explanation for differences in plant diversity along the largest environmental gradient of all (from the Equator to the poles), disease pressure must vary along a latitudinal gradient. As supportive evidence, a large number of studies point to the pervasiveness and importance of density-dependent mortality in tropical forests (e.g. Webb & Peart 1999; Harms *et al.* 2000). However, Hille Ris Lambers *et al.* (2002) compared a number of published studies of density-dependent mortality in temperate and tropical forests, and concluded that the proportion of species affected does not change systematically along a latitudinal gradient. They suggest that for density-dependent mortality to explain high diversity of tropical forests, its strength must be greater in the tropics. This idea has yet to be tested.

A formal comparison of the diversity and severity of diseases in forests along a latitudinal gradient or along an environmental gradient within the tropics would be the ideal test of Gillett's and Givnish's predictions, but such a study is complicated by changes in the plant species along those same gradients. Wellman (1968) provides a surrogate measure of disease pressure along a latitudinal gradient. From on a variety of sources and field observations, he catalogued the number of diseases on certain important crop species that were grown under similar conditions both in the temperate United States and in tropical Latin America. For 11 of 12 crops there were significantly more diseases at tropical latitudes than temperate (Fig. 6.2).

Change through time

> Every time that pest pressure helps a species to spread out of the region in which it evolved and to whose conditions it is adapted, a new evolutionary process is started to adapt the species to the conditions of its new habitat. (Gillett 1962)

Some temperate grasses (van der Putten & Peters 1997) herbs (Bever 1994; Bever *et al.* 1997) and trees (Hansen & Goheen 2000) have been shown to build up soil pathogens over time that reduce their own competitiveness and allow for less

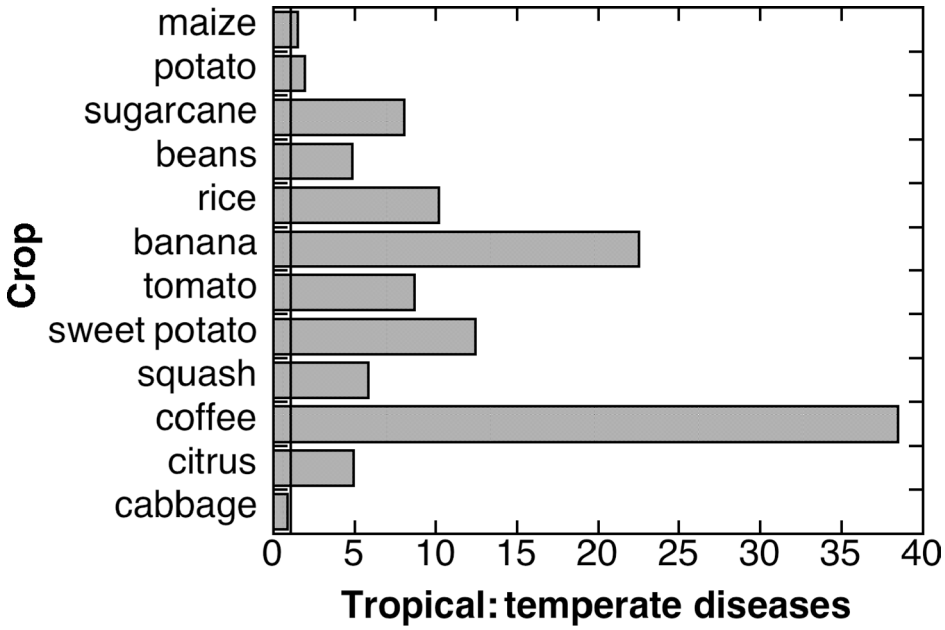

Figure 6.2 The ratio of number of diseases on important crops grown in both tropical and temperate American regions. All crops except cabbage had more diseases in the tropics. The vertical line indicates 1:1 ratio. Data are from Wellman (1968).

susceptible species to replace them. This temporal buildup, followed by a gradual decrease in pathogen pressure, is the basis for crop rotation in agricultural systems. In tropical forests, dispersal of the host to pathogen-free space gives temporal respite from disease pressures, and may help maintain plant species diversity in a dynamic mosaic driven by pathogen feedback. Such feedback may be driven primarily by numerical increase in host-specific pathogens. It can also involve rapid evolution of increased virulence of a pathogen on a particular host. This is particularly likely when the life cycle of the pathogen is much shorter than the life cycle of the host, which would be the case for many pathogens of trees. Selection for greater virulence can occur very quickly; virulence of individual isolates of flax rust (*Melampsora lini*) increased significantly through a single growing season of a wild population of *Linum marginale* (Burdon & Jarosz 1991).

