5.1 Introduction

Mounting evidence indicates that plant pathogens can influence the population density, genetic structure, and spatial structure of their hosts (Gilbert 2002). At the same time, it is increasingly clear that the spread of plant pathogens is influenced not only by the density, genetic structure, and spatial structure of single-species host populations, but also by the structure of entire communities. Together, such reciprocal effects between plant and pathogen populations create the potential for dynamic feedbacks between the spread of plant pathogens and the structure of ecological communities. This chapter focuses on these feedbacks and the mechanisms that may drive them. To set the stage for summarizing such feedbacks, we first separately review recent evidence for effects of plant community structure on pathogen spread, and effects of pathogens on plant community structure. In each case, specialist and generalist pathogens (here defined as infecting one versus multiple species within a focal host community) are seen to produce contrasting dynamics. We also extend this community-level framework for plant-pathogen interactions to include other species in the biotic community, such as herbivores and microbial symbionts.

Why focus on plant pathogens? First, plants play a fundamental role in communities, as the primary producers on which most other organisms and ecological processes ultimately depend. This fundamental role of plants suggests that plant pathogens may have impacts that reverberate through entire communities. For example, the structure of plant communities, particularly their species composition and diversity, can control aspects of ecosystem functioning (Loreau et al. 2001; Tilman et al. 2002; van Ruijven and Berendse 2003), so pathogens may influence ecosystem functioning through their impacts on plant community structure.

Second, plant-pathogen interactions provide excellent model systems for understanding general principles in disease ecology (Antonovics et al. 2002). They are ideal for directly testing theory about the effects of host community structure on pathogen spread, because host community structure can be readily manipulated on relevant spatial scales in the field (Mitchell et al. 2002; Mitchell et al. 2003; Power and Mitchell 2004). The presence or abundance of plant pathogens can also be manipulated in the field (Paul et al. 1989; Mitchell 2003; Power and Mitchell 2004), although typically this is more logistically challenging than manipulations of their plant hosts.

A great strength of disease ecology to date is that theoretical and empirical progress have been tightly linked (Grenfell and Dobson 1995; Hudson et al. 2002; Dwyer et al. 2004). However, because of the relatively large spatial scale and mobility of many wildlife populations, independently manipulating multiple species in a community will often be impractical. Although other tools such as analysis of long-time series and parameterization of theoretical models can still provide direct tests of theory (Begon et al. 1999; Gilbert et al. 2001; Tompkins et al. 2003), experimentation is the most rigorous approach for understanding causality in complex feedbacks between hosts and pathogens. Thus, the potential to independently manipulate dozens of plant species on the spatial scale of several square meters provides an excellent opportunity to understand the
dynamics of feedbacks between communities of hosts and pathogens.

What pathogens infect plants? There are over 10,000 named species of plant pathogens (Agrios 2004), but this is almost certainly an underestimate by at least an order of magnitude. For comparison, there are 250,000 species of vascular plants to serve as potential hosts (Whitton and Rajakaruna 2001). Plant pathogens are phylogenetically diverse as well, including fungi, viruses, bacteria, nematodes, and protists. Over three-quarters of known plant pathogens are fungi, but they have probably been more completely enumerated because they are typically more visually obvious.

Many, probably most, plant species are susceptible to infection by multiple pathogen species (Farr et al. 1989). The limited available theory, largely developed for animal hosts, suggests that specialist pathogen community structure and diversity will depend on host population density (Dobson 1990), and thus perhaps host community structure. However, there is a striking paucity of empirical studies that consider interactions among multiple pathogens infecting wild plant populations.

Plant pathogens have long been studied primarily because of their impact on agricultural production. Even with current efforts to control them, they reduce global crop production by approximately one-sixth, roughly equal to the respective impacts of herbivores and weeds (Oerke et al. 1994). Although crop diseases have been known since at least the time of the ancient Greeks (Agrios 2004), a substantial factor in their recent large impact is undoubtedly the common practice of growing crops as monocultures, often genetic monocultures. As a result, since the start of the green revolution over forty years ago, there has been rising interest in increasing diversity within cropping systems. This interest has led to the development of conceptual frameworks and an extensive body of empirical tests of the spread of plant pathogens in diverse host populations (Power 1987, 1991; Boudreau and Mundt 1997; Garrett and Mundt 1999; Zhu et al. 2000; Mundt 2002). While this interest in diversity was largely inspired by observations of natural plant communities, there have been few experimental tests of these processes in complex natural communities.

