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Disease in Natural Plant Populations, Communities, and Ecosystems: Insights into Ecological and Evolutionary Processes

Overview and History

Pathogens are associated with virtually all plant species, from a diverse array of habitats. Although we know the most about diseases of economically important plants, the goal of this article is to describe current work on host–pathogen interactions in natural or unmanaged systems outside of the crop field or forestry plantation. Agricultural scientists, of course, have contributed to this research. For example, disease resistance genes are often discovered as a result of surveys of wild relatives of crop plants, and it is well known that crop disease levels can be influenced by diseases in nearby weeds. Starting in the late 1970s, however, research on natural plant–pathogen interactions began to both increase and diversify, with an explicit goal of understanding the ecology and evolution of these interactions. This article will trace the history of this young discipline and provide highlights of ongoing research areas. I will focus on work by ecologists and evolutionary biologists that often is not published in plant pathology or forestry journals.

A first step is to define a *natural* system as opposed to an *agricultural* or human-managed system. In contrast to most row crops, most natural populations occur in complex spatial arrangements with tens to hundreds of other species, are genetically diverse, and consist of plants of different ages and developmental stages. Seeds produced by a population in one year are the source of future generations at the site. Natural populations need not be pristine; as defined above, roadside weeds are as natural as prairie wildflowers. This natural versus agricultural dichotomy is also, of course, simplistic. Forest plantations and pasture landscapes have many natural attributes, and the degree of distinction between agricultural and natural ecosystems varies around the world.

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Although there have always been researchers intrigued by the role of pathogens in nature (58), publications in the 1970s and 1980s were influential in introducing the study of plant disease to ecologists and evolutionary biologists. For example, John Harper, a British biologist, published a landmark book in 1977 called *Population Biology of Plants* (61). This large tome (892 pages) encouraged ecologists to view plants not as vegetation, but as populations of individuals. One of his 17 chapters was titled "Pathogens" and emphasized the effect that disease could have on the structure and dynamics of plant populations and communities. Harper's book was followed a decade later by Jeremy Burdon's 1987 book *Diseases and Plant Population Biology* (30). Burdon expanded on the ecological consequences of disease and also emphasized genetic interactions between host and pathogen. Both Harper and Burdon emphasized the relevance of crop research and theories developed in an agricultural context, such as van der Plank's (101) work in epidemiology and Flor's gene-for-gene hypothesis (50). Interestingly, at the same time period, several plant pathologists were discussing disease in natural systems (130). Browning (29), in particular, advocated using genetically diverse natural ecosystems as models for development of new approaches to control crop disease. Several groups were also documenting the diversity of resistance genes present in wild relatives of crop plants (42,135). Perhaps all these developments, as well as a general interest in biotic interactions (43,67), contributed to the appearance of papers on plant disease in the ecological and evolutionary biology literature. This research focus has continued to grow and diversify.

In this article, I will consider disease in the context of two broad scales of organization: first, the ecology and genetics of plant populations; and second, the structure and function of plant communities and ecosystems. My examples emphasize the effects of fungi, since these are the groups most often studied.

Population Ecology and Genetics

What are the effects of disease on individual plants? On plant populations? In agricultural systems, researchers focus on

the effect of disease on crop yield. However, in natural systems, where seeds produced one year affect the numbers in subsequent generations, population ecologists strive to understand the effect of disease on the dynamics of the host plant population, defined as changes in the number (=population size) of the population over time. A first step is to understand the effect of disease on the survival and reproduction of individual plants (=fitness [12]). Obviously, pathogens have diverse effects, including killing individual plants (e.g., soilborne diseases affecting seeds and seedlings), reducing plant growth (many foliar diseases, some wilt and canker diseases of trees, and many viral infections), and reducing seed production either directly (such as the smuts or ergots) or indirectly because of diminished plant growth (54,69). In a crop monoculture, it is obvious when individual plants die since gaps are left in planted rows; in a more variable natural population, reduced vigor or death due to disease is often not apparent unless individual plants are followed throughout their lives (3). Interpretation of some pathogen effects is complex. For example, fungal infection of seed heads of exotic goatgrass by *Ulocladium atrum* reduces seed mass and seed number. However, infection also breaks down the woody seed head, leading to early seed germination and overall higher establishment (45). Plant ecologists are likely to completely miss the effects of some infectious agents; for example, variation in plant growth and size may be due to undetected viral infection (105).

