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EVOLUTIONARY ECOLOGY OF PLANT DISEASES IN NATURAL ECOSYSTEMS

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■ **Abstract** Plant pathogens cause mortality and reduce fecundity of individual plants, drive host population dynamics, and affect the structure and composition of natural plant communities. Pathogens are responsible for both numerical changes in host populations and evolutionary changes through selection for resistant genotypes. Linking such ecological and evolutionary dynamics has been the focus of a growing body of literature on the effects of plant diseases in natural ecosystems. A guiding principle is the importance of understanding the spatial and temporal scales at which plants and pathogens interact. This review summarizes the effects of diseases on populations of wild plants, focusing in particular on the mediation of plant competition and succession, the maintenance of plant species diversity, as well as the process of rapid evolutionary changes in host-pathogen symbioses.

INTRODUCTION

Plant pathogens play Jekyll and Hyde roles in the structure, dynamics, and evolution of natural plant communities. As destructive agents, plant pathogens can cause mortality, reduced fitness of individual plants, rapid declines of populations of particular host species, or dramatic shifts in the structure or composition of plant communities. At the same time, pathogens can help maintain plant species diversity, facilitate successional processes, and enhance the genetic diversity and structure of host populations. Understanding the impacts of diseases in natural plant communities requires integrating numerical dynamics, rapid evolutionary changes, and spatial structure of both host and pathogen populations, and an appreciation for how destructive actions at one scale can be the foundation for positive outcomes at another.

There have been a number of important reviews of plant diseases in natural ecosystems. Early contributions included the original summons to plant ecologists by Harper in 1977 (120), the first significant review of the field by Dinoor & Eshed in 1984 (84), and Burdon's seminal *Diseases and Plant Population Biology* in

1987 (46). The following decade saw a number of important reviews on various aspects of plant diseases in natural communities (10, 14, 29, 48, 52, 82, 140). Here I expand upon and update what has already been said.

In this review I address three broad areas of the role of plant diseases in natural plant ecosystems. First, I look briefly at how pathogens with different life histories affect wild plants at different stages in their life cycles, focusing on effects of reduced survivorship, growth, and fecundity that could influence numerical dynamics of the host. Second, I look at diseases that affect host population dynamics or that affect plant community diversity, structure, and dynamics by mediating plant competition or successional processes. Finally, I explore some of the rapid evolutionary changes associated with plant diseases in natural ecosystems, particularly looking at patterns and consequences of local adaptation. In examining these themes, I try to integrate existing theoretical frameworks with empirical examples from the literature. Whenever possible, examples come from studies in natural ecosystems, but in some cases, I refer to experimental studies in agriculture or model systems to indicate what we might expect to find in natural systems. Finally, for each broad question, I point out key areas in which critical data are lacking. Perhaps because of early development of theory for the role of plant diseases in high diversity systems, there is an unusual depth to the literature of diseases in natural communities in the tropics, and that is reflected in this review.

HOST MORTALITY, GROWTH, AND REPRODUCTION

Pathogens can affect host population dynamics through direct effects on the survival, growth, and fecundity of individual plants. Examples in agricultural systems pervade the literature, and although documentation of direct effects outside of crop plants is rarer, a wide range of pathogen types clearly affect wild host plants in many of the same ways. Plants killed by disease before reproducing do not contribute to the next generation. Additionally, because fecundity is usually correlated with plant growth and size, diseases that affect growth are likely to reduce reproductive output, as do pathogens that directly attack flowers and developing fruits. Because the impacts of diseases depend on the life stage of the plant that is attacked and the life history of the pathogen, I have categorized examples of wild plant diseases as (*a*) seed decay, (*b*) seedling diseases, (*c*) foliage diseases, (*d*) systemic infections, (*e*) parasitic plants, (*f*) cankers, wilts, and diebacks, (*g*) root and butt rots, and (*h*) floral diseases. Similarly, I categorized the effects of the diseases on (*A*) host survival, (*B*) growth, or (*C*) fecundity. In Table 1 I use this cross-categorization to summarize a collection of representative studies of the effects of diseases on wild plants. The discussion is organized to follow the life cycle of a plant, and I highlight some important themes and emphasize disease systems seldom included in discussion of diseases in natural ecosystems.

TABLE 1 Representative studies of the direct effects of plant diseases on individual plants in natural systems

	Type of effects on host		
Plant stage	**Survival**	**Growth**	**Fecundity**
Seed decay	(73–75, 146, 156, 160)	N.A.	N.A.
Seedling diseases	(16, 25, 27, 28, 30, 92, 142, 170, 222)	?	N.A.
Foliar diseases	(13, 76, 77, 164, 165, 182, 214, 231)	(85, 88, 144, 154, 216)	(13)
Viruses, viroids, and phytoplasmas	(11, 13, 67, 70, 140, 188, 204, 221, 224)	(70, 94, 112, 116, 140, 188, 190–193, 205, 224)	(9, 58, 65, 94, 140, 185, 186, 188)
Parasitic plants	(19, 34, 161, 218)	(161, 202, 218)	(194)
Canker, wilt, and dieback diseases	(1, 20, 21, 43, 77, 96, 100, 102, 114, 131, 141, 147, 159, 178, 180, 195, 196)	(99, 102, 131)	?
Root diseases and butt rots	(82, 83, 117, 125, 181, 189, 223)	(18, 41)	(167)
Floral infections	N.A.	N.A.	(8, 9, 12, 15, 17, 58, 140, 207, 217, 227)

N.A. = not applicable, ? = no examples found, but effects are probable.

Seed Decay

The highest rates of disease-related mortality of plants in natural systems are usually due to seed and seedling diseases. Fresh seeds are susceptible to fungal infection, but rates of attack are often affected by handling by frugivorous animals. Seeds of the common forest tree *Strychnos mitis* (Strychnaceae) in Uganda suffered 88% mortality, primarily from fungal attack, although when fruits were first handled by frugivorous monkeys fungal attack was nearly eliminated (146). In contrast, fungal attack of seeds of fresh blueberries (*Vaccinium angustifolium*, Ericaceae) in Nova Scotia increased dramatically when fruits were first consumed and passed by birds (74).