One major simplification in agricultural systems is that there is little potential for long-term feedbacks from pathogens to host population dynamics because the host population is directly controlled by humans. This has allowed models of disease in agricultural systems to assume that host abundance is not a dynamic variable (van der Plank 1963; Leonard 1969; Jeger et al. 1981; Garrett and Mundt 1999; Segarra et al. 2001). Because of such differences between agricultural and unmanaged systems, and because the spread of disease in agricultural mixtures has recently been reviewed (Mundt 2002), we focus here on disease in unmanaged plant communities. We note, however, that studies of unmanaged communities would be much more difficult were it not for the conceptual and methodological developments inspired by studies of crop pathogens. In particular, numerous studies show that the diversity and composition of crops both influence, and can be affected by, the spread of plant pathogens (Power 1987, 1990, 1991; Boudreau and Mundt 1997; Garrett and Mundt 1999; Zhu et al. 2000; Mundt 2002). These are the agricultural roots of plant community epidemiology.

5.2 Plant community effects on pathogens

Species richness (the number of species present) and species composition (the identity of species present) are two characteristics of plant communities that are likely to influence the spread of pathogens. The potential for species richness and composition to influence disease arises from the fact that species inherently vary in characteristics that influence disease dynamics. The greater the extent of such functional variation among species in a community, the greater is the potential for strong effects of species richness and composition (Tilman and Lehman 2002).

Many characteristics that vary among plant species—from defensive compounds to photosynthetic pathway to architecture to phenology—might influence pathogen spread either directly or indirectly. Surprisingly little work has examined variation among wild plant species in components of pathogen transmission such as susceptibility or infectiousness (Box 5.1). In a recent exception, a
Box 5.1 Transmission of fungi and viruses in diverse plant communities

 Transmission of plant pathogens is necessarily a multistage process, which can be broadly divided into pre-dispersal events (production and liberation of propagules from an infected host), dispersal events (movement and survival of propagules), and post-dispersal events (survival and infection of a susceptible host). While all of these components are well studied in various crop-pathogen systems (McCartney 1997; Jones 1998; Garrett and Mundt 1999; Aylor 2003; Gray and Gildow 2003), they are likely to operate differently in species-rich, unmanaged plant communities (Boudreau and Mundt 1997; Garrett and Mundt 1999). Additionally, for a given plant pathogen, each stage is likely to depend on the species composition of the host community because host species will likely vary in traits influencing each component of transmission. The limited theory available on multi-host pathogen systems suggests that correlations among host species in their effects on these various components can strongly influence disease dynamics (Leger et al. 1981; Garrett and Mundt 1999), and that transmission of specialist pathogens can be highly sensitive to the identity of non-host species in the community (Boudreau and Mundt 1997). Moreover, for most pathogens, each stage of the transmission process is highly dependent on abiotic conditions (McCartney 1997) and perhaps on interactions with other species, particularly vectors (Power and Gray 1995; Gray and Gildow 2003). This creates an enormous number of factors that could potentially control transmission processes, raising the question of which are essential for understanding particular systems. Notably, in the vast majority of agricultural studies, the details of transmission are not explicitly considered, and the simplifying assumption is made that observed patterns of disease prevalence or severity reflect the process of transmission.

More mechanistically, Garrett and Mundt (1999) have developed a conceptual framework for disease spread in genetically diverse crop populations, which we summarize here and seek to extend to unmanaged, multispecies communities. They conclude that five major traits of plant-pathogen systems should contribute to reduce spread of disease in more species-rich populations. Adapting their terminology for unmanaged multispecies communities, those traits are: (1) small contiguous area of each host species or small plant size; (2) shallow pathogen dispersal gradient; (3) small pathogen lesion size or large pathogen carrying capacity; (4) short pathogen generation time; and (5) strong host specialization by the pathogen. They also conclude that disease spread will depend on host species composition if plants vary in traits such as competitive ability and potential for compensatory growth. Finally, they argue that the importance of these various traits will vary among systems and among environments, for example, depending on whether climate is favorable to epidemic development, and whether epidemics in the focal populations are driven by inoculum from distant populations. Garrett and Mundt (1999) did not try to incorporate vector-transmitted pathogens into their framework. For vector-transmitted pathogens, vector behavior is likely to be a key control on pathogen transmission (Power 1991; Power and Gray 1995; Gray and Gildow 2003). For example, strong host specialization by a vector might substitute for pathogen specialization as a contributor to reduced disease spread in more diverse populations. For directly transmitted pathogens, the factors outlined by Garrett and Mundt (1999) provide the best starting point for a mechanistic understanding of the processes controlling pathogen transmission in species-diverse plant communities.

A study of 18 wild clover species in California found that species with larger leaves retained more water on the surface, and species with greater water retention were more susceptible to infection by a generalist foliar fungal pathogen (Bradley et al. 2003). This study provides unusual mechanistic insight into sources of among-species variation in pathogen susceptibility. Having documented this variation at the species level, it would be interesting to see if this allows prediction of processes at the community level, e.g., how the richness and composition of communities of clover influence spread of the shared pathogen, and how those processes vary along moisture gradients.