Plants and pathogens can be studied at a variety of spatial scales (Fig. 1). Negative effects of disease on survival and reproduction of individual plants can, for example, lead to reductions in host population size and changes in spatial distributions. Such effects can occur with native pathogens or introduced pathogens. An example of the former is the laminated root rot fungus, *Phellinus weirii*, which kills individuals, leading to local population declines (59). Diseases due to introduced pathogens have received more attention (e.g., chestnut blight [8], anthracnose of flowering dogwood [65], sudden oak death effects on tanoaks [107]). In addition to these examples of infection of mature individuals, pathogens can also lead to declines in dormant seed populations, potentially affecting the local persistence of species (81). It is of particular interest whether disease spread and the effect of a pathogen on plant fitness are dependent on the density of the host population. If effects of pathogens increase with plant density, pathogens have the potential to regulate host population size and alter spatial distributions (4,51). Several studies have demonstrated important density-dependent effects of disease in natural plant populations (18,21,55,75). Of course, plant survival, size, and growth rates also depend on plant density, and compensatory effects can alter the effect of disease on plant populations. For example, Alexander and Mihail (5) found that high seedling mortality of an annual legume due to *Pythium* infection did not translate into decreased reproductive output of experimental plant populations. In this case, the rela-

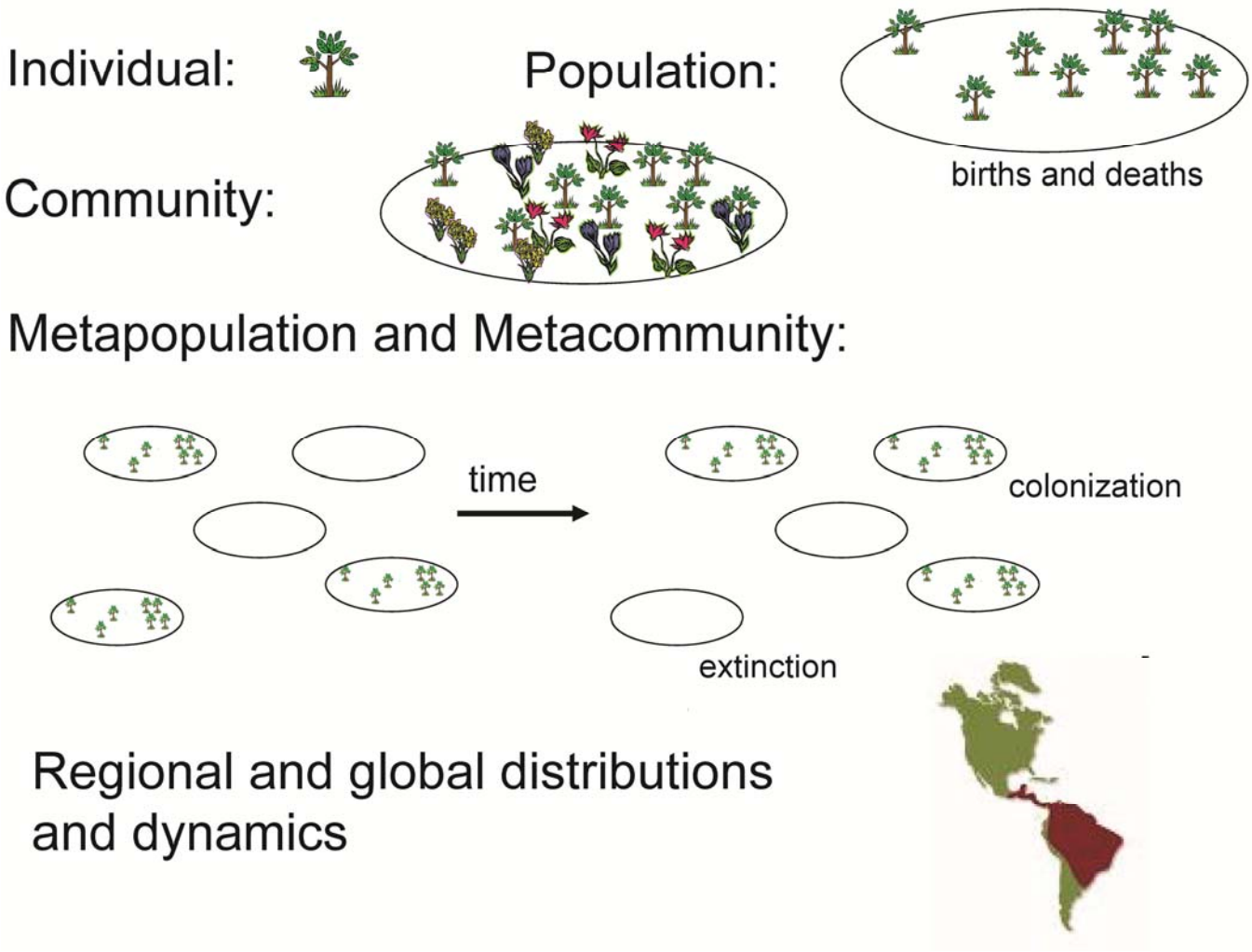

Fig. 1. Ecologists studying disease in natural or unmanaged systems focus on a variety of levels of organization, including effects of disease on survival and reproduction of individual plants, number of plants within a population (population size) and how it changes over time (population dynamics), and composition of a multispecies community. Researchers focusing on metapopulations consider multiple populations across a landscape. Local extinctions can occur for individual populations, and empty but suitable habitat can be colonized. Metacommunities refers to a similar concept, but at the community level. Finally, pathogens can interact with plants on broad regional and global spatial scales.

tively few plants that survived the seedling stage experienced low density conditions and had increased growth and seed production.

