CHAPTER TWELVE

Effects of Pathogens on Terrestrial Ecosystem Function

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Summary

Many studies have demonstrated that pathogens can have strong effects on the performance of individual organisms, population dynamics, and community interactions. A more limited set of studies suggests that pathogens can alter a wide range of ecosystem functions in terrestrial systems; however, we are lacking a framework to predict the type and magnitude of ecosystem effects that a given pathogen will have. In this chapter, we present a number of general principles that determine how pathogens influence ecosystems over time, based on the well-developed fields of disturbance ecology and the ecosystem effects of species composition. Our focus is not only on pathogens as disturbances but also as drivers of ecosystem processes, even when their presence is not readily apparent.

Introduction

Pathogens are infectious biological agents that alter the normal functioning of their hosts. Many studies have demonstrated that pathogens can have large impacts on host physiology, population dynamics, and community composition (reviewed in Burdon 1991; Gilbert 2002; Kranz 1990; see also Collinge et al., chapter 6, Clay et al., chapter 7, and Lafferty, chapter 9 this volume). Based on what is known about organisms' effects on ecosystems, many pathogen-induced changes in host populations, communities, and traits are dramatic enough that we would expect to see an accompanying change in ecosystem dynamics. However, relatively few studies have documented the consequences of pathogens on ecosystem function. These studies have demonstrated that epidemics affecting dominant plant species can alter a wide range of ecosystem functions, including net primary productivity (Kranz 1990; Spedding and Diekmahns 1972), hydrology (Bari and Ruprecht 2003; Batini et al. 1980; Hobara et al. 2001), decomposition (Cromack et al. 1991; Waring et al. 1987), nutrient cycling (Matson and Boone 1984; Waring et al. 1987), nutrient export (Hobara et al. 2001; Ohte et al. 2003), erosion

(Graniti 1998; Johnson and Wilcock 2002), and disturbance regimes (Dickman 1992). Even when there are no obvious visible signs of pathogen-induced damage or mortality, pathogens can substantially alter ecosystem processes. For example, pathogens can substantially decrease belowground production without any change in aboveground production (Agrios 2005; Mitchell 2003).

Although these studies have demonstrated that plant pathogens can alter ecosystem function, we have relatively little ability to predict the type and magnitude of ecosystem effects caused by any particular pathogen. In this chapter, we present some general principles to help account for the ecosystem impacts of pathogens. We discuss how these principles can be used to predict the ecosystem impacts of plant pathogens that act as disturbance agents and as key but invisible players in the everyday functioning of ecosystems. We also explore the ecosystem impacts of pathogens that infect organisms other than plants. Finally, we address when it is critical to consider pathogens in order to understand and manage ecosystems.

Predicting the Ecosystem Effects of Plant Pathogens: General Principles

The ecosystem effects of pathogens are largely mediated by the impacts of pathogens on their hosts. A number of key factors determine the ecosystem impacts of plant pathogens. These are briefly described below and discussed in more detail later in the chapter:

(**1**) *Pathogen impact on host survival, physiology, behavior, and reproduction*. Pathogens vary in their ecosystem impacts, depending on the host tissues they attack and whether they act as "killers, debilitators, or castrators" (Burdon 1991). The magnitude of the impact largely depends on host susceptibility, which can vary across species and across individuals within a species (Henry et al. 1992; Remold 2002).

(**2**) *Life stages of a host vulnerable to a pathogen*. Specific pathogens usually act on only a subset of plant life stages, or act differently on distinct life stages of a host (Agrios 2005; Castello et al. 1995; Kranz 1990). Whether a pathogen attacks its host in an immature or mature phase determines its ecosystem effects and our ability to notice the impact of pathogens on hosts and ecosystems.

(**3**) *Proportion of individuals/biomass infected at a site*. The proportion of individuals or biomass infected is largely a function of the susceptibility of hosts in relation to the specificity of pathogens. A highly host-specific pathogen can have large ecosystem impacts when it infects

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dominant or keystone species (discussed by Collinge et al., chapter 6, this volume) but may have no ecosystem impact in a diverse host community that is largely resistant to the pathogen. In contrast, a diverse host community can be destroyed by a generalist pathogen (e.g., *Phytophthora cinnamomi* can infect many different kinds of plants in Australian jarrah forests) (Weste 2003; Weste et al. 2002).

(**4**) *Spatial extent and distribution of infection*. The type and intensity of ecosystem change induced by a pathogen are strongly influenced by how large an area it infects and the distribution of infection. For example, if a pathogen were to kill 30% of all trees in a watershed, the ecosystem effects would likely differ if the infected individuals were all clumped in one area versus being evenly spread throughout the watershed.

(**5**) *Rate of pathogen effects on hosts in relation to rate of response or recovery by hosts or individuals replacing the hosts*. Pathogens can vary greatly in the rates at which they influence hosts. Similarly, the composition of host communities can greatly influence the rate of vegetation recovery in response to pathogen-induced damage or mortality of plants. In a host community that is diverse in its susceptibility to a pathogen, a slowly progressing disease will likely have minimal short-term ecosystem impacts, since neighboring plants will rapidly fill in space made available from dying branches and roots. In contrast, a pathogen that causes rapid mortality of many overstory individuals will likely have large ecosystem impacts, since replacement of the canopy will rely on seedlings or saplings.