Loss of soil seed bank to fungal attack ranged from 10% in the invasive shrub *Mimosa pigra* (Mimosaceae) in tropical Australia (156) to 47% and 39% annual mortality of the seed bank of two tropical pioneer tree species in a lowland moist tropical forest in Panama (75), and from negligible to more than 90% mortality for seeds of five plant species in the soil of Wyoming shrub-steppe (73). In general, impacts of seed pathogens may be greatest for plant species that rely heavily on periodic disturbances for regeneration from long-lived seed banks, including

forest-gap-specialist pioneer species and fire-released species from prairie and chaparral habitats. However, a major lacuna in research remains the effects of pathogens on seeds in the soil.

Seedling Diseases

Damping-off diseases of seedlings are probably the most widely studied lethal diseases of plants in natural communities. Augspurger and coworkers (25, 27, 28, 142) first brought damping-off to the attention of plant ecologists with a series of studies of seedling mortality in the tropical forest of Barro Colorado Island (BCI) in Panama. Damping-off affected 80% of tested species (25) and was the primary cause of death for seedlings of six of nine tree species, killing up to 74% of a parent tree's annual reproductive output (28). Most studies of damping-off in natural ecosystems have been done in tropical forests, but Packer & Clay (170) found that black cherry seedlings (*Prunus serotina*, Rosaceae) in Indiana suffered up to 65% mortality from *Pythium*.

Environmental heterogeneity can have strong effects on the development of damping-off caused by Oomycetes like *Pythium* and *Phytophthora*. For seedlings of several tropical trees, damping-off was more severe in soils with blocked drainage (92) or in areas of deep shade (25, 28, 222) than in drier or sunnier areas. Such effects of environmental heterogeneity are common for damping-off caused by Oomycetes, which rely on water-filled soil pores for zoospore motility.

Foliar Diseases

Foliar pathogens, including the fungi, bacteria, and viruses that cause spots, necrosis, chlorosis, shot-hole, early senescence, and leaf abscission, all reduce leaf area and associated photosynthetic activity that would otherwise contribute to plant growth and reproduction. For seedlings in particular, foliar diseases may significantly increase mortality, since the probability of seedling survival is strongly affected by seedling size (104). Defoliation of *Larix* (Pinaceae) by the needle-cast fungus *Mycosphaerella laricinia* reduced seedling radial growth, root biomass, and new shoot production in the following year (144). Although foliar diseases may sometimes kill plants directly, reduced plant size from foliar or other diseases also places affected seedlings at a competitive disadvantage to larger, healthier neighbors (220). This disadvantage may be most important in light-limited environments, such as forest understories or dense grasslands.

Foliar diseases can also affect growth and reproduction of larger plants, although they probably rarely kill them. Fungal pathogens accounted for 34% of leaf damage on the leaves of 10 tree species in a lowland tropical forest in Panama, and were the leading cause of damage on 4 of the host species (35). Two thirds of host species and 43% of leaves surveyed in a Mexican tropical rain forest were damaged by fungi, but leaf area damaged was on average less than 1%, and always less than 20% (98). Similarly, foliar infection of seedlings in forest fragments in the central Amazon

was less than 2% (38). The petiolar pathogen *Phylloporia chrysita* caused 52% reduction in growth rates on infected individuals of the tropical tree *Erythrochiton gymnanthus* (Rutaceae) in Costa Rica (88).

Effects of foliar plant pathogens are not restricted to terrestrial plant communities. Marine slime molds in the genus *Labyrinthula* cause wasting disease in a wide range of seagrasses worldwide (214). *Labyrinthula* infection of turtlegrass (*Thalassia testudinum)* causes necrotic lesions on the leaves and has dramatic effects on photosynthetic efficiency (85). Wasting disease decimated populations of eelgrass (*Zostera marina*) in the early 1930s, and again in the 1980s and 1990s on both the eelgrass (164, 165) and turtlegrass (182, 231). Temperature, light, and sediment nutrients all strongly influence production of defensive phenolic compounds in seagrasses (45, 213, 215), and some authors suspect that outbreaks may be related to changes in environmental conditions (213). Recently, Jackson postulated that the ultimate cause of wasting outbreaks may have been the much earlier ecological demise of green sea turtles, which were dominant grazers on seagrasses (133). Loss of the dominant herbivores may have released turtlegrass populations from top-down population regulation, resulting in dense host populations and altered nutrient cycling, making *Thalassia* vulnerable to an outbreak of the endemic pathogen *Labyrinthula*.

Systemic Infections

In 1990, Harper made a plea for increased work on the role of viruses in natural plant communities (120). However, despite the recognition that viruses, viroids, and phytoplasmas cause systemic and persistent infections in a wide range of host plants, their importance in natural communities has still received much less attention than other pathogens. There are numerous reports of wild plants as reservoirs for phytoplasmas, viruses, and viroids that affect economically important crops (60, 90, 127, 137, 145, 148, 157), but the effects of these infections on the wild hosts are rarely studied (112, 116, 191, 193). With the increased availability and portability of PCR-based and immunologically based assays for phytoplasmas and viruses, our understanding of their role in plant population dynamics outside of agriculture should increase dramatically.

Some fungi and Oomycetes cause systemic infections with severe effects. Systemic infection of seedlings of Shepherds Purse, *Capsella bursa-pastoris* (Brassicaceae), by *Albugo candida* or *Peronospora parasitica* caused up to 88% mortality (13), and the systemic smut *Urocystis trientalis* reduced survival of *Trientalis europaea* (Primulaceae) by 50% (221).

In contrast, systemic infections of grasses by fungal endophytes are often viewed as important mutualisms because some endophytes protect the host plant from herbivory and increase the plant's competitive ability (67, 70, 224). Such antiherbivory benefits to the host plant may not be universal, however (205), and in many cases host growth, competitive ability, and reproduction suffer directly from endophyte infection in the absence of herbivory [see reviews in (66, 140, 188)].

Parasitic Plants

Dwarf mistletoe (*Arceuthobium* spp., Viscaceae) is often a major pathogen of coniferous trees, causing reduced host growth (161, 202), increased mortality (19, 34, 161), and reduced cone and seed production (194). Dwarf mistletoe can cause up to 65% reduction in growth in severely infected Douglas-fir (*Pseudotsuga menziesii*) individuals, and mortality in severely infected stands was three- to fourfold greater than in healthy stands (161). Watson has provided a recent review of the ecology of mistletoes (218).