Gillett also recognized that pest pressure could affect plant diversity at levels other than species diversity: 'The effect of pest pressure in producing genetic diversity applies at the intraspecific as well as at the specific, generic, or family level' (Gillett 1962). When pathogens have more limited dispersal than their hosts, local adaptation to particular genotypes may become important. Local adaptation of a pathogen that completes numerous generations on a single

mother tree could accentuate the impact of the pathogen on offspring of that tree, while providing a possible rare-genotype advantage to seedlings from distant mothers of the same species. However, in a reciprocal transplant test of this idea for three species in Panama, Davidson (2000) found no evidence to support a rare-genotype advantage for resistance to seedling diseases. She concluded that for tropical tree species, long-range gene movement and heterogeneity in the rainforest environment are likely to be more important to maintenance of genetic diversity within host species.

Gillett (1962) also proposed that as pathogens force plant species out of disease-ridden areas, the plants are forced to adapt to different environmental conditions and to competition with a different set of neighbouring plant species. In this way, pathogen pressure could drive plant evolution even beyond selection for resistant species and genotypes. Givnish (1999) also suggests that a random walk over evolutionary time through changing pressures from pests and pathogens may work better than Hubbell's ecological drift model (Hubbell 1997, 2001) to account for non-random dominance by certain plant families, or for the predictable shifts in species composition across environmental gradients. Most work to date has considered the role of pathogens in maintenance of diversity, but consideration is due to the role of pathogens in the genesis of species diversity in tropical forests as well.

Concluding remarks

Gillett's 1962 address is too often overlooked in discussions of diseases as driving forces in the maintenance of diversity in tropical forests. It established the framework for later theoretical developments by Janzen (1970), Connell (1971), and Givnish (1999), and outlined the needed empirical work provided by a diverse and growing number of researchers. Since 1962 we have accumulated a significant but still thin body of literature that shows that in tropical forests, plant pathogens could be Gillett's 'inevitable, ubiquitous factors', that they have significant impacts on host performance, and that their effects are variable in both space and time. Some data indicate significant host specificity in tropical forests, but there is still great need for improved understanding of the scale of host specificity and the degree to which dominant plant species must be affected more strongly. Careful, community-based, comparative work is key to advancing our appreciation for the various dimensions of influence of plant pathogens in tropical forests.

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References

Alexander, H. M., & J. Antonovics. 1988. Disease spread and population dynamics of anther-smut infection of *Silene alba* caused by the fungus *Ustilago violacea*. *Journal of Ecology* **76**:91–104.

Alexander, H. M., & R. D. Holt. 1998. The interaction between plant competition and disease. *Perspectives in Plant Ecology Evolution and Systematics* **1**:206–220.

Alexander, S. A., J. M. Skelly, & R. S. Webb. 1981. Effects of *Heterobasidion annosum* on radial growth in southern pine beetle-infested loblolly pine. *Phytopathology* **71**:479–481.

Anderson, R. C., D. E. Gardner, C. C. Daehler, & F. C. Meinzer. 2001. Dieback of *Acacia koa* in Hawaii: ecological and pathological characteristics of affected stands. *Forest Ecology and Management* **5576**:1–14.

Anthony, P. A., J. A. M. Holtum, & B. R. Jackes. 2002. Shade acclimation of rainforest leaves to colonization by lichens. *Functional Ecology* **16**:808–816.

Arnold, A. E., Z. Maynard, & G. S. Gilbert. 2001. Fungal endophytes in dicotyledonous neotropical trees: patterns of abundance and diversity. *Mycological Research* **105**:1502–1507.