(**6**) *Functional similarity of infected individuals vs. replacements*. Many pathogens substantially alter host community composition and structure (see Clay et al., chapter 7, Collinge et al., chapter 6, and Lafferty, chapter 7, this volume). A substantial body of literature has shown that ecosystem processes can be greatly affected by shifts in plant species composition and plant diversity (reviewed in Eviner and Chapin 2003; Hooper et al. 2005), suggesting that pathogen-induced shifts in host communities can impact ecosystems. Even when there is no shift in species composition, pathogens can replace mature individuals with juveniles, and the plant traits that influence ecosystem processes can vary greatly within a species across its life stages (reviewed in Marschner 1995).

(**7**) *Frequency and duration of pathogen impact*. Over the long term, the ecosystem impacts of a pathogen are strongly influenced by the duration and return interval of infection. When a pathogen persists over the long term at a site, it may continuously exclude the former dominant hosts (e.g., chestnut blight, *Cryphonectria parasitica*) (Jaynes and Ellis-ton 1982; Paillet 1988). As with other disturbances, the frequency with which a pathogen recurs at a site can have large impacts on ecosystems through its effects on host community composition and recovery of

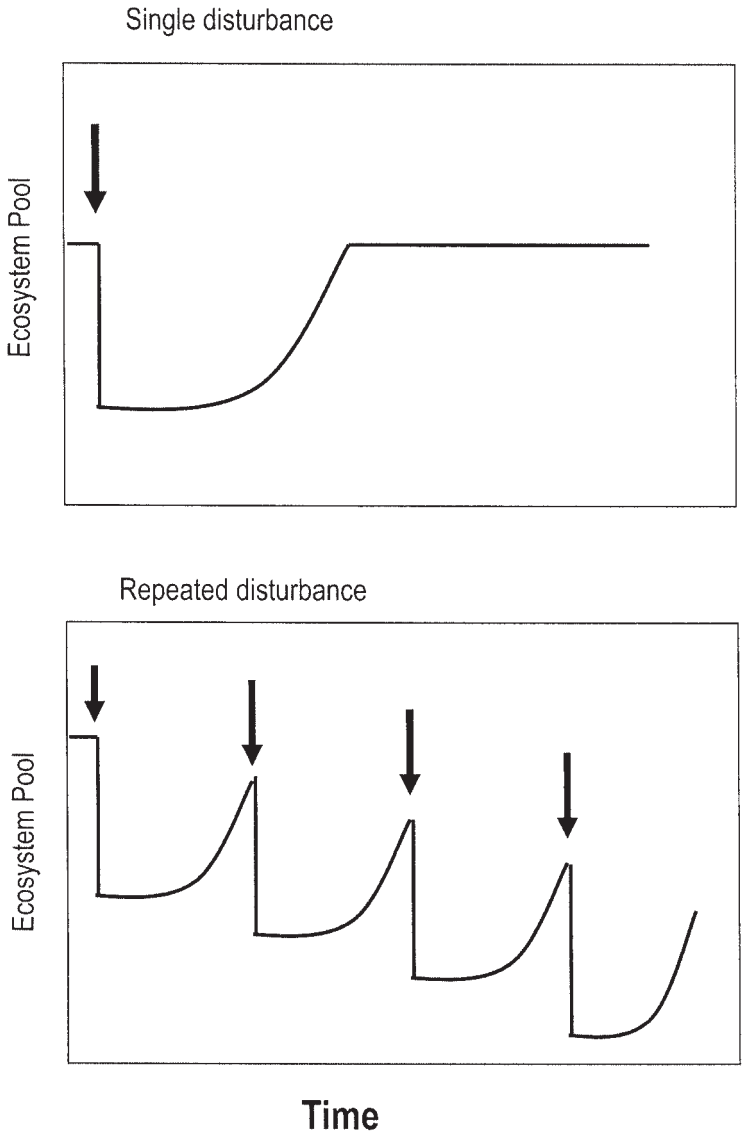

Figure 12.1. Response of ecosystem pools (e.g., ecosystem carbon and nitrogen, plant biomass) to disturbance. Arrows indicate time of disturbance. (a) An ecosystem disturbance results in an immediate decrease in biogeochemical pools, which slowly recover with time after disturbance. (b) The frequency of disturbance can lead to long-term changes in ecosystem pools, particularly when the disturbance recurs before the ecosystem pool fully recovers from the previous disturbance. (Figure adapted from Vitousek and Reiners 1975, Bormann and Likens 1979, Chapin et al. 2002.)

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vegetative biomass, ecosystem structure, and biogeochemical fluxes and pools (figure 12.1) (Bormann and Likens 1979; Chapin et al. 2002; Vitousek and Reiners 1975).

In this chapter, we explore how these seven factors contributes to a mechanistic understanding of the short- to long-term ecosystem effects of pathogens, focusing primarily on plant pathogens.