Recent work on natural plant–pathogen interactions has considered broader spatial scales and the realization that plant and pathogen populations are often patchy in their distributions. Further, individual populations can go extinct; rates of extinctions and colonizations will be important in the overall distribution and abundance of both plants and pathogens. This metapopulation perspective (60) has impacted both ecological and genetic studies of disease (123) (Fig. 1). For example, Smith et al. (117) explored epidemiological patterns of a rust and its perennial host plant across a collection of islands. Using long-term survey data and statistical models, they inferred patterns of dispersal of pathogens within and among island chains. Further, they found that the existence of a few large plant and fungal populations was disproportionately important to the overall stability and persistence of the larger system of populations. In research on Australian beach populations of *Cakile maritima* infected by *Alternaria*, Thrall et al. (127) found that large spatial scale environmental factors affected the severity of epidemics. However, the temporal courses of epidemics were unique to individual populations, suggesting that disease processes occurred independently at each site.

Interactions between the anther-smut pathogen, *Microbotryum violaceum*, and plants in the carnation family (Caryophyllaceae) provide a particularly useful example of research on multiple spatial scales, and on using a variety of research approaches (Fig. 2A). Flowers of systemically infected plants produce anthers full of fungal spores; insects that visit flowers move spores between plants. On the individual level, the pathogen leads to the sterilization of the plant. On the population level, increased disease levels are associated with reduced plant population growth (15,22,37) (Fig. 2B). Studies of large geographic areas in Sweden revealed that levels of anther-smut disease of *Lychnis alpina* are highest in plant populations that are more continuous in their distribution (36), presumably because of the greater likelihood of spore dispersal among populations. Over an archipelago of islands, anther-smut disease levels were often dependent on host population age, size, and density (37), with loss of disease in some small populations. Cross-species disease transmission can also be important in the local persistence of the disease at some sites (35).

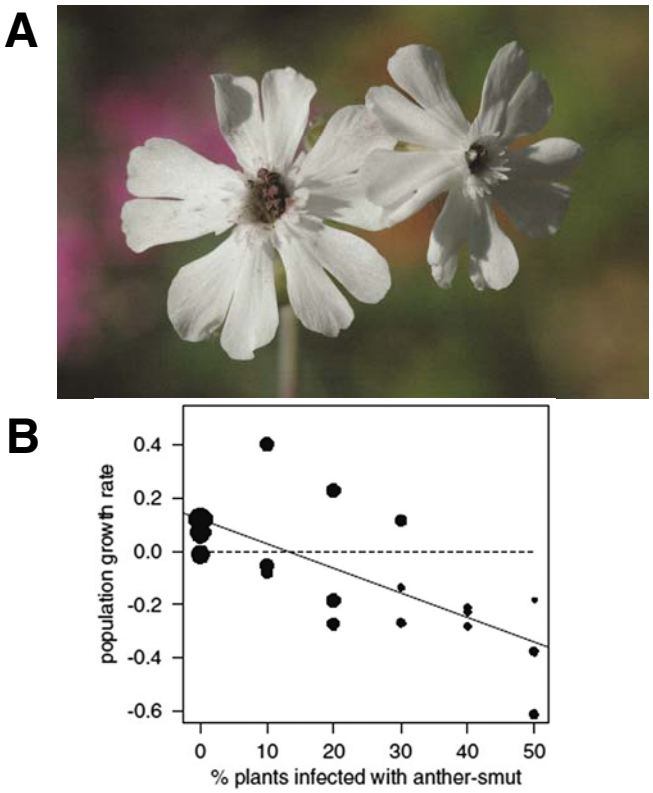

Fig. 2. *Silene latifolia* is a herbaceous plant in the Caryophyllaceae that can be infected by the anther-smut pathogen, *Microbotryum violaceum. S. latifolia* has separate male and female plants. **A,** Infected flowers of both sexes produce teliospores in the anthers. In females, the ovary aborts at an early stage, and anthers are produced that are full of spores. Photograph by Michael Hood. **B,** Populations with a higher proportion of diseased plants have lower rates of population growth (data from 1989-1993, Giles Co. Virginia; dot sizes are proportional to number of populations). Adapted by permission from Macmillan Publishers Ltd: Bernasconi et al. (22).