Arnold, A. E., Z. Maynard, G. S. Gilbert, P. D. Coley, & T. A. Kursar. 2000. Are tropical fungal endophytes hyperdiverse? *Ecology Letters* **3**:267–274.

Arthur, J. C., & G. B. Cummings. 1933. New species of Uredinales. *Annales Mycologica* **31**:41.

Augspurger, C. K. 1983a. Offspring recruitment around tropical trees – changes in cohort distance with time. *Oikos* **40**:189–196.

1983b. Seed dispersal of the tropical tree, *Platypodium elegans*, and the escape of its seedlings from fungal pathogens. *Journal of Ecology* **71**:759–771.

1984. Seedling survival of tropical tree species – interactions of dispersal distance, light-gaps, and pathogens. *Ecology* **65**:1705–1712.

1990. Spatial patterns of damping-off disease during seedling recruitment in tropical forests. In J. J. Burdon & S. R. Leather, eds. *Pests, Pathogens, and Plant Communities*. Oxford: Blackwell Scientific Publications, pp. 131–144.

Augspurger, C. K., & C. K. Kelly. 1984. Pathogen mortality of tropical tree seedlings: experimental studies of the effects of dispersal distance, seedling density, and light conditions. *Oecologia* **61**:211–217.

Barone, J. A. 1998. Host-specificity of folivorous insects in a moist tropical forest. *Journal of Animal Ecology* **67**:400–409.

Bayman, P., P. Angulo-Sandoval, Z. Báez-Ortiz, & D. J. Lodge. 1998. Distribution and dispersal of *Xylaria* endophytes in two tree species in Puerto Rico. *Mycological Research* **102**:944–948.

Bayman, P., L. L. Lebron, R. L. Tremblay, & D. J. Lodge. 1997. Variation in endophytic fungi from roots and leaves of *Lepanthes* (Orchidaceae). *New Phytologist* **135**:143–149.

Bella, I. E., & S. Navratil. 1988. Western gall rust dynamics and impact in young lodgepole pine stands in west-central Alberta. *Canadian Journal of Forest Research* **18**:1437–1442.

Benitez-Malvido, J., G. García-Guzman, & I. D. Kossmann-Ferraz. 1999. Leaf-fungal incidence and herbivory on tree seedlings in tropical rainforest fragments: an experimental study. *Biological Conservation* **91**:143–150.

Bethancourt, E. 2000. Un método de muestreo para el estudio de la diversidad de hongos endófitos asociado a especies arbóreas en un bosque tropical. Unpublished Master's thesis, Universidad Tecnológica de Panamá.

Bever, J. D. 1994. Feedback between plants and their soil communities in an old field community. *Ecology* **75**:1965–1977.

Bever, J. D., K. M. Westover, & J. Antonovics. 1997. Incorporating the soil community into plant population dynamics: The utility of the feedback approach. *Journal of Ecology* **85**:561–573.

Bills, G. F., & J. D. Polishook. 1994. Abundance and diversity of microfungi in leaf litter of a lowland rain forest in Costa Rica. *Mycologia* **86**:187–198.

Bloomberg, W. J., & D. J. Morrison. 1989. Relationship of growth reduction in Douglas-fir to infection by Armillaria root disease in southeastern British Columbia [Canada]. *Phytopathology* **79**:482–487.

Bradley, D. J., G. S. Gilbert, & I. M. Parker. 2003. Susceptibility of clover species to fungal infection: the interaction of leaf surface traits and environment. *American Journal of Botany* **90**:857–864.

Burdon, J. J., & G. A. Chilvers. 1982. Host density as a factor in plant disease ecology. *Annual Review of Phytopathology* **20**: 143–166.

Burdon, J. J., & A. M. Jarosz. 1991. Host-pathogen interactions in natural populations of *Linum marginale* and *Melampsora lini*. 1. Patterns of resistance and racial variation in a large host population. *Evolution* **45**:205–217.

Cannon, P. F., & D. L. Hawksworth. 1995. The diversity of fungi associated with vascular plants: the known, the unknown and the need to bridge the knowledge gap. *Advances in Plant Pathology* **11**:277–302.