Plant Pathogens as Disturbances

Pathogens are often viewed as disturbance agents, organisms that disrupt the normal functioning of other organisms or ecosystems. Most studies that have investigated the ecosystem effects of pathogens have focused on large-scale epidemics that kill mature individuals, and these stand-replacing epidemics have been compared with other disturbance events such as clear-cutting (Hobara et al. 2001; Waring et al. 1987). In general, a stand-level disturbance can result in an initially large ecosystem change as a result of losses of carbon and nutrient stocks from the ecosystem. These pools return to their predisturbance baseline over time as the vegetation recovers (figures 12.1a, 12.2a) (Bormann and Likens 1979; Loucks 1970; Matson and Boone 1984; Waring et al.1987).

The key lesson to be learned from the disturbance framework is that the ecosystem effects of pathogens change over time. We outline four key stages of pathogen effects on ecosystems (figure 12.2):

- *Impact on host*. The initial impacts of a pathogen on ecosystems are largely determined by how it impacts its hosts (e.g., shifts in traits, mortality). For many pathogens, their effects on ecosystems change during this impact stage, as infection progresses from a debilitating phase to a killing phase.
- *Initial response*. The plant community usually responds during or shortly after pathogen disturbance of hosts, and the ecosystem effects of pathogens are strongly influenced by which community members respond and how rapidly they respond. This initial community response is often determined by plants already occupying the site.
- *Midterm response*. The midterm response is characterized by the second generation of establishment after pathogen disturbance.
- *Long-term response*. Whereas the three previous stages are transitory in nature, the long-term effects of pathogens are

characterized by whether the ecosystem recovers to predisturbance conditions or is set on a new trajectory.

The classic disturbance curve (figure 12.2a) is based on a stand-replacing event mortality of a large portion of the vegetation over a relatively short time. This disturbance would be followed by the rapid establishment of a plant community. Only a subset of pathogens would be expected to mirror the ecosystem effects of the classic disturbance curve (e.g., acute pathogen outbreaks that rapidly kill most plants in a given area). In many cases, the ecosystem effects of pathogens do not follow the classic disturbance curve, for a number of reasons:

1. The long-term effects of pathogens can be strongly influenced by the frequency at which they disturb plant communities. A single disturbance event can require a given amount of recovery time before biogeochemical pools and fluxes return to predisturbance levels (see figure 12.1a). If a pathogen were to return repeatedly before the ecosystem recovers, it would likely set the ecosystem on a new trajectory (figure 12.1b) (Aber and Melillo 2001; Bormann and Likens 1979; Chapin et al. 2002).
2. The short- to long-term ecosystem impacts of pathogens are largely mediated by the rate of recovery of vegetation biomass and whether this recovery involves shifts in plant species composition. When the individuals that replace the killed hosts are functionally different from the hosts, large changes in ecosystem function and structure can occur (dashed lines in figure 12.2b). For example, beech bark disease (*Nectria coccinea* or *N. galligena*) in forests of the northeastern United States can lead to gradual shifts in community composition that, in turn, alter nutrient cycling (Lovett et al. 2007). As beech (*Fagus grandifolia*)–sugar maple (*Acer saccharum*) communities shift to maple dominance, nitrification significantly increases, because sugar maple stimulates nitrification rates. Similarly, in beech-hemlock (*Tsuga canadensis*)–dominated sites, shifts from beech to hemlock lead to lower rates of nitrogen cycling and nitrogen loss (Lovett et al. 2007).
3. In some cases, there may be a time lag between plant species shifts and changes in ecosystems (dotted line in figure 12.2b). For example, in southeastern Alaska, a 3.8-fold increase in landslides occurred fifty years after large-scale mortality of yellow cedars (*Chamaecyparis nootkatensis*). Even though cedars were killed relatively rapidly, the effects of their roots on slope stability

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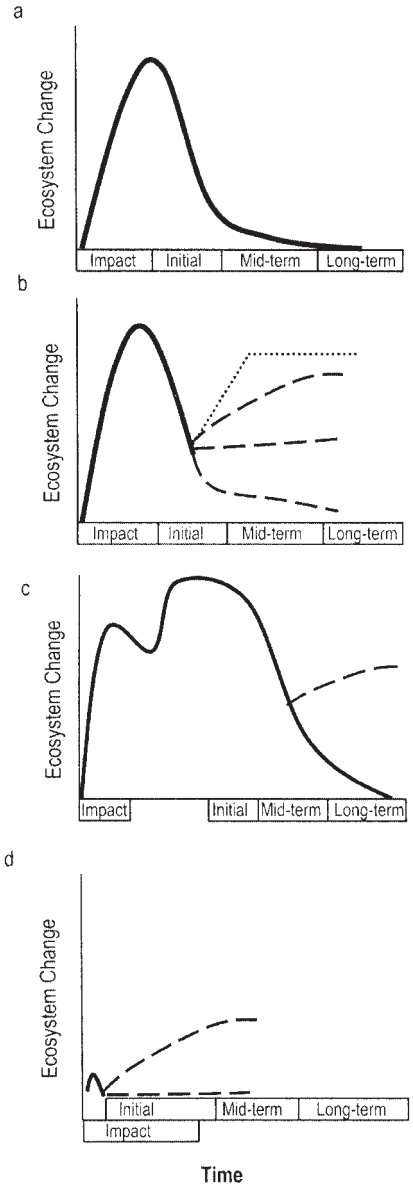