Antonovics and coworkers have amassed an unusually long record of spatial and temporal patterns of disease with two decades of annual surveys of anther-smut disease of roadside plants of *Silene latifolia* (10,15,16). These studies reveal a dynamic system: local healthy and diseased populations frequently went extinct, disease was often lost from healthy populations, and disease transmission led to new disease foci. By creating mathematical models that captured the essential dynamics of the host and pathogen populations, researchers could do "experiments" on ecological and genetic questions that would be logistically impossible to perform over large geographic areas (9,15). For example, Antonovics (9) explored what would happen if a hypothetical resistance gene spread through the metapopulation and eliminated the disease. As might be expected, there was an increase in the abundance of the plants in the simulation. However, the magnitude of the increase was much larger than expected. Not only did elimination of the disease increase seed production per plant, but the increased seed production resulted in larger healthy plant populations that were less likely to go extinct and had an increased probability of colonizing new sites. Studying the effect of disease on plant populations thus requires a broad understanding of plant dynamics, including processes that may not be obviously related to the individual level effect of disease on plant survival, growth, or reproduction.

Diseases also have been postulated to affect plant distributions at geographical spatial scales (Fig. 1). For example, environmental conditions conducive to disease could limit the ability of a plant species to persist in a particular region. A particularly intriguing question is the ecology of host–pathogen interactions at the edge of a species range. The classical expectation is that disease incidence will decline in marginal populations (11); Alexander et al. (6), for instance, found that small, isolated host populations of *Carex blanda* at the edge of the species range were more likely to be free of an ovarian smut disease, presumably due to limited dispersal by the fungus (Fig. 3). However, a decline in disease at the margins of a range is not inevitable; recent theoretical work reveals many other potential outcomes depending on host demography and the mode of pathogen transmission (11). Given the logistical challenges of field surveys at large spatial scales, researchers have explored the use of herbaria surveys for distributional information (6,14). Herbaria surveys also provide a unique opportunity to explore temporal patterns in disease; two studies of obligate pathogens showed increases in disease over 100-year spans of collections (6,14).

What are the genetic interactions between plants and pathogens in natural plant populations? How does one integrate genetic and ecological questions? The resistance genes used by

Fig. 3. Studies of smut infection of the sedge *Carex blanda* caused by *Anthracoidea blanda* **(A)**. In addition to field surveys, Alexander et al. (6) used surveys of herbarium specimens **(B)** to study distribution of diseased plants at regional spatial scales.

crop breeders are ultimately derived from wild populations of crop progenitors or their relatives, and genetic variation in disease resistance has been commonly reported within natural populations. A major focus of evolutionary research on disease in nature has been to explore the causes and consequences of such variation in plant populations, as well as to study variation in virulence in pathogen populations.

The most extensive research on genetic variation in plant resistance and pathogen virulence outside of agriculture is work on the Australian wild flax species (*Linum marginale)* and the flax rust fungus (*Melampsora lini*) (Fig. 4A). This work has been led for the last 20+ years by Jeremy Burdon and Peter Thrall and includes studies on a broad range of spatial scales. Flax rust primarily affects plant survival, especially overwintering of the host following severe epidemics (68). Researchers developed efficient inoculation protocols to study genetic variation in host resistance and pathogen virulence, using a large set of plant and fungal lines from multiple populations (126). On the spatial scale of the continent of Australia, Barrett et al. (19) found that pathogen isolates are of two evolutionary lineages: these lineages differ in origin, geographical distribution, and interactions with host plants and the environment. There is a general trend of isolates being less likely to infect host lines from more distant locations (34). On a regional spatial scale, plants and pathogens exist as a collection of populations that vary in their isolation as well as their permanence. For example, rust may disappear from local host populations for a season, but then reappear due to long-distance spore dispersal. Patterns of local adaptation and coevolution exist at this spatial scale (i.e., populations separated by 5 to 10 km) (126), with populations of more resistant hosts selecting for more virulent pathogens (125) (Fig. 4B). Intriguingly, it appears that lower rates of spore production by highly virulent pathogen isolates may explain why such isolates do not dominate rust populations (125).

There is often considerable variation in resistance and virulence structure both within and between local populations in the wild flax–rust system (31). The complexity of these patterns suggests that gene flow (movement of genes between populations through spore and seed dispersal) and genetic drift (chance loss of genes due to small population size or local extinctions) are likely to have a large effect on patterns of resistance or virulence. The wild flax–rust system is thus often cited as an example of Thompson's "geographic mosaic of coevolution" (121,122), where temporal and spatial variation in gene flow, genetic drift, and selection jointly determine the strength of coevolutionary interactions.