Cankers, Wilts, and Diebacks

In the past century, North American forests have suffered devastating effects from introduced diseases including chestnut blight (20), Dutch elm disease (100, 147), pitch canker of Monterey pine (114), beech bark disease (131), white pine blister rust (195), and recently, sudden oak death (96). These epidemics were particularly dramatic due to the widespread, rapid death of mature host trees when the pathogens girdle them or otherwise interfere with vascular transport. Sudden oak death, caused by *Phytophthora ramorum*, has killed tens of thousands of true oaks (*Quercus* spp.) and tanoaks (*Lithocarpus densiflorus*) (Fagaceae) in California since its appearance in 1994 (96, 180). The known taxonomic host range of *P. ramorum* is growing rapidly, with infections detected in at least five host genera outside of the Fagaceae (180). Sudden oak death threatens to transform oak woodlands of the Pacific coast of the United States, as well as oak-dominated systems elsewhere. Important canker and vascular diseases are not necessarily caused by introduced pathogens. The endemic Hawaiian tree koa (*Acacia koa*), a keystone species in upper-elevation forests, suffers from a dieback disease caused by the systemic wilt pathogen *Fusarium oxysporum* f. sp. *koae* (21). Epidemics of dramatic consequence may have prehistoric precedent. Fossil evidence from England indicates there was a large neolithic decline of elms, thousands of years before the Dutch elm disease epidemic of recent years (110, 177). However, such dramatic impacts of introduced diseases may become increasingly frequent phenomena with growth of international trade, precipitating the need for strengthened policies on the movement of biological materials (113).

Root and Butt Rots

Root and butt rots of trees can cause significant mortality of mature forest trees. The introduced pathogen *Phytophthora lateralis* has caused severe mortality on the California-Oregon endemic Port-Orford Cedar (*Chamaecyparis lawsoniana*, Cupressaceae) (117), and *Phytophthora cinnamomi* introduced to the Jarrah (*Eucalyptus marginata*, Myrtaceae) forests of Western Australia caused widespread destruction of Jarrah and dozens of other susceptible host species (82, 189, 223). The native fungi *Phellinus weirii* and *Heterobasidion annosum* cause high mortality in conifer forests in western North America (83, 125, 181). The mortality caused

by all these pathogens is particularly notable because it affects large, established trees, and death of dominant species can lead to dramatic shifts in forest structure. Such effects are not new—there is some evidence for epidemic tree mortality in the Late Triassic. A relatively thin, but widespread stratum in the Petrified Forest National Park in Arizona shows that certain trees were damaged by decay similar to present-day damage caused by *H. annosum* (72).

Root rots are not always lethal, but may reduce host growth or reproduction. In British Columbia, *Armillaria ostoyae* reduced radial growth of Douglas-fir by up to 60% relative to healthy trees (41). Similarly, *H. annosum* reduced mean annual radial growth of loblolly pine (*Pinus taeda*) by 36% (18).

Floral Diseases

The most direct effect of disease on plant fecundity, short of killing the host, is the attack of flowers or developing fruits, thereby preventing fruit production. Few floral diseases have been studied in natural systems, but available data suggest that floral diseases may have large effects on host fecundity. In the Appalachian Mountains of the eastern United States, flower galls of flame azalea (*Rhododendron calendulaceum*, Ericaceae) caused by *Exobasidium vaccinii* led to a 50% reduction in flower number, as well as reduced fruit production (227). Similarly, attack of flowers and ovaries of the tropical understory tree *Faramea occidentalis* (Rubiaceae) by the rust *Aecidium farameae* reduced fruit set by 75% (207). The herb *Plantago lanceolata* (Plantaginaceae) infected with the floral pathogen *Fusarium moniliforme* var. *subglutinans* were more likely to be male-sterile, and had reduced seed production compared to healthy plants (12).

By far, however, the best known of floral diseases in wild plants is the sexually transmitted, pollinator-vectored anther smut (*Microbotryum violaceum*) of *Silene* spp. (Caryophyllaceae). The cost of infection to host fitness is great, since the fungus replaces stamens and staminoids with spore-bearing structures (8). Infected populations produced lower densities of seedlings (58), but infection caused significant host mortality only in years of mild winters (204). Differential susceptibility to anther smut across host genotypes, and strong effects on host reproduction combine to link numerical effects of disease on the population with evolutionary responses through selection for resistant genotypes (17). [See excellent reviews in the evolutionary ecology of this disease system by Jarosz, Alexander, and coworkers (17, 140)].

In a bizarre interaction, systemic infection of *Arabis* spp. (Brassicaceae) by the rust *Puccinia monoica* inhibits flowering and causes a radical morphological change in the host plant, with clusters of infected leaves creating pseudoflowers of similar appearance to unrelated co-occurring flowers (185). Pollinators attracted to the pseudoflowers fertilize the rust. The rust blocks sexual reproduction in the host plants and can also affect reproduction in nonhost neighbors. The increased density of showy pseudoflowers can attract additional pollinator visitation locally, but may reduce pollinator efficiency when pollinators spend time “pollinating” pseudoflowers (186).

Recent years have witnessed significant growth in the number of studies of plant diseases in natural ecosystems. Nevertheless, the number of empty or nearly empty cells in Table 1 indicates the need for studies on a diversity of pathogen types and their effects on various stages in the host life cycle.

POPULATION DYNAMICS, SPECIES INTERACTIONS, AND DIVERSITY

The past two decades have seen an expansion in our appreciation of the ecological dynamics of plant diseases in natural communities. Three interrelated themes have emerged as leading areas of ecological inquiry: (*a*) the importance of plant diseases in density-dependent population dynamics, (*b*) the importance of compensatory and cross-generational effects, and (*c*) the direct effects of pathogens on shifting competitive interactions among plant species, effects on plant community succession, and the maintenance of plant species diversity.

Plant Diseases and Density-Dependent Population Dynamics

Density dependence is important to all aspects of plant demography from seed production and germination to plant growth and mortality. The mechanisms underlying density-dependent responses include plant competition, seed-germination inhibition, seed predation, competition for pollinators, herbivory, and of course, diseases. Host density can in turn affect disease incidence and severity either through direct effects on host-pathogen encounter rates or through indirect effects on disease development by changing environmental factors (49). These effects on diseases are created through (*a*) changes in the number of hosts available for infection through space or time, (*b*) the distance between susceptible hosts and the probability of transmission of pathogen propagules among plants, (*c*) effects of competition on host vigor, (*d*) frequency-dependent effects on vector behavior or herbivore damage, (*e*) effects on the density and composition of associated plant species in the community, and (*f*) the physical environment in which the pathogen and host interact.