Figure 12.2. (a) The classic disturbance curve, where ecosystem processes initially change greatly in response to disturbance and then gradually recover to predisturbance levels. (b) Mid- to long-term effects of pathogens on ecosystems are determined by the composition of the plant community that reestablishes at a site after hosts are killed by pathogens (dashed lines). The effects of pathogens on ecosystems over time are also influenced by the persistence of the ecosystem effects of hosts, even after they are killed. Once these legacy effects wear off, the ecosystem can be set on a new trajectory (dotted line). (c) A lag time between

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persisted for decades, until the roots finally decomposed (Johnson and Wilcock 2002).

4. Departures from the standard disturbance curve are also due to variations in the duration of each impact/response stage and the extent to which they overlap (figure 12.2). For example, *Phytophthora cinnamomi* can kill 50%–75% of overstory and understory species in Australian jarrah forests, sometimes resulting in land that is totally devoid of vegetation for a number of years (Weste 2003). In addition to the initial ecosystem change associated with disturbance, this time lag between mortality and recovery can increase the susceptibility of an ecosystem to larger changes, such as massive erosion events or continued mineralization of soil carbon and nitrogen (figure 12.2c). Midterm to long-term impacts are again largely influenced by which plant species dominate in the community over time. For example, in the most intensely affected areas of jarrah forest, forests were completely replaced by sedges and rushes (Weste 2003; Weste et al. 2002). Pathogens can determine which plant species establish over the mid- to long term by directly excluding host plants through persistent infection. Pathogen-induced ecosystem changes may also affect which plant species establish at a site. The classic disturbance curve also fails to account for the situation of pathogens gradually killing their hosts. In these cases there is often substantial overlap between the stages of pathogen impact and initial community response, resulting in minimal disturbance (figure 12.2d). For example, beech bark disease and Dutch elm disease (*Ophiostoma ulmi* or *O. novo-elmi*) often cause gradual mortality of individual trees within a mixed-species forest. As branches slowly die, neighboring overstory trees expand their own canopy, minimizing gaps (Agrios 2005). Chestnut blight causes a similarly slow die-off of individuals (Woods and Shanks 1959), but because the American chestnut was so dominant in many forests in the eastern United States, neighboring trees were often chestnuts that were also infected and could not compensate for the slow decline of neighbors, leading to small gaps in the

pathogen-induced host mortality and initial recovery by the community can lead to larger ecosystem changes than predicted based on the classic disturbance curve in a. Dashed lines as in b. (d) The ecosystem effects of disturbance by pathogens can be minimal when the impact stage overlaps with the initial recovery stage (e.g., when an individual gradually dies and neighbors immediately fill in the empty spaces in the canopy). Dashed lines as in b.

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forest. Such gaps can increase or decrease rates of nitrogen cycling, depending on the forest structure. For example, in one study, nitrogen cycling rates increased in forests with little or no understory, similar to classic disturbance scenarios (see figure 12.2a). However, in forests with an understory, the understory plants rapidly filled in the gaps created by the death of canopy trees (figure 12.2d), so that there was no disturbance-induced stimulation of nitrogen cycling (Mladenoff 1987).

Disturbances occur over a continuous range of spatial scales, ranging from single-plant gaps to large regional disturbances. Many pathogens act at scales intermediate to these, and others move from gap agents to large patch disturbances as the disease progresses (e.g., root rots) (Agrios 2005; McCauley and Cook 1980). It is unclear whether the ecosystem effects of pathogens increase linearly with infected patch size or whether there are some thresholds below which pathogen impacts on ecosystems are minimal and above which they are substantial. Batini et al. (1980) proposed that substantial shifts in water yield occurred once pathogens killed 20% of trees in a forest. It is also critical to consider how mortality is distributed within an area. For example, for a fixed area of pathogen infection in a landscape, the magnitude of ecosystem changes will likely be much higher when the infection is in one large patch compared to infection of isolated gaps randomly distributed throughout the landscape.

The well-developed fields of disturbance ecology and species effects on ecosystems provide a strong theoretical basis that we can use to better understand the ecosystem impacts of large pathogen-induced mortality events of mature individuals. In the next section we explore the more subtle roles of pathogens in ecosystems.