Carlsson-Graner, U. 1997. Anther-smut disease in *Silene dioica*: Variation in susceptibility among genotypes and populations, and patterns of disease within populations. *Evolution* **51**:1416–1426.

Chen, W.-Q., D. E. Gardner, & D. T. Webb. 1996. Biology and life cycle of *Atelocauda koae*, an unusual demicyclic rust. *Mycoscience* **37**:91–98.

Clark, D. A., & D. B. Clark. 1984. Spacing dynamics of a tropical rain forest tree: evaluation of the Janzen–Connell model. *American Naturalist* **124**:769–788.

Clark, D. B., & D. A. Clark. 1985. Seedling dynamics of a tropical tree: Impacts of herbivory and meristem damage. *Ecology* **66**:1884–1892.

Clay, K. 1995. Correlates of pathogen species richness in the grass family. *Canadian Journal of Botany* **73**:S42–S49.

Connell, J. H. 1971. On the role of natural enemies in preventing competitive exclusion in some marine animals and in rain forest trees. In P. J. Boer & G. R. Graadwell, eds. *Dynamics of Numbers in Populations (Proceedings of the Advanced Study Institute, Osterbeek 1970)*. Wageningen: Centre for Agricultural Publication and Documentation, pp. 298–312.

Dalling, J. W., M. D. Swaine, & N. C. Garwood. 1998. Dispersal patterns and seed bank dynamics of pioneer trees in moist tropical forest. *Ecology* **79**:564–578.

Davelos, A. L., H. M. Alexander, & N. A. Slade. 1996. Ecological genetic interactions between a clonal host plant (*Spartina pectinata*) and associated rust fungi (*Puccinia seymouriana* and *Puccinia sparganioides*). *Oecologia* **105**:205–213.

Davidson, J. M. 2000. Pathogen-mediated maintenance of diversity and fitness consequences of near versus far pollination for tropical trees. Unpublished Ph.D. dissertation, University of California, Davis.

Esquivel, R. E., & J. Carranza. 1996. Pathogenicity of *Phylloporia chrysita* (Aphyllophorales: Hymenochaetaceae) on *Erythrochiton gymnanthus* (Rutaceae). *Revista de Biologia Tropical* **44**:137–145.

Fail, G. L., & J. H. Langenheim. 1990. Infection processes of *Pestalotia subcuticularis* on leaves of *Hymenaea courbaril*. *Phytopathology* **80**:1259–1265.

Ferrer, A., & G. S. Gilbert. 2003. Effect of tree host species on fungal community composition in a tropical rain forest in

Panama. *Diversity and Distributions* **9**:455–468.

Fox, L. R., & P. A. Morrow. 1981. Specialization: species property or local phenomenon? *Science* **211**:887–893.

García-Guzmán, G., J. J. Burdon, J. E. Ash, & R. B. Cunningham. 1996. Regional and local patterns in the spatial distribution of the flower-infecting smut fungus *Sporisorium amphilophis* in natural populations of its host *Bothriochloa macra*. *New Phytologist* **132**:459–469.

García-Guzmán, G., & R. Dirzo. 2001. Patterns of leaf-pathogen infection in the understory of a Mexican rain forest: incidence, spatiotemporal variation, and mechanisms of infection. *American Journal of Botany* **88**:634–645.

Gardner, D. E. 1994. The native rust fungi of Hawaii. *Canadian Journal of Botany* **72**:976–989.

Gause, G. F. W. 1934. *The Struggle for Existence*. Baltimore: Williams and Wilkins.

Gilbert, G. S. 1995. Rain forest plant diseases: The canopy-understory connection. *Selbyana* **16**:75–77.

2002. Evolutionary ecology of plant diseases in natural ecosystems. *Annual Review of Phytopathology* **40**:13–43.

Gilbert, G. S., & D. de Steven. 1996. A canker disease of seedlings and saplings of *Tetragastris panamensis* (Burseraceae) caused by *Botryosphaeria dothidea* in a lowland tropical forest. *Plant Disease* **80**:684–687.

Gilbert, G. S., A. Ferrer, & J. Carranza. 2002. Polypore fungal diversity and host density in a moist tropical forest. *Biodiversity and Conservation* **11**:947–957.