In their classic 1982 review, Burdon & Chilvers (49) evaluated studies on 46 host-pathogen combinations for patterns of density-dependent disease incidence. Fungal diseases overwhelmingly showed positive density dependence. The few cases of negative density dependence were associated either with short-term effects where primary inoculum was limiting at high host densities (as for some soilborne pathogens), or with heteroecious rusts where lower densities of one host allowed the development of higher densities of the obligate alternate host plant. On the other hand, vector-borne viral diseases usually showed negative density-dependent incidence, either because infective vectors were limiting at high host densities or because vector behavior changed with respect to crop density. Although most of the examples reviewed were from managed settings, it is clear both that density-dependent disease development can be an important factor in disease ecology

anywhere, and that natural history traits of the pathogen are key to the outcome. Since 1982 there have been numerous publications on density-dependent disease development in natural systems congruent with the patterns described in the review (56, 58, 97, 121, 154) and few contrary examples (7, 62).

Counterweights to Numerical Effects

Disease can cause significant plant mortality, but it is only important to population dynamics if the reduced number of plants leads to decreased seed production at the population level. For example, local density of seedlings may be reduced through damping-off diseases, but if the remaining healthy plants respond to the reduced intraspecific competition by increased growth and reproduction, these compensatory responses can offset numerical losses (14, 16). In a field experiment, *Pythium* reduced both the number and average size of seedlings of the annual *Kummerowia stipulacea* (Papilionaceae), but at maturity plant size was largest in the plots with the highest initial disease incidence (16). The compensatory response of the survivors to reduced competition eliminated differences in seed production between diseased and nondiseased plots. Similarly, *Portulaca oleraceae* (Portulacaceae) compensated for disease loss when as much as 50% of a stand was infected with *Cucumber mosaic virus* (94). Even strong effects of diseases on fitness of individual plants may not affect numerical population dynamics. Research on the ability of diseases to regulate host population dynamics must focus not just on numerical responses in one generation, but should incorporate the compensatory responses to reduced intraspecific competition on neighborhood fitness.

Cross-generational effects may modulate the numerical response of subsequent host generations. Diseased maternal plants may produce inferior seed, reducing the potential fitness of the next generation of plants (139). In contrast, it is possible that defenses induced in maternal plants may be transmitted to the progeny, increasing resistance to disease in the next generation. This, in effect, allows the inheritance of increased disease resistance without selection for resistant genotypes. Although not yet explored in disease systems, such maternally transmitted resistance has been shown in response to herbivores (4).

In summary, the past two decades of research have shown that although plant diseases can and do have significant impacts on plant populations in natural ecosystems, predicting the effects of a particular disease requires far more than determining the effects on an individual plant. Compensatory responses in surviving plants, multigenerational variation in numerical responses, cross-generational induced resistance and effects on offspring vigor, and the dynamics of selection for more resistant host genotypes and more virulent pathogens all contribute to the effects of plant pathogens on the population biology of their hosts.

Competition, Succession, and Plant Diversity

Competitive interactions and host-pathogen dynamics may be strongly interdependent. Stress from competition for scarce resources can alter susceptibility

to disease so that bottom-up competitive interactions lead to increased top-down effects of pathogens. Similarly, differential susceptibility or tolerance to diseases can lead to different competitive interactions in the presence or absence of disease. Alexander & Holt (14) provide an excellent recent review on interactions between diseases and plant competition.

DISEASE AND COMPETITION Competition may stress plants and make them less tolerant to infection. The rust *Puccinia recondita* has a large impact on plant growth of *Impatiens capensis* (Balsaminaceae) at high natural densities but not at thinned densities (154). Thus, competition reduced host tolerance to rust infection.

Although differences in shade tolerance are usually attributed to physiological differences, the presence of disease may create differences among individuals in their abilities to compete for light. Survival of stump sprouts of *Salix viminalis* (Salicaceae) is directly related to their height and thus ability to compete for light. Infection by the rust *Melampsora epitea* greatly reduced shoot growth, and reduced the plant's competitive ability, ultimately killing infected stumps (216).

Specialist pathogens like rusts can affect the competitive interactions between susceptible and nonsusceptible plant species or genotypes. Paul & Ayres found that inoculation of groundsel (*Senecio vulgaris*, Asteraceae) with the rust *Puccinia lagenophorae* significantly reduced groundsel growth, and reduced the competitive ability of groundsel with both lettuce (*Lactuca sativa*) (175) and the co-occurring weed petty spurge (*Euphorbia peplus*, Euphorbiaceae) (174). Rust infection directly affected the competitive ability of the host beyond the effects of infection on plant growth. Similar effects of rust infection have been found for intraspecific competition, including between infected and noninfected groundsel (175) and between susceptible and resistant skeletonweed (*Chondrilla juncea*, Asteraceae) (51). Research of this sort is sorely needed outside of agroecosystems.

APPARENT COMPETITION Apparent competition in plants describes the situation where two species appear to be in competition for some limiting resource, but the negative effect is actually due to an indirect interaction via an herbivore or pathogen that attacks them both (126). Generalist pathogens may play an unrecognized role in interspecific interactions in natural communities, mimicking or modifying competitive interactions (14). An intriguing example of this exists between the annual legume *Chamaecrista fasciculata* (Caesalpiniaceae) and the large, dominant perennial grass *Andropogon gerardii* (Poaceae), which coexist in the Kansas tallgrass prairies. Holah & Alexander (123) found that both species grew more poorly in soil from the root zone of *Chamaecrista* than in *Andropogon* soil, and that the effect was associated with fungi found uniquely on *Chamaecrista* roots. Pathogenic fungi "cultured" on *Chamaecrista* roots shift the competitive outcome against the dominant perennial grass, facilitating coexistence of the two plant species. Understanding the host ranges of pathogens within a local plant assemblage and the possible adaptation by plants to actively culture pathogens that increase their competitive ability is a largely unexplored, but potentially fruitful field.