Pathogens and Ecosystem Dynamics: Beyond the Disturbance Framework

Ecosystem ecologists have largely focused on pathogens as disturbance agents; leave as is the mortality of mature individuals is an obvious occurrence that can have an impact on ecosystems. However, these highly visible effects of pathogens are a small subset of the roles that pathogens play in ecosystems. It is easy to overlook the role of pathogens in regulating host population and community dynamics through pathogen-induced changes in host traits or reproduction, or when mortality occurs in immature individuals and stands. A wide range of pathogens is present but often unnoticed in natural ecosystems, as indicated by agricultural studies,

which demonstrate that natural ecosystems commonly act as reservoirs for many crop diseases (reviewed in Power and Mitchell 2004). Kranz (1990) suggested that 58%–78% of disease cases in mixed plant populations go unnoticed. Is this because pathogens are present in low numbers in natural systems? Are the natural hosts less susceptible than crop hosts? Or is the presence of pathogens apparent if we were to look more closely at these systems? Undoubtedly there are individual cases that fall into any of these categories. For example, some studies have shown that pathogens are less likely to cause epidemics in natural systems (Dinoor and Eshed 1984), while others have demonstrated that pathogen impacts on natural systems can be as severe as in crop systems (Holah et al. 1993; Kranz 1990). In this section, we explore potential cases in which pathogens play a critical, but often overlooked role in the functioning of ecosystems. In contrast to considering how pathogen-induced disturbances alter ecosystems (see figure 12.2), this section explore how pathogens help to determine the *baseline* of ecosystem function, so that by removing pathogens, we would see changes in ecosystem function (figure 12.3).

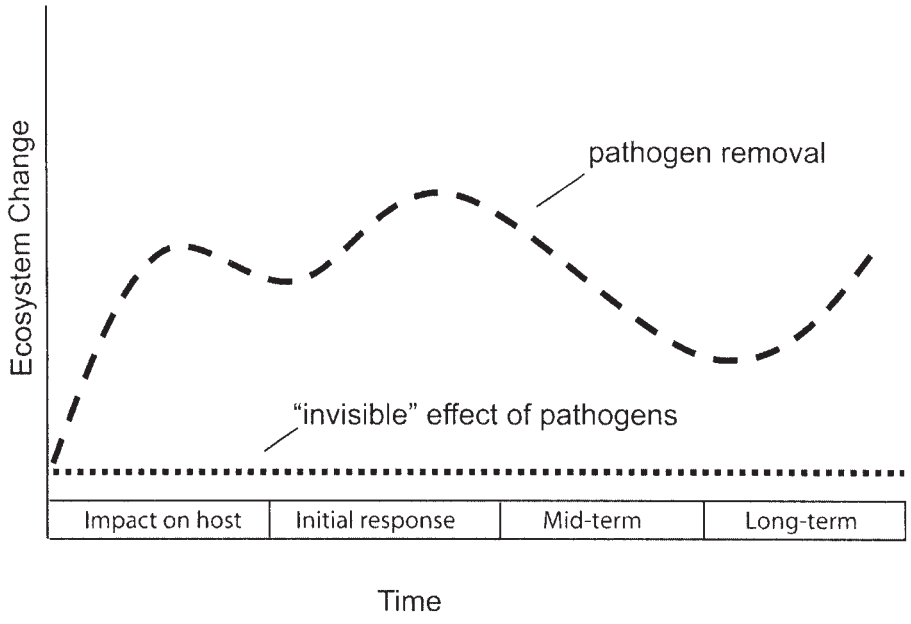

Figure 12.3. Pathogens can be critical players in the normal functioning of ecosystems. Their activity may be responsible for establishing the baseline of ecosystem function (dotted line), so that only on removal of a pathogen would a significant change in ecosystem functioning be apparent (dashed line).

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Pathogens and Self-Thinning

Although we often focus on disturbances caused by pathogen-induced mortality of mature plants, most pathogen-induced plant mortality occurs at the seedling stage (Gilbert 2002) and is mediated by generalist pathogens (Kranz 1990). Self-thinning of seedlings is a critical population and community process in most plant communities (Dewar 1993; Heady 1958; Puettmann et al. 1992; Sims and Coupland 1979; Westoby 1981), and pathogens can greatly increase thinning rates in herbaceous (Spedding and Diekmahns 1972; Paul and Ayres 1986; Malmstrom et al. 2007) and woody (Castello et al. 1995) ecosystems.

Although we are not aware of any studies that have demonstrated the ecosystem effects of pathogen-induced thinning, seedling thinning plays an important role in ecosystem dynamics. Since thinning occurs at peak plant growth and competition (Heady 1958), any nutrients released from dying individuals are likely to be sequestered in growing plants. This sequestration accounts for the observations that nutrient loss is minimal and nutrient use is optimal at the self-thinning stage of stand development (Bormann and Likens 1979; Midgley 2001). In California annual grasslands, self-thinning of seedlings provides as much plant-available nitrogen as does decomposition of plant litter senesced at the end of the growing season (Eviner and Vaughn 2007). In other systems, thinning of seedlings can actually enhance the establishment of surviving seedlings (reviewed in Gilbert 2002) by initially taking up space and resources, and releasing them to surviving seedlings later in the growing season (Smith 1977).