With the wild flax–rust system and many other diseases, there is clear evidence for gene-for-gene interactions (122). Natural selection can, however, potentially operate on any trait that affects likelihood of disease. For example, with diseases that are spread by pollinators, selection may occur on quantitative genetic traits that make plants more attractive to floral insect visitors (for example, timing of flowering, rates of flower production, and floral morphology [1,25,57]). Further, selection for tolerance is likely to be common, where disease occurs yet there is no reduction of plant fitness (112,116). In fact, Roy et al. (113) has argued that widespread selection for tolerance may be important in explaining the ubiquity of disease in natural systems. Recent work by Inglese and Paul (66) explores the physiology behind tolerance and provides an example where there is greater expression of tolerance in *Senecio* to a native compared to an introduced rust.

It is challenging to explicitly combine ecological and genetic studies, and we know very little about many of the key components of these interactions (7). In a particularly strong linkage of theory and experimental work, Thrall and Jarosz (129) created multiyear experimental populations of *Silene latifolia* with different densities and frequencies of anther-smut disease; plants in the populations differed in resistance. By following the loss or persistence of disease, they could explicitly link studies of host genetic structure with disease incidence and plant population dynamics. Larger spatial scale surveys also show an inverse relationship between disease prevalence and host resistance (powdery mildew [73]; rust [118,124]). A recent development is integration of modeling approaches with ecological data to infer potential genetic interactions (94).

Plant pathologists have led the way in another research area that combines ecology and genetics, the degree to which genetic variation within a host population affects disease spread. Several researchers have shown, for example, reduced disease spread in multiline crops and variety mixtures (89,138); competitive interactions between plants in mixtures also can affect disease outcomes (49). Our understanding of the effects of host genetic variation on reduction of disease spread in nature is less clear. Small-scale variation in genetic diversity within populations has been associated with reduced disease in some studies (114) but not in others (2,109). On a larger spatial scale, Thrall and Burdon (124) did report a negative association between disease prevalence and host resistance diversity by surveying multiple host populations in the wild flax–rust pathosystem.

Finally, an intriguing and important ecological-genetic question concerns host shifts and the possibility of newly emerging diseases (115). In two different studies where the anther-smut pathogen was found to infect previously unknown hosts, disease symptoms on the new host were atypical, suggesting poor adaptation (13,76). Inoculation experiments with the new host revealed considerable variation in susceptibility, suggesting the potential for rapid evolution of host resistance (13). Although one might predict that pathogens would move to genetically related host species, Roy (110) found that geographic proximity of potential hosts was a stronger

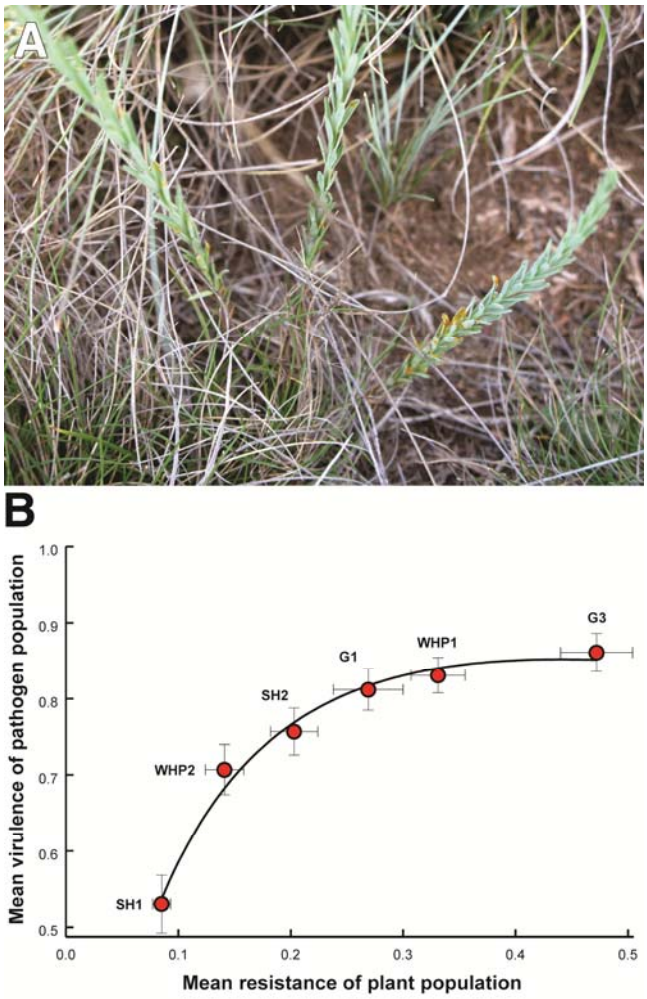

Fig. 4. Flax rust (caused by *Melampsora lini*) on wild flax, *Linum marginale*. **A,** Pustules on leaves. Photograph by Anna-Liisa Laine. **B,** There is a positive relationship between mean resistance of plant populations and mean virulence of associated fungal populations (125).

predictor of host shifts than phylogenetic relationships in a study of rusts on crucifers.