SOIL FEEDBACKS In other cases, host-specific microorganisms cultured in the rhizosphere of a host may build up over time, with increasingly negative biological effects on the host. Bever and coworkers (39, 40) developed the concept of feedback as a framework for the role of soil microbial communities in regulating plant population dynamics and plant diversity. Negative feedbacks—the buildup of detrimental pathogens over time—are widely recognized in agricultural settings, where crop rotation is a standard practice to escape the buildup of disease pressure. Crop rotation, when integrated over time, effectively increases the plant species diversity in a particular field. In natural communities, negative feedbacks could help maintain local plant diversity by preventing individual species from increasing to complete dominance. Bever (39) looked at the effects of soil microbial communities in soils "cultured" by one of four old-field species: *Krigia dandelion* (Asteraceae) and three grasses (*Danthonia, Anthoxanthum,* and *Panicum*). *Krigia* mortality doubled, and grass growth rate decreased when grown in their own soil compared to being grown in soil from any other species. For some species but not others, the effects were associated with accumulation of *Pythium* in the soil, indicating host-specific effects of the cultured pathogens (163). These studies suggest that the local development of host-specific soilborne pathogens may establish a temporally dynamic mosaic of deleterious "home" and benign "away" sites, and contribute to the maintenance of host diversity in grasslands. Such effects on plant diversity could be quite strong. Based on spatial lottery models, as long as there is no intrinsic correlation between host susceptibility and competitive ability, plant diversity should increase monotonically with increased addition of host-specific enemies (169).

Negative feedback loops may be important in many kinds of natural systems, but traits of pathogens and hosts that favor longer-term spatial structure are likely to be key elements. In particular, pathogen dispersal distances need to be more limited than host dispersal distances; thus many soilborne pathogens may be more likely to be involved in negative feedbacks than would wind-dispersed pathogens. Similarly, sexually reproducing plants with mobile seeds are more likely to escape from the fouled "home" sites than would primarily clonal hosts. Strongly dynamic systems with high levels of disturbances may not maintain enough spatial structure for feedbacks to develop.

DISEASES IN PLANT COMMUNITY SUCCESSION Diseases can cause changes in plant community composition through the dynamic patchworks of Bever's old-field communities, or through a predictable succession of seres. In Europe, marram grass (*Ammophila arenaria*, Poaceae) colonizes and thrives in the wind-blown sands of coastal foredunes, but declines once the dunes are stabilized. A complex of pathogenic soil fungi and nematodes develops in the *Ammophila* rhizosphere through negative biotic feedback (79–81, 210, 212), which causes the degeneration of the dominant *Ammophila*, and allows the resistant species, sand fescue (*Festuca rubra* ssp. *arenaria,* Poaceae), to ultimately dominate the stabilized dunes (211). In the northeastern United States, *Ammophila breviligulata* dominates the high beach zone and is associated with high densities of growth-reducing parasitic nematodes

(151, 152), but greenhouse studies suggest the development of mycorrhizal networks may be more important than negative feedback for plant species succession in this system (143, 151). Further, coordinated work on mycorrhizae, pathogenic fungi, and pathogenic nematodes in these parallel systems would be valuable in understanding variation in microbial control of plant successional processes.

Forest succession can also be driven by differential susceptibility to pathogens. In particular, dramatic changes in species composition can be driven by epidemics from introduced pathogens. Loss of American chestnut to chestnut blight in the 1910s to 1940s led to a peak in recruitment of *Quercus rubra*, *Tsuga canadensis*, and a suite of other species that now dominate forests in the eastern United States (2, 3, 5, 42, 93). Native pathogens can have similarly important effects on forest succession. *Phellinus weirii*, the cause of laminated root rot, affects most conifer species but with different degrees of severity. *Phellinus* spreads radially forming "infection centers," removing the overstory of highly susceptible Douglas-fir and mountain hemlock (*Tsuga mertensiana*) in its wake (82, 125). The removal of the overstory has major effects on the plant community, with local resistant species then becoming dominant in the infection centers (124). Hansen & Goheen (118) provide an excellent recent review of the importance of *Phellinus* in driving forest structure and successional processes in western conifer forests.

These two systems, soilborne pathogens in beach grasses and *Phellinus* in conifer forests, demonstrate the potential for native pathogens to drive succession in low-diversity plant communities, and to affect the spatial distribution of susceptible host species and their resistant competitors. In high-diversity plant communities, such as tropical rain forests, the effects of disease on the spatial patterns and coexistence of plant species may be more complex, and are brought together under the framework of the Janzen-Connell hypothesis.

THE JANZEN-CONNELL HYPOTHESIS The puzzle of how so many plant species can coexist in species-rich ecosystems like tropical rain forests has been the focus of a tremendous body of literature [see reviews in (63, 111, 229)]. Given a suite of species competing for a single resource, the competitive exclusion principle predicts that only one species, the competitive dominant, will persist. Processes that increase negative interactions within a species relative to negative interactions between species break the trajectory toward low diversity caused by interspecific competition, and are essential to stabilize species coexistence (63). Specialist natural enemies, including plant pathogens, herbivores, and seed predators, can stabilize coexistence in tropical forests by limiting populations of each species independently. Janzen (136) and Connell (71) brought into the ecological limelight a model first proposed by Gillett (109) in 1962, now known as the Janzen-Connell hypothesis. There was considerable empirical support for the Janzen-Connell hypothesis in tropical forests as early as 1984 (64), and it is still the leading conceptual framework for understanding how plant diseases affect forest community structure and diversity.

The Janzen-Connell hypothesis begins with the recognition that in natural forests, most seeds do not disperse far, but instead form a "seed shadow," so that

the seedlings in a forest are highly clumped around their mother tree. A parent tree could act as a reservoir for specialist pathogens or pests that are then transmitted to nearby offspring, and high densities of seedlings near to the parent tree would result in greater pressure from specialist natural enemies due to density-dependent attack. With proportionally greater mortality of seedlings close to the parent tree than at greater distances, intraspecific clumping should decrease through time, so that the spatial distribution of mature trees should be less clumped than expected through random mortality throughout the original seedling distribution.

The Janzen-Connell hypothesis also predicts that distance- and density-dependent mortality caused by specialist natural enemies should reduce interspecific competition. Seeds of nonsusceptible plant species that dispersed into the seed shadow of a susceptible species would then be at a competitive advantage over the more numerous progeny of the susceptible species. In a diverse forest with associated specialist natural enemies, individual trees would then have stronger negative effects on survival of nearby conspecifics than on heterospecifics. Rare species would be more likely than common species to find pathogen-free space, and thus enjoy a competitive advantage. Intraspecific apparent competition would then stabilize species coexistence and act to maintain tree diversity.