Pathogens Alter Species Composition

Pathogens have been shown to play large roles in structuring plant community composition and diversity (reviewed in Gilbert 2002; see also Collinge et al., chapter 6, Clay et al., chapter 7, this volume). Such changes in plant species composition (reviewed in Eviner and Chapin 2003) and plant diversity (reviewed in Hooper et al. 2005) can have large effects on ecosystem processes. For example, a crown rust (*Puccinia coronata)* can change a pasture dominated by ryegrass (*Lolium perenne*) to one dominated by clover (*Trifolium repens*; Burdon and Shattock 1980), and such shifts from a grass to a legume can have a large impact on the pools and fluxes of carbon and nutrients (Eviner et al. 2007). Similarly, in a stand of *Setaria viridis* and *Bromus hordeaceus,* barley yellow dwarf virus (BYDV) decreased the fitness of *S. viridis* but had no effect on *B. hordeaceus* (Remold 2002). *Setaria* is a C4 grass and *Bromus* is a C3 grass. Since C4 grasses tend to contain higher lignin concentrations and are less

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digestible than C3 grasses (Trlica 2005), BYDV likely increased rates of decomposition and nitrogen cycling in this system. Such species shifts are usually attributed to competition, and the roles of pathogens in mediating these species shifts are often overlooked.

In some ecosystems, pathogens are major drivers of plant successional dynamics (Holah et al. 1997; USDA 2004). Pathogens can speed succession by increasing the competitive ability of species in the next successional stage, or delay succession by shifting competitive dominance to early successional species or by decreasing nitrogen fixers in primary succession (Dobson and Crawley 1994; Holah et al. 1997). Pathogen-induced successional shifts from herbaceous to woody species in dunes (van der Putten et al. 1993) have the potential to significantly alter erosion rates, carbon storage, water fluxes, and nutrient dynamics (reviewed in Eviner and Chapin 2003).

Although it is clear that pathogen-induced changes in plant species composition can alter ecosystems, there are few data with which to evaluate how often these shifts in plant species result in changes to ecosystem functions such as productivity and nutrient cycling. Do pathogens tend to favor species that are functionally different from the plants they displaced? Lignin, phenolics, and other secondary compounds can be responsible for plant resistance to certain pathogens (Agrios 2005), and pathogen-induced shifts to species with high concentrations of these compounds could result in large changes in ecosystems because these substances can be major drivers of plant effects on biogeochemical cycling (Fierer et al. 2001; Hattenschwiler and Vitousek 2000; Schimel et al. 1996, 1998). Although pathogens have the potential to cause such functional shifts in plant communities, it is unclear if such shifts are common in natural ecosystems.

Pathogens Alter Plant Traits

Most of the discussion in this chapter has focused on pathogens as agents of mortality; however, host plants can live for many years before succumbing to pathogens, and many pathogens do not directly cause mortality. Pathogen-induced changes in plant physiology and morphology have been well documented in plants with commercial value (reviewed in Agrios 2005) but are more difficult to detect in natural systems, owing to the large variation in plant traits across individuals in wild populations (Remold 2002). Pathogens (particularly viruses) can induce morphological changes in plants, such as stunting, dwarfing, brooming, and changes in tillering rates (Agrios 2005). Such changes in structure can influence microclimate and habitat for other key organisms in the ecosystem (reviewed in Eviner and Chapin 2003). Many

pathogens cause extensive root damage or decreases in root versus shoot allocation (Agrios 2005; Holah and Alexander 1999; Mitchell 2003), without an accompanying change in aboveground biomass (Agrios 2005; Mitchell 2003). Such declines in root biomass can decrease rhizosphere stimulation of soil biogeochemical cycles, decrease root contributions to soil carbon and nutrient storage, and increase susceptibility for soil erosion and landslide events (reviewed in Eviner and Chapin 2003). Decreases in root biomass can also increase susceptibility to windthrow (Robbins 1988) and mortality by repeated grazing (Smith et al. 1986). On the flip side, pathogen-induced increases in tillering can increase the ability of grasses to withstand heavy grazing (Kranz 1990).

Pathogens can also cause large changes in plant tissue chemistry, which is a key regulator of plant effects on carbon and nutrient dynamics (Hobbie 1992; Hattenschwiler and Vitousek 2000; Fierer et al. 2001). In many plants, infection by pathogens induces secondary compounds with strong antimicrobial properties, including lignin, tannins, gums, phenolics, alkaloids, isoflavanoids, terpenoids, and proteins with antimicrobial activity (e.g., thionins, chitinase) (Belanger et al. 2003; Cao et al. 2001; Daayf et al. 2003; De Ascensao and Dubery 2000; Ishimoto et al. 2004; Krischick et al. 1991; Pegg and Ayres 1988; Ramamoorthy et al. 2002; Saunders and O'Neill 2004; Thangavelu et al. 2003; Thipyapong et al. 2004; Witzell and Shevtsova 2004; reviewed in Agrios 2005). For example, pathogens have been shown to increase lignin concentrations by 26% in alfalfa (*Medicago sativa*) (Lenssen et al. 1992), phenolics by 25%–200% in bilberry (*Vaccinium myrtillus*) (Witzell and Shevtsova 2004), and soluble phenolics by 30-fold in potato tubers (*Solanum tuberosum*) (Agrios 2005). In general, pathogens are less susceptible to these compounds than nonpathogenic fungi (Agrios 2005), suggesting the potential for large changes in microbially mediated biogeochemical cycling. These compounds can decrease feed quality, palatability, and digestibility (Clarke and Eagling 1994; Lewis et al. 1996), change soil microbial community structure (Rumberger and Marschner 2003), and alter carbon and nutrient cycling (Fierer et al. 2001; Hattenschwiler and Vitousek 2000; Schimel et al. 1996, 1998). For example, isothiocyanates decrease ammonium oxidizer populations and inhibit nitrification rates (Bending and Lincoln 2000).