Gilbert, G. S., S. P. Hubbell, & R. B. Foster. 1994. Density and distance-to-adult effects of a canker disease of trees in a moist tropical forest. *Oecologia* **98**:100–108.

Gilbert, G. S., M. Mejía-Chang, & E. Rojas. 2001. Fungal diversity and plant disease in mangrove forests: salt excretion as a possible defense mechanism. *Oecologia* **132**:278–285.

Gilbert, G. S., & W. P. Sousa. 2002. Host specialization among wood-decay polypore fungi in a Caribbean mangrove forest. *Biotropica* **34**:396–404.

Gillett, J. B. 1962. Pest pressure, an underestimated factor in evolution. *Systematics Association Publication Number* **4**:37–46.

Givnish, T. J. 1999. On the causes of gradients in tropical tree diversity. *Journal of Ecology* **87**:193–210.

Hansen, E. M., & E. M. Goheen. 2000. *Phellinus weirii* and other native root pathogens as determinants of forest structure and process in western North America. *Annual Review of Phytopathology* **38**: 515–539.

Harms, K. E., S. J. Wright, O. Calderon, A. Hernandez, & E. A. Herre. 2000. Pervasive density-dependent recruitment enhances seedling diversity in a tropical forest. *Nature* **404**:493–495.

Hawksworth, D. L. 2001. The magnitude of fungal diversity: the 1.5 million species estimate revisited. *Mycological Research* **105**:1422–1432.

Hille Ris Lambers, J., J. S. Clark, & B. Beckage. 2002. Density-dependent mortality and the latitudinal gradient in species diversity. *Nature* **417**:732–735.

Hubbell, S. P. 1997. A unified theory of biogeography and relative species abundance and its application to tropical rain forests and coral reefs. *Coral Reefs* **16**: S9–S21.

2001. *The Unified Neutral Theory of Biodiversity and Biogeography*. Princeton University Press, Princeton.

Huhndorf, S. M., & D. J. Lodge. 1997. Host specificity among wood-inhabiting pyrenomycetes (Fungi, Ascomycetes) in a wet tropical forest in Puerto Rico. *Tropical Ecology* **38**:307–315.

Infante, L. A. 1999. Complex interactions: exploring the role of soilborne plant pathogens in tropical seedling communities. Unpublished M.Sc. thesis, University of California, Berkeley.

Janzen, D. H. 1970. Herbivores and the number of tree species in tropical forests. *The American Naturalist* **104**:501–527.

Kitajima, K., & C. K. Augspurger. 1989. Seed and seedling ecology of a monocarpic tropical tree, *Tachigalia versicolor*. *Ecology* **70**:1102–1114.

Laesse, T., & D. J. Lodge. 1994. Three host-specific *Xylaria* species. *Mycologia* **86**:436–446.

Langenheim, J. H. & W. H. Stubblebine. 1983. Variation in leaf resin composition between parent tree and progeny in *Hymenaea*: Implications for herbivory in the humid tropics. *Biochemistry Systematics and Ecology* **11**:97–106.

Leben, C. 1965. Epiphytic microorganisms in relation to plant disease. *Annual Review of Phytopathology* **3**:209–230.

Lee, S. S. 1999. Forest health in plantation forests in south-east Asia. *Australasian Plant Pathology* **28**:283–291.

Lindblad, I. 2000. Host specificity of some wood-inhabiting fungi in a tropical forest. *Mycologia* **92**:399–405.

2001. Diversity of poroid and some corticoid wood-inhabiting fungi along the rainfall gradient in tropical forests, Costa Rica. *Journal of Tropical Ecology* **17**:353–369.

Lodge, D. J. 1997. Factors related to diversity of decomposer fungi in tropical forests. *Biodiversity and Conservation* **6**:681–688.

Lodge, D. J., & S. Cantrell. 1995. Fungal communities in wet tropical forests: variation in time and space. *Canadian Journal of Botany* **73**:S1391–S1398.

Lodge, D. J., P. J. Fisher, & B. C. Sutton. 1996. Endophytic fungi of *Manilkara bidentata* leaves in Puerto Rico. *Mycologia* **88**: 733–738.