5.2.1 Specialist pathogens

For pathogens specialized on one or a few host species within a community, decreased species richness in that community is predicted to increase spread of directly transmitted pathogens (Leonard 1969; Burdon and Chilvers 1976; Chapin et al. 1997; Garrett and Mundt 1999). The primary hypothesized mechanism is that decreased species richness
decreases competition for resources among the remaining species, allowing them to increase in abundance, on average (Burdon and Chilvers 1976; Boudreau and Mundt 1997; Chapin et al. 1997). In turn, increased abundance of those species increases disease transmission of their specialized pathogens by decreasing the average distance between host individuals (Burdon and Chilvers 1982; Gilbert 2002). Vector transmission may complicate this expectation by partially decoupling transmission rate from inter-host distance (e.g. Power 1990; Antonovics and Alexander 1992). While this long-standing hypothesis is supported by numerous tests with highly specialized pathogens of individual crop genotypes (Mundt 2002), whether the same process occurs in species-diverse unmanaged plant communities has only recently been tested experimentally.

In two field experiments with perennial grassland species in Minnesota, decreased plant richness increased pathogen load of foliar fungi (percentage of leaf area infected across the plant community) (Mitchell et al. 2002, 2003). In both experiments, pathogen load was nearly three times as high in monocultures than in communities of approximately natural species richness (i.e. 16–24 species). Species richness was manipulated directly by seeding plots with randomly chosen species numbers, then removing unplanted species by hand-weeding. Most individual diseases followed the same pattern as at the community level in that they were more severe in less species-rich plant communities. As predicted by theory, change in the relative abundance of individual host species was the chief mechanism for the effects of species richness on disease. Decreasing species richness increased transmission of individual diseases that persisted in the community by allowing their host species to increase in abundance.

When plant species richness changes through species loss or introduction, the species composition of the community necessarily changes as well. Thus, it is important to separate the effects of composition and richness. In the two experiments in Minnesota, species composition and richness both independently influenced pathogen load (Mitchell et al. 2002, 2003). Species composition more strongly influenced pathogen load than did richness. In this case, species composition was quantified functionally in terms of the constituent species’ disease proneness, or their percentage of leaf area infected in the most species-rich communities. Across the gradient in species richness, communities comprised of more disease-prone species had heavier pathogen loads and communities of less disease-prone species had lighter pathogen loads (Fig. 5.1), reflecting the joint control of pathogen load by species composition and richness. However, in contrast to the effects of plant species richness, the spread of individual diseases was not strongly influenced by community composition. Thus, the effect of species composition at the community level resulted not from an effect of species composition on transmission, but simply from the presence or absence of disease-prone host species and their associated pathogens.

### 5.2.2 Generalist pathogens

Several bodies of theory predict that the spread of generalist plant pathogens can be influenced by host community richness, but that this will depend critically on species composition. Theory developed for directly transmitted crop pathogens predicts that decreased richness can increase or decrease

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**Figure 5.1** Foliar fungal pathogen load was jointly controlled by plant species richness and composition in a Minnesota grassland community. Pathogen load (percentage of leaf area infected across the plant community) increased logarithmically as species richness decreased. Across the gradient in species richness, communities composed of highly disease-prone species (black circles) had higher pathogen loads than communities composed of moderately disease-prone species (gray circles) and communities composed of weakly disease-prone species (white circles). Data from Mitchell et al. (2002); figure previously unpublished.
spread of generalist pathogens, depending on host species composition, particularly differences in resistance traits among hosts (Jeger et al. 1981; Garrett and Mundt 1999). Experimental tests with directly transmitted generalist crop pathogens have generally found weak, inconsistent reductions of disease at higher richness (Mundt 2002), perhaps because effects of richness are overridden by composition. Two more general theoretical investigations both recently concluded that decreased richness was more likely to decrease establishment of directly transmitted generalist pathogens and increase establishment of vector-transmitted generalist pathogens (Holt et al. 2003; Dobson 2004). Further, these two studies predict that the effects of richness depend on species composition, particularly host species' effects on the pathogen population (and the vector population in the vector-transmitted case).

Pathogen spillover among host species is thought to be one mechanism for effects of species composition on generalist pathogens. Pathogen spillover occurs when a pathogen population builds up in a highly susceptible host population, then is transmitted from individuals in that population to individuals in a more resistant host population (Daszak et al. 2000; Dobson 2004; Power and Mitchell 2004). Spillover results from asymmetries in transmission rates among species. It is thus predicted to be a common feature of generalist pathogens in communities including multiple host species because host species almost inevitably vary in key epidemiological traits that can generate such asymmetries (Woolhouse et al. 2001; Bradley et al. 2003; Holt et al. 2003; Dobson 2004; Power and Mitchell 2004). Spillover has been reported for numerous pathogens, but most examples center on domesticated populations, and the process has rarely been examined experimentally (Daszak et al. 2000; Power and Mitchell 2004).