Pathogens as Castrators

Pathogens can cause significant decreases in seed yield even without appreciable effects on living biomass (Spedding and Diekmahns 1972). Hudson and Dobson (1995) have hypothesized that pathogens that re-

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sult in host mortality can stabilize host population density, while pathogens that reduce fecundity can result in population crashes. However, when the host species has a substantial seed bank, the composition of the community could be resilient to a temporary decrease in seed yield. When boom and bust cycles do occur, they have the potential to alter ecosystem processes when the susceptible host population is replaced by species with different functional attributes.

From Case Studies to General Principles

A number of case studies have demonstrated that pathogens can play important roles in mediating ecosystem dynamics, but pathogens are rarely included in conceptual models of ecosystem function. This absence is likely due to the lack of a generalizable, predictive framework to assess the types and magnitude of ecosystem impacts of pathogens. Some ecologists have integrated pathogens into ecosystems through their role as disturbance agents. However, it is critical to move beyond the largely artificial designation of pathogens as disturbances versus part of natural systems because we are biased in how we categorize pathogen activities. For example, we can recognize the visibly obvious signs of dieback of individual trees in forests, but tend to overlook the same process in grasslands, where it can be nearly impossible to track individual plants. The seven general principles outlined at the beginning of this chapter provide a basis to build a framework that moves beyond the artificial designation of pathogens as disturbances or intrinsic parts of ecosystems. For example, when we use the seven principles to compare pathogen-induced self-thinning to pathogens that cause disturbances (as small and large gaps) (table 12.1), the only difference is the life stage of hosts that the pathogens infect. It is likely that thinning is viewed as an invisible effect of pathogens rather than as a disturbance because ecologists don't tend to track individuals in intensely crowded juvenile stands, and tend to notice deaths only when they leave empty spaces in the canopy. The seven general principles allow for a more ecologically meaningful categorization of the activity of pathogens that will strengthen our ability to predict the types and magnitude of ecosystem effects that pathogens can have. For example, ecologists are more likely to recognize the role of pathogens in shaping community dynamics in forests than in grasslands because the signs of individual infection and mortality are far more visible in a forest. Recognizing that these processes are similar allows us to integrate our understanding of interactions between infected individuals, neighbors, and biogeochemical

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TABLE 12.1
Comparison of different roles of pathogens according to the seven general principles that determine the impact of pathogens on ecosystems

Attribute	*Large Gap*	*Small Gap*	*Thinning*	*Competition*	*Succession*	*Change in Host Traits*
Impact	Mortality	Mortality	Mortality	Mortality: shifts in physiology, behavior	Mortality	Shift in physiology, behavior
Life stage	Mature	Mature	Juvenile to immature	Juvenile to mature	Immature to mature	Juvenile to mature
% Infected	High	Low	High	Low to high	Medium to high	Low to high
Spatial concentration	High	Low	Medium	Low to high	Low to high	Low to high
Timing						
Effect on host	Slow to rapid	Slow	Rapid	Slow to Rapid	Slow to rapid	Slow to rapid
Recovery	Slow	Rapid	Rapid	Rapid	Rapid	—
Likelihood of functional change	Low to high; species shift	Low to high; species shift	Low to medium; species shift	Low to high; species shift	Low to high; species shift	Low to high; shift in effect of the same species
Frequency of recurrence	Low to medium	Medium to high	Medium to high	Medium to high	Low to high	Low to high
Ecosystem functions:						
Nutrient retention	*	****	*****	Depends on species shift	Depends on species shift	Depends on trait shift
Nutrient turnover	****	**	*****			
Pathogens	Fungi, nematodes	Fungi	Fungi, bacteria	Fungi, bacteria, viruses	Fungi	Viruses, fungi, nematodes, bacteria

* indicates degree of impact.

fluxes that are easily documented in forests, with ecosystem-scale measures of yield and input/output budgets that are more feasibly studied in grasslands.

Linking the characteristics in table 12.1 to predictions of pathogen effects on the magnitude and type of ecosystem impact will require further investigation into a number of areas. These include:

- What is the relative importance of each of these factors in determining pathogen impacts on ecosystems? Do they vary by ecosystem and stage of ecosystem development? By pathogen type?
- How do interactions among these factors determine ecosystem dynamics?
- Are there tendencies for some of these factors to covary with one another?
- How often do different ecosystem processes covary versus vary independently in their response to these multiple factors? For example, the rates of nutrient retention and nutrient turnover are both very high in self-thinning stands, but in stand-replacing events, nutrient retention is low, while nutrient turnover remains high (see table 12.1).
- Do the ecosystem effects of pathogens vary depending on the type of pathogen? Most of the plant disease literature focuses on fungal pathogens, likely because 10,000 fungal species have been identified as plant diseases, compared with 100 bacterial species (Agrios 2005). But the impacts of fungi on host plants can be very different from the impacts of other pathogens such as viruses (Agrios 2005).