According to the mechanisms outlined in the Janzen-Connell hypothesis, for plant diseases to favor higher host species diversity, (*a*) the pathogens, if not host specialists, must have differential effects on different local host species; (*b*) there must be a diversity of pathogens with different host preferences; and (*c*) both hosts and pathogens must be dispersal limited (ubiquitous pathogens would not produce distance-dependent disease gradients except through distance-dependent environmental interactions). Specialist pathogens that are most likely to cause Janzen-Connell–like effects are those that (*a*) infect both mature and juvenile plants, but cause little damage to trees and have severe effects on seeds, seedlings, or saplings; (*b*) have strong density-dependent infection or impacts on hosts; or (*c*) have long-lived soilborne propagules that permit the local buildup of pathogen inoculum.

JANZEN-CONNELL EFFECTS IN TROPICAL FORESTS There is strong evidence that plant diseases affect rain forest diversity, dynamics, and spatial structure, as predicted by the Janzen-Connell model. First, there is overwhelming support for the importance of density-dependent mortality in shaping the diversity and dynamics of tropical forests (e.g., 119, 219, 225). The first empirical tests of the role of plant diseases for the Janzen-Connell hypothesis were by Augspurger and coworkers, as described above under Seedling Diseases (25, 27, 28). In a series of studies on BCI in Panama, they showed that damping-off diseases were important causes of seedling mortality for a range of tree species, but that susceptibility varied widely across host species (28). Seedlings were most likely to escape damping-off at greater distances from the parent tree when close to the parent, and the proportion of seedlings dying was greater in areas with higher seedling density (27, 142). For *Platypodium elegans* (Papilionaceae), the median distance of offspring to the parent tree increased from 15 to 31 meters from seed dispersal to surviving saplings, greatly reducing species clumping as compared to what would be expected from

random mortality (26). Thus, damping-off of seedlings in the lowland moist tropical forest of BCI shows all the predicted elements of the Janzen-Connell mechanism for the maintenance of diversity among tree species: strong distance-to-adult and density-dependent mortality with differential effects across host species, causing a temporal reduction in clumping of individual species. Direct effects on local maintenance of species diversity were not investigated. Recently, however, Harms et al. (119) followed seed rain and seedling recruitment at 200 sites on BCI, and found strong negative density-dependent effects on 53 focal species, which led to a significant increase in local species diversity in the transition from seeds to seedling recruits over four years. The studies by Augspurger and coworkers strongly suggest that a large proportion of this seedling mortality could have been caused by damping-off pathogens.

Other diseases are also important for Janzen-Connell–like mortality patterns in tropical forests. Also on BCI, the canker-fungus *Botryosphaeria dothidea* reduced growth rate and increased mortality of seedlings and small saplings of *Tetragastris panamensis* (Burseraceae), and canker formation was significantly greater on seedlings under the canopy of parent trees than at further distances (102). Another canker disease of various species of *Ocotea* and *Nectandra* (Lauraceae) caused by *Phytophthora* sp. (105, 106) showed that Janzen-Connell effects may continue to be important determinants of the spatial distribution of mortality of susceptible hosts decades old. Mortality of juvenile *Ocotea whitei* (1–30 cm dbh) and reduction in clumping of juveniles over an 8-year period was consistent with spatial patterns of the canker disease. In a study of 13,000 seedlings in two cohorts of *O. whitei* at the same site, mortality was similarly density dependent and caused a significant shift away from parent trees over time (104). Particularly for forest trees like *O. whitei* that reproduce on a supra-annual basis so that seedling densities vary greatly among years (104), disease-driven reductions in host density may lead to reductions in disease pressures, setting up coupled density-dependent cycles for both the host and pathogen populations.

The Janzen-Connell theory predicts that adult individuals should be less clumped than expected from random mortality of the original seed distribution, but not necessarily randomly or regularly distributed in space. Although in some cases distance-dependent mortality can cause a shift from an initially aggregated distribution of seedlings to a random distribution of adults (36, 37), many tropical species retain a significantly clumped distribution at maturity (92, 105, 132). This may be due either to the overwhelming effect of seed shadows or to the effects of environmental heterogeneity on plant survival. Regardless, this pattern is not contrary to the predictions of the Janzen-Connell hypothesis. True tests of the Janzen-Connell hypothesis do not rely on static analyses of the spatial distribution of entire species or even juveniles with respect to distance from conspecific adults, but rather look at the changes in degree of clumping over time compared to the initial distribution of seeds and seedlings.

JANZEN-CONNELL EFFECTS IN TEMPERATE FORESTS The Janzen-Connell hypothesis was developed as an explanation for maintenance of high diversity in tropical

forests and most tests of the model have been in those ecosystems, but it is now clear that this phenomenon can also be important in temperate forests. Packer & Clay (170) provide the first complete test of the Janzen-Connell hypothesis in a temperate forest, as well as the first direct evidence for the local maintenance of species diversity. In Indiana, black cherry seedlings suffered 100% mortality from damping-off caused by *Pythium* spp. in soil from beneath parent trees, whereas mortality was low for competing tree species in the same soil. Disease-induced mortality decreased sharply with distance from the parent trees, causing a significant shift in the spatial distribution of seedlings away from the mother tree. This study clearly shows that the negative feedback of increased soilborne pathogens beneath the parent canopy puts less susceptible seedlings of locally rare species at a competitive advantage over the parent's much more common offspring, thus maintaining local plant diversity in a temperate forest.

Although the Janzen-Connell effect clearly can be important in temperate forests, several studies suggest it may be less common there than in tropical forests (122, 128–130). At least three factors could contribute to the difference between forest types. First, lower diversity and corresponding higher density of individual species in temperate forests may lead to a strong overlap in seed and seedling shadows from different mothers of the same species, negating the advantage of dispersal (128). Second, the Janzen-Connell effect depends on differential susceptibility of hosts to a particular pathogen. In some temperate forests, particularly in conifer forests where most individuals may belong to just a few genera in one or two families, many pathogens may be capable of infecting a majority of individuals in the forest. Nevertheless, the expanding foci of infection, differential effects on local tree species, and greater survival of less susceptible conifer species in the wake of diseases caused by *Phellinus wierii* (118, 124, 125) and the *Heterobasidion annosum* complex (95, 181) could be considered special cases of the Janzen-Connell effect. Finally, for Janzen-Connell effects to play an important role in tree diversity and distributions, disease effects cannot be overshadowed by large catastrophic disturbances such as fire or windstorms that can cause catastrophic stand replacement (208). Fire disturbance is much more frequent in forests of western North America (44, 197, 198) than in the wet tropics (209), but even in tropical systems diseases may be less likely to play a key role in those areas that are subject to frequent hurricanes, landslides, or other disturbances (24).