In an experiment in 1999 with annual wild grasses in central New York State, community prevalence (percentage of individuals infected) of a generalist aphid-vectored virus was controlled by species composition, but not by species richness (Power and Mitchell 2004). Communities that contained one species previously identified as highly susceptible to the virus, *Avena fatua* (wild oats), were over 10 times more heavily infected than communities lacking *Avena*. These results suggest that host species richness was a less important factor in pathogen spread than species composition, particularly the presence of a highly susceptible species. High community virus prevalence resulted in part from the high rate of infections in the population of *Avena* itself, but also—and more interestingly—because *Avena*’s presence increased prevalence in more resistant host populations through pathogen spillover. Spillover from *Avena* to some species was very strong—for example, presence of *Avena* in the community increased virus prevalence in *Setaria lutescens* (yellow foxtail) by about an order of magnitude (Power and Mitchell 2004).

To further investigate the dynamics and consequences of pathogen spillover, we conducted an additional experiment in 2001 in which *Avena* presence was factoredly manipulated with virus presence and nitrogen addition. *Avena* presence increased virus prevalence across the rest of the community (three other species) by 60% (Fig. 5.2) (Power and Mitchell 2004). As a result of pathogen spillover, aboveground primary production was lower in communities that included *Avena* and that were inoculated with the virus (Mitchell and Power, unpublished data).

These experiments support the view that the spread of generalist pathogens is crucially dependent on host community species composition. The

![Figure 5.2](image)

Figure 5.2 Virus prevalence across *Digitaria sanguinalis* (black bars), *Lolium multiflorum* (white bars), and *Setaria lutescens* (gray bars) in experimental communities planted with *A. fatua* or lacking *A. fatua* in 2001. The presence of *A. fatua* increased virus prevalence across the other three species (*P < 0.05*), demonstrating pathogen spillover. Data shown are means ± SE. Redrawn from Power and Mitchell (2004).
epidemiology of generalist pathogens is inherently a community-level process—understanding disease dynamics in any host population, especially non-reservoir species, requires knowledge of disease dynamics in other host populations. Pathogen prevalence in a population is a function not just of factors intrinsic to a focal host population, such as genetically based resistance, physiological stress, and population abundance, but of the community context in which host and pathogen are embedded.

In complement to these few highly controlled experiments with annual grasses, many non-manipulative studies of generalist forest pathogens have revealed a strong dependence of disease dynamics on landscape-scale variation in plant community structure. For example, sudden oak death disease, caused by the protist Phytophthora ramorum, is of current public concern primarily because of its devastating effects on canopy trees, especially oaks (Quercus spp.) and tanoaks (Lithocarpus densiflora) in coastal forests of California and Oregon (Rizzo and Garbelotto 2003). Tanoaks appear capable of sustaining epidemics themselves, probably because the pathogen can infect almost any part of a tanoak, from leaves to trunk (Rizzo and Garbelotto 2003). However, the best biological predictor of disease-induced mortality of oaks across a California landscape was abundance of an understory shrub, bay laurel (Umbellularia californica) (Kelly and Meentemeyer 2002). Preliminary evidence suggests that bay laurel drives epidemics in oak populations (Rizzo and Garbelotto 2003; Meentemeyer et al. 2004; Davidson et al. 2005). The pathogen infects laurel leaves, where it can produce many spores, apparently without greatly increasing mortality. In contrast, oak infections are limited to the trunk and branches, which can girdle and kill trees, but produce no spores for further transmission (Davidson et al. 2005). Thus, variation in tolerance to the negative effects of disease, not just susceptibility to infection, can drive pathogen spillover. As a result, conservation of these endangered forests may hinge on understanding the community epidemiology of sudden oak death.

The sudden oak death pathogen appears to have been recently introduced to the United States, perhaps from Asia, although its native range remains undetermined (Ivors et al. 2004). Introduction of species to new ranges is one of the most pervasive impacts of human activities on ecological communities. While some of the most devastating invaders are plant pathogens (Anderson et al. 2004), these introductions may be something of an exception to the rule. On average, when plant species are introduced to new habitats, surveys of host–pathogen associations indicate that they leave the vast majority of their pathogens behind (Wolfe 2002; Mitchell and Power 2003; Torchin and Mitchell 2004). Because most ecological communities are rapidly accumulating introduced plant species, the escape of introduced species from pathogens has important ramifications at the community level. It implies that species introductions are lowering the ratio of pathogen species to plant species within communities. This estimation needs further examination with data from the field, but if true, it may reduce the potential for the rich array of ecological effects of plant pathogens, to which we will now turn.