Understanding ecosystems also requires considering the interactions between different pathogens, and between pathogens and other biotic and abiotic factors. A large amount of plant damage and mortality is a result of a complex of diseases and their interactions with insects and abiotic disturbance agents (Castello et al. 1995; Worrall and Harrington 1988).

Ecosystem Impact of Nonplant Pathogens

Thus far we have focused on plant pathogens; however, pathogens of herbivores, predators, and decomposers can also cause large-scale

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changes in ecosystem function (Agrios 2005; Briggs et al. 1995; Lovett et al. 2007; Prins and van der Jeugd 1993; Read 2003; Middelboe, chapter 11, this volume). The most dramatic ecosystem effects of animal and insect pathogens occur through changes in vegetation composition. For example, the decline of impala populations due to anthrax led to shrub encroachment in northern Tanzania (Prins and van der Jeugd 1993). In the Serengeti, the decline of herbivores due to rinderpest increased the fuel load, which stimulated fires and led to the decline of the bush (reviewed in Prins and van der Jeugd 1993). Pathogens can be key regulators of insect outbreaks (reviewed in Briggs et al. 1995), allowing for the persistence of plants that are susceptible to insect attack. The decline of rabbits due to myxomatosis led to the development of an oak woodland in England (reviewed in Dobson and Crawley 1994) and significantly increased the establishment of native species in Australia (Read 2003). Rabbits not only decimated vegetation in the Australian outback, they also produced burrows, provided food for predators, increased soil turnover, and affected water penetration and runoff. Because some native Australian mammals are now rare or extinct, some activities such as soil digging are not replaced when rabbits decline due to myxomatosis or rabbit hemorrhagic disease, and ecosystem processes vary in space and time due to disease-induced fluctuations in rabbit populations (Read 2003). Decomposer organisms are also likely affected by pathogens. Pathogens can retard decomposition by limiting decomposer populations and can stimulate decomposition by inducing turnover of microbial biomass. For example, viruses that infect soil bacteria could potentially increase rates of carbon and nutrient cycling by stimulating bacterial turnover, as occurs in marine systems (Middelboe, chapter 11, this volume).

How Well Can We Predict Ecosystem Processes Without Considering Pathogens?

Although pathogens may mediate thinning, succession, and competition in many systems, do we need to explicitly understand the roles that pathogens play when these population and community processes are often predictable and repeatable? Why should we further complicate ecosystem models by explicitly incorporating the role of pathogens, rather than just looking at host population and community dynamics? Global change studies have demonstrated that we must account for the response of pathogens to a changing environment in order to understand host physiological, population, and community responses. For example, the presence of pathogens influences plant community

response to nitrogen additions in boreal forests (Strengbom et al. 2002) and host physiological response to elevated CO_2 (Malmstrom and Field 1997).

As is discussed in the following chapter, pathogen-induced changes in disturbance regimes, such as increased susceptibility to fire (Castello et al. 1995), windthrow (Worrall and Harrington 1988), or erosion (Wondzell 2001), could lead to state changes in the ecosystem that would not be predictable simply from host population dynamics. Some of the most significant ecosystem effects of pathogens are determined by human management of ecosystems to prevent disease and in response to pathogen outbreaks. For example, salvage logging in response to tree pathogen outbreaks accelerates soil erosion because of the increased disturbance of vegetation and soil associated with harvesting and transporting timber (Wondzell 2001). Similarly, the heavy use of antibiotics in humans and livestock has increased the prevalence of antibiotic-resistant microbes in natural systems (Burgos et al. 2005; Kolpin et al. 2002; Lateef et al. 2005; Mallin 2000). Since microbially produced antibiotics play a role in structuring microbial interactions (Burgess et al. 1999; Davelos et al. 2004; Zvenigorodskii et al. 2004), antibiotic resistance can lead to shifts in microbial community composition that can alter microbially mediated biogeochemical processes (Vaclavik et al. 2004).

We may also need to explicitly include pathogens in ecosystem models because they can represent a major pool of carbon and nutrients in many ecosystems (Mitchell 2003; Smith et al. 1992; Lafferty, chapter 9, this volume). The C and nutrients in pathogen pools may differ from other ecosystem pools in terms of how they respond to seasonal and environmental changes, their turnover times, and how labile they are. They also may drive changes in other ecosystem pools (e.g., plant or soil pools). Thus, explicitly including pathogen carbon and nutrient pools will increase our understanding of biogeochemical pools and fluxes in some ecosystems.

Conclusions

Whether their presence is readily apparent or not, pathogens play a key role in mediating many ecosystem processes. In order to integrate pathogens into our conceptual understanding of ecosystems, it is critical to consider how pathogen impacts will vary over time. Ecologists need to move beyond case studies to generalizations that can be used to predict the type and magnitude of ecosystem impacts of a pathogen. The seven key principles discussed in this chapter can guide generalizations of ecological response to disease, but ultimately, the largest

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ecosystem changes will arise from how humans manage ecosystems in response to disease.

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