May, R. M. 1991. A fondness for fungi. *Nature* **352**:475–476.

Meza, M. R. 1992. La pudrición del duramen de los Arboles en pie en Venezuela. El caso de la Mora de la Guayana. Unpublished Master's thesis, Universidad de Los Andes.

Opoku, I. Y., A. Y. Akrofi, & A. A. Appiah. 2002. Shade trees are alternative hosts of the cocoa pathogen *Phytophthora megakarya*. *Crop Protection* **21**:629–634.

Packer, A., & K. Clay. 2000. Soil pathogens and spatial patterns of seedling mortality in a temperate tree. *Nature* **404**: 278–281.

Polishook, J. D., G. F. Bills, & D. J. Lodge. 1996. Microfungi from decaying leaves of two rain forest trees in Puerto Rico. *Journal of Industrial Microbiology* **17**:284–294.

Putz, F. E., P. D. Coley, K. Lu, A. Montalvo, & A. Aiello. 1983. Uprooting and snapping of trees: structural determinants and ecological consequences. *Canadian Journal of Forest Research* **13**:1011–1020.

Richardson, K. A., & R. S. Currah. 1995. The fungal community associated with the roots of some rainforest epiphytes of Costa Rica. *Selbyana* **16**:49–73.

Rizzo, D. M., & M. Garbelotto. 2003. Sudden oak death: endangering California and Oregon forest ecosystems. *Frontiers in Ecology and the Environment* **1**:197–204.

Rodrigues, K. F. 1994. The foliar fungal endophytes of the Amazonian palm *Euterpe oleracea*. *Mycologia* **86**:376–385.

Sánchez Garduño, C., S. J. Wright, & C. Potvin. 1995. Seed predation. In S. J. Wright & M. Colley, eds. *Accessing the Canopy.* Panama: United Nations Environment Programme / Smithsonian Tropical Research Institute, pp. 18–19.

Schoeman, M. H., B. Q. Manicom, & M. J. Wingfield. 1995. Epidemiology of powdery mildew on mango blossoms. *Plant Disease* **79**:524–528.

Sequiera, L. 1958. The host range of *Mycena citricolor* (Berk. & Curt.) Sacc. *Turrialba* **8**:136–147.

Stevens, F. L. 1927. Fungi from Costa Rica and Panama. *Illinois Biological Monographs* **11**:1–255.

Suryanarayanan, T. S., & K. Rajagopal. 2000. Fungal endophytes (Phellophytes) of some tropical forest trees. *Indian Forester* **126**:165–170.

Tilman, D. 1982. *Resource Competition and Community Structure*. Princeton, NJ: Princeton University Press.

Travers, S. E., G. S. Gilbert, & E. F. Perry. 1998. The effect of rust infection on reproduction in a tropical tree (*Faramea occidentalis*). *Biotropica* **30**: 438–443.

van der Putten, W. H., & B. A. M. Peters. 1997. How soil-borne pathogens may affect plant competition. *Ecology* **78**: 1785–1795.

Vánky, K. 2002. *Illustrated Genera of Smut Fungi*, 2nd edn. St Paul, MN: APS Press.

Webb, C. O., & D. R. Peart. 1999. Seedling density dependence promotes coexistence of Bornean rain forest trees. *Ecology* **80**:2006–2017.

Wellman, F. L. 1968. More diseases on crops in the tropics than in the temperate zone. *Ceiba* **14**:17–28.

Wellman, F. L., & E. Echandi. 1981. The coffee rust situation in Latin America in 1980. *Phytopathology* **71**:968–971.

Weste, G., & G. C. Marks. 1987. The biology of *Phytophthora cinnamomi* in Australasian forests. *Annual Review of Phytopathology* **25**:207–229.

Weston, W. H. 1933. The fungi of Barro Colorado. *Science Monthly* **36**.

Wilson, D. 1995. Endophyte – the evolution of a term, and clarification of its use and definition. *Oikos* **73**.

Worrall, J. J., & T. C. Harrington. 1988. Etiology of canopy gaps in spruce-fir forests at Crawford Notch, New Hampshire [USA]. *Canadian Journal of Forest Research* **18**:1463–1469.