5.3 Impacts of pathogens on plant communities

In a recent review of plant pathogens in natural ecosystems, Gilbert (2002) concluded that pathogens can drive density-dependent plant population dynamics, cause rapid evolution in plant populations, mediate plant competition, accelerate plant community succession, and help maintain plant species diversity (e.g. Burdon 1991; Bever et al. 1997; Van der Putten and Peters 1997; Alexander and Mihail 2000; Packer and Clay 2000). We focus here on impacts of pathogens that were not emphasized in this and previous reviews (Augspurger 1987; Burdon 1987; Kranz 1990; Burdon 1991; Alexander 1992; Dickman 1992; Jarosz and Davelos 1995; Alexander and Holt 1998). In particular, most research on plant pathogens in natural communities, and thus most reviews of this topic, have focused on their evolutionary ecology (Gilbert 2002), rather than on processes operating at the community level.

5.3.1 Specialist pathogens

Specialist pathogens can influence community structure by decreasing the abundance of their host
species, particularly if the host species is a dominant member of the community when relatively free of disease. Such effects of pathogens can be revealed by changes in other ecological processes. For example, global change may alter plant–pathogen interactions in ways that could fundamentally re-shape plant communities. Perhaps the best example of this potential to date is a series of studies examining the effects of nitrogen deposition on the understory vegetation of Swedish forests. Increased rates of fertilizer production and fossil fuel burning result in the volatilization of reactive nitrogen compounds in the atmosphere, where they can be transported for thousands of kilometers (Vitousek et al. 1997; Galloway and Cowling 2002; Matson et al. 2002). In Sweden, the experimental addition of nitrogen nearly quadrupled prevalence of infection of leaves of the shrub Vaccinium myrtillus by the fungal pathogen Valdensia heterodoxa (Fig. 5.3(c); Stengebom et al. 2002). Within 3 years of starting the treatment, Vaccinium abundance declined by one-third, and the grass Deschampsia flexuosa also nearly quadrupled in abundance in disease foci (Fig. 5.3(b) and (a), respectively; Stengebom et al. 2002). A subsequent experiment indicated that this shift in community structure occurred because the disease reduced competition for light from the shrub (Stengebom et al. 2004). Furthermore, a national survey of the abundances of the plant species and the pathogen found the same patterns along geographic gradients of nitrogen deposition rate (Stengebom et al. 2003). These multiple lines of evidence suggest that increased disease due to nitrogen deposition is causing the conversion of forest understory from shrub-dominated to grass-dominated, a fundamental shift in species composition that could have dramatic impacts on processes from nutrient cycling to animal habitat use.

5.3.2 Generalist pathogens

Several studies of beach dune vegetation have shown that soil organisms, particularly pathogenic nematodes, can accelerate plant community succession by negatively affecting dominant early successional species (de Rooij-van der Goes 1995; Van der Putten and Peters 1997). However, these studies have primarily focused on the population dynamics of a single such species, Ammophila arenaria (marram grass). A recent study of grasslands has extended understanding of pathogen effects on plant succession to the community level (De Deyn et al. 2003). Inoculating experimental plant communities with soil biota from successional

Figure 5.3 Vegetation responses to the three nitrogen treatments in a Swedish boreal forest (squares, control; triangles, 12.5 kg N ha⁻¹ year⁻¹; circles, 50 kg N ha⁻¹ year⁻¹) over the 3 years in terms of the abundance of D. flexuosa, V. myrtillus and disease prevalence of V. heterodoxa on V. myrtillus leaves. Vertical bars denote ± 1 SE (n = 6), where large enough to be visible. Reprinted from Stengebom et al. (2002).
grasslands revealed that the soil microfauna, especially parasitic nematodes, inhibited growth of early- and mid-successional species and facilitated growth of late-successional species, increasing the rate of succession. Also, as a result of suppressing the otherwise dominant early-successional species, inoculation with soil microfauna increased the evenness of the plant community. This study suggests that pathogens may control the tempo of succession, maintain plant diversity, and aid in community restoration.

Aquatic community dynamics can also be driven by pathogens. While almost unstudied until the late 1980s, evidence has since been rapidly accumulating that viruses can control the dynamics of phytoplankton communities in both marine and freshwater systems. Initially, viruses were recognized and manipulated primarily as a filtered size-class, limiting the potential to understand species-level dynamics, but recent methodological advances are beginning to bring them out of the black box (Culley et al. 2003). Viruses can regulate individual phytoplankton populations, and mediate competition among species (Brussaard 2004). In the broader community context, viruses often increase in abundance during phytoplankton blooms, thus accelerating succession of phytoplankton, zooplankton, and bacteria populations (Park et al. 2004). As a result of these activities, aquatic viruses are major regulators of food web structure and nutrient dynamics (Fuhrman 1999; Wilhelm and Suttle 1999).