Community and Ecosystem Ecology

What is the effect of disease on the number of species in a plant community and their relative abundance? Community ecologists focus on interactions among multiple species and how these interactions affect community composition and function; pathogens are likely to have powerful effects on food webs through both direct and indirect interactions (72). For example, if a disease solely affects a competitively dominant plant species, the effect of disease may be to increase the abundance of other species. In contrast, if disease only affects a rare plant species, the pathogen may lead to the decline of that species and reduce the diversity of the plant community. Of course, effects of pathogens on community structure are rarely this simple. For example, the laminated root rot fungus, *Phellinus weirii,* infects several species, but there is considerable variation in susceptibility. Pathogen-induced tree mortality leads to slowly increasing mortality centers, and thus the disease is a major agent of disturbance (59) (Fig. 5). In some community types, the action of *P. weirii* is to reverse successional change, while in other community types the effects are less predictable (63). In another classic example, the community effects of *Phytophthora cinnamomi* invasion of Australian forests are immense, with field-resistant sedge and grassland communities replacing susceptible *Eucalyptus* trees (137). Three decades later, there is evidence of susceptible plants reinvading some infection sites, potentially suggesting long-term disease/plant community cycles (136).

The existence of multiple hosts for a pathogen can lead to particularly interesting effects if one or more plant species harbors the pathogen but is not greatly impacted. As noted by Haldane, "a nonspecific parasite…is a powerful competitive weapon" (58). For example, infected but tolerant hosts can contribute to the growth of the pathogen population and its spread to more susceptible species (pathogen spillover [103]). Such increased disease can in turn lead to decline in the population size of these susceptible host species, resulting in the phenomenon of apparent competition between the tolerant and susceptible species (4,64). A likely current example is the spread of sudden oak death in forest communities in California, where sublethal infections of nonoak species contribute to pathogen persistence and transmission (107,108) (Fig. 6). Apparent competition might be a particularly common phenomenon with viruses, given their broad host range (102). In California grasslands, for instance, both native perennial and exotic annual grasses are infected by *Barley yellow dwarf virus* (BYDV) (Fig. 7A). The presence of the annual exotic *Avena fatua* increases spring populations of aphid vectors and leads to the doubling of BYDV incidence in native grasses (79) (Fig. 7B). Since infected native grasses have reduced survival and reproduction (78), viral infection thus appears to facilitate the invasion of the exotic grasses. Malmstrom et al. (80) also isolated viral RNA from herbarium specimens of both native and exotic grasses from the early 1900s, supporting the hypothesis that viral infection may have played an important role in early periods of grassland transformation.

The above examples emphasize an important global change phenomenon: we have an unprecedented rate of biological invasions, with both plants and pathogens moving from their points of origin to other parts of the world. As already noted, disease often has been invoked as a part of the invasion process (99,131). The enemy release hypothesis, for example, postulates that the movement of plants to new locations leads to the loss of associated pathogens, and that the invasiveness of these introduced plants results from the lower disease pressure. To test these ideas, researchers have used multiple approaches. For example, Mitchell and Power (85) explored the use of pathogen databases and concluded that 84% fewer fungal pathogens and 24% fewer virus species infect plant species in their naturalized ranges in North America than in their native ranges in Europe. Further studies with this database suggested that the role of pathogens in invasiveness may be greater in plants from more mesic or nitrogen-rich environments (27). Analysis of pathogen databases of North American plants invading Europe, however, did not demonstrate as strong support for the enemy release hypothesis (134). Field research on disease and invasiveness has provided mixed results. It is intriguing, for example, that the soil biota has a net negative effect on black cherry establishment in its native range, but a positive effect in introduced European populations (104). However, there was little evidence of enemy release in studies of the effects of the soil biota on beach grass invasion in California (20). Similarly, Parker and Gilbert's (98) analysis of foliar pathogens on 18 species of native and nonnative clovers found both invasive and native species were infected by the same pathogens and at similar levels. A current thrust in ecological research is how best to design studies to untangle the importance of pathogens in the success of exotic plants (84), and