HOST DENSITY AND PATHOGEN SPECIFICITY The Janzen-Connell effect requires a degree of host specificity, but for a specialist fungus to remain a viable part of a forest ecosystem a suitable host must not only be present, but present at sufficiently high density to ensure that the fungus can colonize new host individuals. Dispersal-limited, host-specialized pathogens would not likely dominate in a high-diversity forest where most host species will be at low density; such forests should be dominated by fungi with broad local host ranges. In contrast, a low-diversity forest, with relatively high densities of each of the component species, should favor the development and maintenance of a fungal community dominated by host specialists, as long as the available hosts are not all too closely related to

facilitate host specialization. The effects of host community diversity and structure on fungal diversity and host specificity are largely unexplored, except for recent studies of the diversity and host specificity of wood-decay polypore fungi in Central American tropical forests. In the high-diversity moist tropical forests of BCI in Panama (103) none of 43 species showed significant host preferences, and in the medium-diversity dry tropical forest of Costa Rica (150) only 3 of 32 species showed host specificity. The fungal assemblages in these forests included many rare species, but the common fungi were generalists. In contrast, in a naturally low-diversity mangrove forest in Panama (only 3 host tree species present, each from a different family), the polypore fungal community is strongly dominated by just a few common species, each highly host specific (107). Considerable work has been done in agricultural settings on how the structure of the plant assemblage affects the development of disease (166). Additional research on the effects of host density, diversity, and spatial structure on the diversity and specificity of pathogenic fungi is clearly needed to understand the dynamic effects of pathogens on competitive interactions in natural plant communities.

RAPID EVOLUTION AND LOCAL ADAPTATION

The Red Queen Hypothesis

The minority advantage afforded rare, resistant species in the Janzen-Connell model, or more generally by disease-driven apparent competition, is of course not static. As a rare host becomes more common, its own pathogens may increase in abundance or virulence through density-dependent feedback and frequency-dependent selection. Additionally, pathogens from one host may evolve to acquire new hosts (96, 180, 187). Natural selection for increased virulence on hosts that recently became common occurs each time a resistant agronomic cultivar is defeated by a new race of pathogen. Wild hosts can also evolve resistance to pathogens, leading to the coevolutionary arms race known as the Red Queen hypothesis (69, 86). Rapid evolution of interactions between hosts and microorganisms are common in natural systems as well (199). In wild plant-pathogen interactions, hosts should evolve toward increased levels of resistance, and pathogens should evolve toward optimum levels of virulence to maximize their own fitness (89, 149). Rates of evolutionary change may not be equal between hosts and pathogens, since differences between partners in rates of molecular change in coevolutionary processes are correlated with generation time (115). Short-lived plants may have generation times similar to their pathogens, and both numerical and evolutionary responses will be important in the dynamics of host-pathogen interactions. For instance, the introduction of the rust *Puccinia chondillina* for biological control of the invasive skeleton weed *Chondrilla juncea* (Asteraceae) in Australia was followed by a dramatic shift within the host population from dominance by the susceptible host biotype to resistant biotypes, due to a reduction in the otherwise superior competitive ability of the susceptible biotype (50, 51). In forest systems, the generation

time of most pathogens will be much faster than that of the host species, and at least in the short term (a few tree generations), pathogens may pass through many generations under strong selection for increased virulence before resistant hosts reach reproductive maturity. Understanding the interplay between numerical and evolutionary dynamics in natural plant-pathogen systems is a key challenge for the coming decade (17, 22).

The ability of hosts to respond evolutionarily to selection pressures from pathogens will depend not only on generation time, but also on host genetic variability and rates of outcrossing. Burdon et al. (55) compared two distinct wild metapopulations of *Linum marginale* infected with the rust *Melampsora lini*; one host metapopulation was strongly inbred and the other was strongly outcrossed. They found that the outcrossed populations showed consistently higher overall levels of resistance, numbers of resistance phenotypes, and levels of polymorphism for specific virulence factors in the pathogen. Many systemic fungal parasites castrate their host plants, allowing only clonal reproduction or autogamy and avoiding Red Queen challenges of sexual recombination in the host (68). Both intrinsic variability in host outcrossing rates and pathogen manipulation of the host may affect the response to selection in evolutionary arms races. Additionally, metapopulation structure, including effects of genetic drift and gene flow, may be equally important in determining the dynamics of reciprocal selection (200).

Local Adaptation and the Scale of Dispersal

The spatial scale of dispersal is central to the ecological and evolutionary dynamics of plant-pathogen interactions (203). The dispersal of pathogens to susceptible hosts, the dispersal of pollen for outcrossing in plants, and the dispersal of seeds away from areas of high disease pressure, all determine the spatial scale of ecological interactions including disease persistence, host distribution, and epidemic development, as well as the relative isolation and genetic differentiation of locally adapting pathogens or plants. Below I briefly examine recent research on the role of dispersal in ecological and evolutionary dynamics of host-pathogen systems.

PATHOGEN DISPERSAL GRADIENTS Although there is a long tradition of study of the spatial spread of plant diseases in agronomic and forestry settings, our detailed knowledge of the spatial scale of pathogen dispersal is limited to a fairly narrow range of plant pathogens, and an even smaller set of pathogens important in natural systems. There have been several excellent reviews of the extensive literature on pathogen dispersal through the air currents, by rain splash, through the soil or subsurface water, and by vectors (31, 32, 57, 91), with nearly all examples coming from agronomic settings. Power law and exponential models often provide reasonably good fits to observed dispersal gradients. Fitt et al. (91) fit 325 data sets of spore or pollen deposition or disease gradients to the two models and found that both models worked well, with exponential models working somewhat better for splash-dispersed systems and power law slightly better for wind-dispersed

systems. They note that the exponential model has the advantage of providing a "half distance" measure where the observed deposition decreases by half with a constant distance increment, and show that half distances are greater for airborne than for splash-dispersed or soilborne diseases. However, these two models and all current empirical methods do not adequately measure or describe the tail of the dispersal curve; improvement both of measurement abilities and mathematical descriptors for dispersal tails is an important current area in spatial ecology.