5.4 Feedbacks between plant and pathogen communities

Given the substantial evidence that plant community structure and pathogen spread can impact one another, we now turn to dynamic feedbacks that can result when such effects occur reciprocally. Several related conceptual models exist for such interactions (Janzen 1970; Connell 1971; Holt and Lawton 1994; Bever 2003). However, empirical work is still limited, perhaps due to the logistical challenges of simultaneously manipulating pathogens and plant community structure. The most venerable conceptual model is the Janzen–Connell hypothesis for the maintenance of species diversity. The model predicts that specialist natural enemies, including pathogens, act as density-dependent regulators of plant populations, decreasing species dominance and maintaining diversity. Evidence that pathogens can drive Janzen–Connell dynamics in both high diversity tropical forests (the original focus) and other plant communities is substantial (Gilbert 2002).

More recently, Bever (Bever 1994, 2003 Bever et al. 1997) has developed a conceptual and mathematical model for feedbacks between plant species and soil biota, including pathogens. This model assumes that plant species differentially influence the functional composition of the soil biota (e.g. abundances of pathogens versus mutualists), and that plant species respond differentially to changes in the composition of the soil biota (e.g. the relative effects of pathogens and mutualists differ among plant hosts). Thus, local growth of a plant species fosters changes in the soil biota that result in either greater performance (positive feedback) or decreased performance (negative feedback) of that species relative to competitors. Negative feedback is predicted to maintain local species diversity. Available evidence indicates that feedback on plant species’ growth is commonly negative, and can be caused by pathogens, but the consequences for community diversity and composition remain largely untested (Bever 2003).

Pathogen spillover (described above) also creates the potential for feedback to host community structure. When pathogen spillover decreases the performance of the focal host population, this is the process of apparent competition (Holt 1977; Holt and Lawton 1994; Alexander and Holt 1998) (Box 5.2). Apparent competition is so called because it is phenomenologically the same as direct competition (one population decreases performance of another), but is mediated by the two populations’ interactions with the shared natural enemy (Fig. 5.4), not the mechanisms of direct competition, such as resource reduction. Apparent competition is a positive feedback in that a population can promote its own growth by inhibiting competing populations via their shared enemy, so it has the potential to decrease community diversity. It thus contrasts with the Janzen–Connell hypothesis and Bever’s model of plant–soil feedback, both of which
Box 5.2 Detecting apparent competition among plant species

Apparent competition (Holt 1977) is an indirect effect involving at least two host species and one shared enemy (Fig. 5.4). Its magnitude can be estimated through a variety of approaches, all of which have advantages and disadvantages that make them suitable for different study systems and circumstances. First, theoretical models may be used to elucidate apparent competition. Greenman and Hudson (Hudson and Greenman 1998; Greenman and Hudson 2000) have developed an intriguing analytic approach called gateway analysis, which is used to understand the equilibrium dynamics of systems such as multiple-host-parasite models that are too algebraically complex for standard mathematical analyses. While promising in its generality, as yet it has been little applied. A more common method is to develop a dynamic model of the interacting species and parameterize it with data from the field, literature, inoculation experiments, or a combination thereof (Gilbert et al. 2001; Tompkins et al. 2003). A major advantage of this approach is its ability to predict the equilibrium outcome of the interaction, but it is limited by the assumption that dynamics of the system are driven solely by the species in the model (typically just two hosts and one parasite). In systems with long-term observational time series data, temporal correlations among host abundances and prevalences can be used to infer the importance of apparent competition and other forms of interaction (Begon et al. 1999). This approach is strong in that it integrates all interactions operating within the community, but difficult to implement because few studies have been run for sufficiently long periods to generate the required data.

Finally, there are numerous experimental approaches to test for apparent competition, which generally involve independent manipulations of apparent competition and direct competition. First, when one host species is suspected to be the main reservoir for the shared pathogen, its direct and indirect effects on the rest of the host community can be dissected by factorially constructing communities with/without the suspected reservoir species (through selectively removing that species, adding it, or planting communities de novo) and with/without the pathogen (Grosholz 1992; Power and Mitchell 2004). The difference in the effect of the reservoir on the other host species with and without the pathogen is the effect of apparent competition. Smaller-scale field experiments can be used to detect the effects of apparent competition on individual plants of a focal species by planting them with/without neighbors of the hypothesized reservoir species. This approach was recently implemented for salt marsh forbs sharing a beetle herbivore (Rand 2003), and could be applied to pathogens expected to be transmitted chiefly between nearest neighbors, such as many soil pathogens. Both of these approaches only allow assessment of the effects of the hypothesized reservoir species on the others. To elucidate all of the pairwise direct and indirect effects in a community, an appropriate experiment would include monocultures of each species and all possible bicultures, each with/without the shared pathogen. This design would allow collection of data to directly parameterize simple models of apparent competition, and thereby predict the equilibrium outcome. This design could be expanded to a multispecies context using the “combined-monocultures” approach, which can also be further elaborated to partition the effects of direct competition into root and shoot competition (Rajaniemi et al. 2003). The combined-monocultures approach would also allow more direct assessment of apparent competition by constructing plots which allowed apparent competition, but not direct competition. In contrast, the previous approaches quantify the magnitude of apparent competition by subtraction. Finally, a simple approach recently employed to detect parasitoid-mediated apparent competition among insects (Morris et al. 2004), might also be extended to plants. In this experiment, the only manipulation was experimental removal of the putative reservoir species, the effect of which was interpreted as the effect of apparent competition because the reservoir and focal host were known not to compete directly. In circumstances where two plant species are known not to compete directly (e.g. because of habitat specificity or phenology), this approach could be employed.