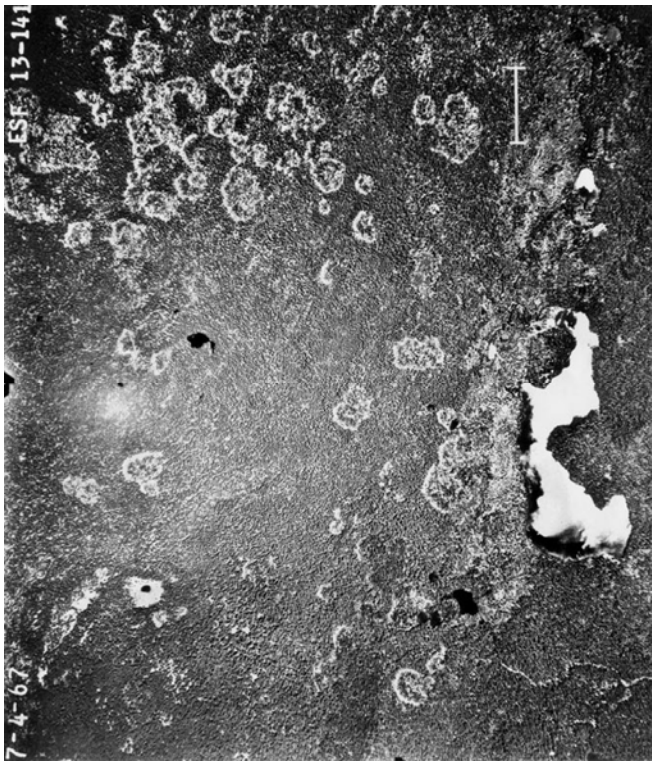

Fig. 5. Aerial photograph showing regions of a mountain hemlock forest in Oregon that contain dying trees due to the spread of *Phellinus weirii*. Photograph from Hansen and Goheen (59). Scale bar represents 200 m.

Fig. 6. Sudden oak death, caused by the oomycete *Phytophthora ramorum*, leads to high mortality of tanoak in California forests. Photograph by D. Rizzo.

more generally to understand the fate of novel host–pathogen interactions (97).

These diverse examples of community level effects of disease emphasize that knowledge of the host range of a pathogen is central to understanding its impact. Despite the great value of compendia of plant–pathogen associations (48), our understanding of host ranges for most pathogens is incomplete. Gilbert and Webb (56) sought to understand the ecology and evolution of host ranges for foliar pathogens in the lowland tropics of Panama. In this study, researchers developed a rapid protocol that allowed them to do over 500 *in situ* inoculations between different fungal and plant species (Fig. 8). Nearly all fungi were found to infect multiple plant species. Their data showed a strong phylogenetic signal, with tree species that were more closely related sharing more pathogens. Besides showing an unusually comprehensive community view of disease, these results have important applications. For example, host range data are often used for risk assessment of pathogens introduced to control weedy plants. Gilbert and Webb's work illustrates that simple rules (i.e., that pathogens will only infect plants in the same genus or family) are inadequate, and they introduce a methodology allowing efficient tests of a large number of plant–pathogen combinations.

For researchers examining soil microbes, one must consider a complex community of organisms including not only pathogenic fungi and nematodes, but also mutualistic organisms such as mycorrhizae and nitrogen fixing bacteria. Much of the ecological research on soil organisms focuses on the concept of feedback, a two-step process that includes both the effect of the plant species on the composition of the associated soil microbial community, and in turn, how the soil biota directly (or indirectly) affects the growth rate of the plant species (23,24). The net feedback can be positive, which tends to favor particular plant species and thus decrease the diversity of species in a plant community. Alternatively, negative feedback occurs when the microbial community that develops with a particular plant species has a net negative effect on its growth: this scenario could contribute to the maintenance of a diverse plant community. For example, Mills and Bever (82) reported that *Pythium* accumulates at different rates in the soil depending on which plant species are present in a local area. Intriguingly, work in a herbaceous community by Klironomos (70) showed that rare species typically showed negative feedback in greenhouse trials (due to the accumulation of soil pathogens). In contrast, positive feedback was more often found for common species (Fig. 9). Microbes, including pathogens, thus may play an important role in determining the relative abundance of plant species in a community. Soil pathogens have also been invoked as an important contributor to temporal change in species abundance. The coastal grass *Ammophila arenaria*, for instance, colonizes sandy shores in The Netherlands, but is replaced by later-successional species as sand dunes become stabilized. Van der Putten and Peters (132) showed that increases in fungi and nematodes under the soil of *A. arenaria* contribute to its eventual decline in the community. Many of these examples may seem rather obvious to agricultural plant pathologists, who are well aware of the buildup of soil pathogens with repeated crop cycles. However, interactions can be complex. For example, in grassland communities in the Netherlands, spatial variation in pathogenic nematode activity is important in determining fine-scale patterns in plant community composition. Variation in nematode numbers, however, depends on soil disturbance due to ant nest construction, and the numbers of ant nests are in turn dependent on rabbit and cattle grazing patterns (26,92).