Empirical measurement of dispersal gradients for a broad range of pathogen types in natural ecosystems, and appropriate mathematical description of dispersal gradients, would provide important insights into the scales of host-pathogen interactions and the potential range of types of interactions in natural systems. Although there are few direct studies of pathogen dispersal in natural ecosystems, the available literature suggests that some fungal spores may travel hundreds of kilometers (176, 201). However, damage from UV irradiation (173, 183, 184) and susceptibility to desiccation (206) limit regular long-distance dispersal of most pathogens, with dispersal usually contained within tens to hundreds of meters for airborne and less than one meter for splash-dispersed pathogens. Some pathogens may move longer distances through "stepping stones" of infection and reproduction, achieving further dispersal in a series of shorter steps (33, 158, 176).

SPATIAL PATTERNS Our understanding of the role of the spatial scale of dispersal for structuring natural microbial communities and populations is growing rapidly. In a wet tropical forest, Lodge & Cantrell (155) found no significant resemblance between litter basidiomycete and microfungal communities in small patches separated by more than 100 m. Arnold et al. (23) found significant differences in fungal endophyte communities within a host species across sites separated by a few hundred meters in a moist tropical forest. Both studies suggest that distance has a strong effect on determining fungal community composition, most likely through the independent effects of dispersal limitation and environmental heterogeneity on component species. Traditional phytosociological studies of the spatial scale of turnover in fungal communities must be complemented with studies of the spatial distribution of individual species, populations, and genotypes.

Individual fungal species often show strong spatial patterns, reflecting both environmental heterogeneity and dispersal limitation. Fungal infection is often aggregrated within individual hosts. Infection by the common endophyte *Discula quercina* was spatially aggregated among leaves within a individual host *Quercus garryana* (Fagaceae) (226). Similarly, the tropical leaf-colonizing fungus *Scolecopeltidium mayteni* on *Trichilia* spp. (Burseraceae) was highly correlated among leaflets within a leaf, but showed high variation among leaves (108). Carroll (59) reviewed endophyte distributions and suggests that within trees the height within the crown is the most important determinant of spatial structure, whereas within individual branches the age of the leaf is most important. Although these studies suggest that propagule dispersal may limit where fungi can infect host plants,

environmental conditions often ultimately control whether infection takes place or disease develops (61, 108, 138, 230). Unfortunately, however, except for important pathogens of widespread crops, our understanding of the effects of dispersal and environment on the large-scale biogeographic distribution of pathogens remains poor. Collaborative work between pathologists working in separate regions or habitats could help further our understanding in the area.

Within a fungal species, genotypes are also often spatially aggregated. Isolates of the chestnut blight fungus *Cryphonectria parasitica* showed strong genetic structure, with isolates of the same genotype closer together than expected at random (162). Genets of *Armillaria* spp. in nine forest sites in New York were dominated by very small genets (one sample point) with a low frequency of larger genets up to 44 m long (228). A number of studies show significant genetic structuring of fungi in natural ecosystems over large geographic distances (e.g., 134, 135, 168). Understanding how different life histories of fungi affect the scale of genetic structure will provide much insight into the pattern and likelihood of local adaptation in plant-pathogen interactions.

METAPOPULATIONS Dispersal limitation will also strongly control the evolutionary and ecological dynamics of host-pathogen metapopulations by coupling the host and pathogen populations of connected demes and decoupling the dynamics of more distant ones. Ericson et al. (87) found strong variation among years and among islands in the dynamics of infection of *Valeriana salina* by *Uromyces valerianae* in 30 discrete populations in an archipelago in Sweden. Pathogen populations commonly went extinct and recolonized healthy host populations, and disease was more likely to spread between neighboring populations. Similarly, wild flax (*Linum marginale*) infected with the rust *Melampsora lini* showed asynchronous disease dynamics and large differences in diversity and frequency of virulence and resistance phenotypes among demes in a metapopulation, but demes closer together had more similar resistance phenotypes than more distant demes (54). Burdon & Thrall (53) have provided a good review of the role of spatial structure for ecological and evolutionary dynamics of plant-pathogen interactions, with particular reference to its effects on coevolutionary dynamics both within single populations and across a metapopulation. Plant-pathogen systems are among the few where both numerical and genetic dynamics have been studied in a spatial context, and where the genes under selection are unambiguous. Research on the spatial context of the evolutionary ecology of plant-pathogen systems is important not only to disease ecologists, but to the field of evolutionary ecology in general.

LOCAL ADAPTATION If the evolutionary dynamics of plant-pathogen interactions are spatially heterogeneous (47, 199), then the dynamics described for interspecific interactions in the Janzen-Connell hypothesis and other forms of disease-driven host dynamics can be extended to ecological interactions between genotypes of the same host species. Rare genotypes should be at an advantage if microbial

feedback processes are driven by the locally dominant host genotypes (153). A pathogen that is reliably "cultured" for generations on the leaves or roots of a single mother tree may adapt to that host genotype and increase in virulence over time; consequently, it may be more virulent on offspring of that tree than on rare seedlings of a more distant mother of a different genotype (101, 179). Isolated host patches with independent local coevolutionary trajectories should lead to different types of disease interactions when rare seeds disperse from one patch to another. There are numerous examples of local adaptation in agricultural systems (e.g., 6), but few published examples from natural systems. Two genetically isolated, but nearby (50 m) populations of the annual, selfing legume *Amphicarpaea bracteata* (Papilionaceae) differed in susceptibility to the specialist *Synchytrium decipiens* (171), with one population completely resistant to the pathogen and the other showing almost no resistance (172). On the other hand, Davelos et al. (78) did reciprocal transplants of the clonal prairie grass *Spartina pectinata* among patches with low and high incidence of species of *Puccinia* rusts. There was no effect of rare-genotype advantage in transplants at a 4.5-km scale, although at 120 km there was an indication of lower disease on transplanted hosts. We still have a limited understanding of the scale of local adaptation of pathogens in natural ecosystems. Reciprocal transplant and common garden experiments will provide significant advances in our understanding of the scale of local adaptation in plant-pathogen systems.

CONCLUSIONS

We must bring a solid understanding of the life histories of both pathogens and plant hosts to our efforts to appreciate the effects that plant disease can have in natural ecosystems. Further empirical work should focus on improving our understanding of pathogen dispersal and host specificity in natural communities, and on the dynamics and spatial scale of rapid evolutionary changes in plant-pathogen symbioses. Just a few decades ago, documenting that diseases were parts of natural systems was exciting news, and our expectations of their effects in natural plant communities were simple extensions of our understanding of agroecosystems. Now we are quickly developing a library of studies on wild plants set within an integrated framework of evolutionary ecology. This empirical work both fuels and builds on an increasingly robust body of theory, underscoring the fundamental place of diseases in ecology and evolution.

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