Figure 5.4 Apparent competition in a simple community module consisting of two host species (circles) and a shared pathogen species (square). Here, host species A can indirectly suppress host species B by acting as a reservoir for the shared pathogen.
emphasize negative feedbacks that limit the abundance of individual species, promoting diversity at the community level. However, these theoretical predictions are all for well-mixed or spatially homogenous communities. In spatially heterogeneous habitats, positive feedbacks that operate on a restricted spatial scale can promote diversity at larger spatial scales (Bever et al. 1997).

Although apparent competition is predicted to be a common phenomenon (Holt 1977; Holt and Lawton 1994; Alexander and Holt 1998), it has rarely been demonstrated in field experiments (Grosholz 1992; Rand 2003; Morris et al. 2004), especially with plant pathogens (Alexander and Holt 1998; Gilbert 2002; Power and Mitchell 2004). A recent field experiment in the Netherlands raised the possibility that parasite-mediated apparent competition can play a key role in community resistance to invasion by introduced species (van Ruijven et al. 2003). In an experiment that manipulated the species diversity and composition of a grassland community, one of the best predictors of resistance to invasion was presence of the species Leucanthemum vulgare (Asteraceae). This was noteworthy because Leucanthemum had very low biomass in monoculture. In contrast, other species that increased community resistance to invasion had large biomass in monoculture, suggesting they decreased invasibility through direct competition. Moreover, the abundance of root nematodes was several times higher in communities including Leucanthemum, suggesting that nematode-mediated apparent competition contributed to community invasion resistance. If this was the case, then pathogen spillover and apparent competition provided a positive feedback at the community level, maintaining abundance of resident species and decreasing abundance of invaders. However, nematodes were not experimentally manipulated, so other factors could have been responsible for the effect (van Ruijven et al. 2003).

Another recent field experiment independently manipulated plant community species composition and pathogen prevalence, thereby confirming the occurrence of apparent competition. As discussed above, this experiment factorially manipulated presence of barley yellow dwarf virus, presence of a highly susceptible plant species (A. fatua, wild oats), and soil nitrogen availability, and found strong virus spillover from Avena to the three other planted species (Power and Mitchell 2004). Moreover, pathogen spillover from Avena resulted in negative effects of apparent competition on aboveground biomass of two of the three other species, Digitaria sanguinalis (hairy crabgrass) and Lolium multiflorum (Italian ryegrass), although not S. lutescens (yellow foxtail). By providing a positive feedback on its abundance, apparent competition served to maintain Avena as a dominant species in the community despite being the most heavily infected. Thus, this experiment suggests that apparent competition can be a key determinant of biotic community structure.

5.5 Interactions with other components of biotic communities

Interactions between pathogens and other microbes or animal herbivores can have important impacts at scales from the leaf to the ecosystem (Blomqvist et al. 2000; Olff et al. 2000; Arnold et al. 2003; Silliman and Newell 2003). Starting at the leaf scale, Arnold et al. (2003) recently documented that infection of leaves of the tropical tree Theobroma cacao ("cacao," the source of chocolate) by naturally occurring assemblages of fungal endophytes reduced leaf mortality caused by a fungal pathogen by two-thirds. Fungal endophytes of woody angiosperms spread within plant leaves without obvious external symptoms. They are evolutionarily distinct from endophytes of grasses (Clay and Schardl 2002). Many are closely related to pathogens, and were not previously thought to have a defensive function against pathogens, although they can reduce insect damage (Faeth and Hammon 1997a,b). A survey of natural infections of cacao by endophytes revealed that they were present in close to 100% of mature leaves, and endophyte species diversity was astonishingly high—344 unique morphotaxa were isolated from 125 leaves. While previous studies have emphasized interactions among pathogenic and nonpathogenic microbes on leaf surfaces (Lindow et al. 2002), these results show that community-level interactions within plant leaves can also be important determinants of pathogen impact.
At the scale of a grassland community, recent field experiments suggest that complex interactions between herbivores and pathogens can drive plant community dynamics. In the Netherlands, digging by rabbits and ants decreased abundances of root-infecting parasitic nematodes, and allowed Carex arenaria to dominate (Blomqvist et al. 2000; Olff et al. 2000). In undisturbed areas, these nematodes strongly inhibited growth of Carex favoring growth of the other dominant species, Festuca rubra. Unidentified soil pathogens were also implicated in patchy declines of Festuca. Over 17 years, this complex web of interactions (Fig. 5.5) resulted in a spatio-temporal mosaic of dominance by these two species. Local sites within the grassland cycled between dominance by Carex and Festuca. Cycles in each site were out-of-phase with other sites, creating a perpetually shifting pattern. Such dynamics have been predicted by spatial simulations of Bever’s plant-soil feedback model (Molofsky et al. 2002). Further, the key role of rabbits and ants illustrates how plant-pathogen interactions are embedded within a broader web of interactions within a community.

As well as inhibiting pathogens, herbivores can facilitate their growth or transmission, even directly cultivating them in a mutualistic attack on the plant. Preliminary grazing by snails in a salt marsh increased leaf infection by pathogenic fungi (“probably primarily ascomycetes in the genera Phaeosphaeria and Mycosphaerella”) (Silliman and Newell 2003). Snail fecal pellets increased fungal growth via a fertilizer effect. The snails then fed preferentially on infected tissue, indicating that they were essentially farming the fungal pathogens. Factorially removing snails and fungi revealed that when snails and fungi acted in concert, they reduced plant biomass to only one-third of what it would be in their absence. Interestingly, the pathogens were responsible for all but half of the biomass reduction in the presence of snails, but did not reduce biomass in the absence of snails (Fig. 5.6). Effects of the pathogens were strictly contingent on the presence of their snail “farmers.” Thus, snails and pathogens jointly regulated net primary

![Figure 5.5](image)

**Figure 5.5** Proposed interaction web depicting the possible relationships between the two investigated plant species (F. rubra and C. arenaria), soil-borne pathogens, and organisms causing soil disturbances (rabbits, ants). Thicker arrows depict larger effects. Solid arrows indicate direct effects, dashed arrows indicate indirect effects. The direct negative effect of Festuca on Carex (competitive dominance in the absence of pathogens) was not investigated, but is postulated on the outcome of pilot experiments. Reprinted from Olff et al. (2000).

![Figure 5.6](image)

**Figure 5.6** Interactive and separate effects of snail presence and fungicide on the total length of grazer-induced wounds per stem (A), fungal biomass (micrograms of ergosterol (erg.) per square centimeter of leaf blade) on green leaves (B), and Spartina aboveground biomass (C). Different letters denote significant pairwise differences in mean values at $P = 0.05$ as determined from Tukey’s post hoc test. Error bars represent ± SE. Reprinted from Silliman and Newell (2003).
productivity, showing that community level interactions between pathogens and other organisms can have major consequences at the scale of entire ecosystems.

5.6 Conclusions

Interactions between a focal plant species and a focal pathogen species are embedded in a broader community of species, many of which can influence the plant–pathogen interaction. Although there are some cases in which plant–pathogen dynamics can be sufficiently understood without reference to this broader community, in many cases—particularly with generalist pathogens—consideration of community-level processes will be essential for understanding dynamics within any single plant–pathogen system. Specifically, we have seen that plant–pathogen interactions can be modified by competing plant species, reservoir plant species, microbial symbionts of plants, and animal herbivores. Thus, parasitism, competition, mutualism, and herbivory will all potentially interact in ecological communities, arguing for approaches that integrate these interactions.

Expanding the focal system from a single host and pathogen to a multispecies complex or the community as a whole opens up a wealth of interactions with potentially profound ecological consequences, both for pathogen dynamics and for community structure. This potential for linked disease and community dynamics is perhaps best illustrated by generalist pathogen spillover from reservoir species to more resistant species, and resulting apparent competition. The range of possible community-level dynamics involving specialist pathogens is somewhat more limited than with generalist pathogens, but pathogen suppression of otherwise dominant host species can have large repercussions. Also, the available evidence suggests that specialists and generalists are sensitive to different aspects of host community structure. Specialist pathogens appear more sensitive to host community species richness, and generalist pathogens more sensitive to the species composition of the host community. However, changes in richness and composition are linked, and so both specialist and generalist pathogens are likely to be impacted by changes in plant community structure. Finally, several studies have shown that plant community disease dynamics can be driven by global change, either in terms of abiotic variables such as nitrogen availability, or biotic variables such as species diversity and composition. The increasing pace of human-induced environmental alterations (Vitousek et al. 1997) suggests that the influence of global change on community disease dynamics will only increase.

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References