The concept of negative feedback with soil organisms is conceptually similar to the Janzen-Connell concept, which focuses on probabilities of seedlings establishing near a conspecific adult (41,67,100). With trees, for example, most seeds fall close to the parent tree but some disperse to farther distances. If pathogens accumulate in the soil underneath the parent tree, successful seed germination and seedling growth will be inversely related to proximity to the parent tree. If the pathogens are specialists, such disease patterns could contribute to the maintenance of diverse plant communities. Early support for these ideas came from Augspurger's work in tropical communities (17); more recently, research by Packer and Clay demonstrated that seedling establishment of cherry trees in temperate forests is affected by soil pathogens that are common under adult trees (95,96). A merging of the ideas of soil-feedback and Janzen-Connell effects and a community level focus was the goal of work by Petermann et al. (100), who examined coexistence in European grasslands of three plant functional groups: grasses, forbs, and legumes. They found that species in each functional group had poorest growth when grown in soil that had previously supported growth of the same functional group; these detrimental effects were strongest when plants were also competing with other species. The effects disappeared when

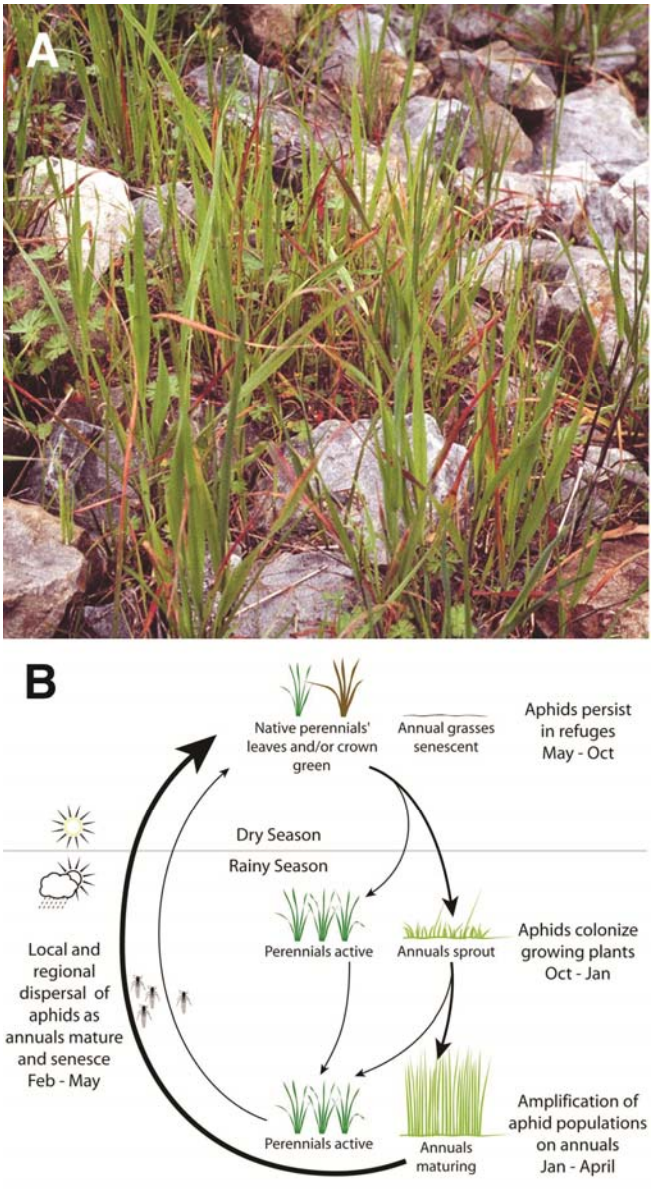

Fig. 7. *Barley yellow dwarf virus* (BYDV) infects both native and exotic grass species in California; aphids are important vectors of the disease. **A,** One of the important exotic annual grasses infected by BYDV is *Avena fatua*. **B,** Invasion of this grass in native grassland communities can contribute to high infection of native species because of the preferences and movement patterns of aphid vectors. In the rainy season, aphid numbers increase on all vegetation, with particularly high densities on annual plants. However, as annual grasses senesce in the dry season, native perennial grasses act as refuges for aphid populations, increasing the likelihood of viral infection of native grasses. Photograph and diagram by Carolyn Malmstrom.

soil was sterilized, suggesting that the biotic soil community was responsible. However, in this and many studies, it is challenging to know what soil organisms were responsible. A focus of future work is likely to be twofold. First, there is a need to dissect the black box of the soil community to understand which organisms and combinations contribute to ecologically relevant effects (106). Second, more detailed studies of patterns of plant density dependence, host specificity of pathogens, and the entire plant life cycle are needed to fully explore these hypotheses (51).

What is the effect of key environmental variables on plant disease, and how may a changing global environment affect the role of disease? Does plant disease alter ecosystem characteristics? The disease triangle is well-known to plant pathologists: pathogens require a susceptible host, a virulent pathogen, and a favorable environment for development of the disease. A current thrust of studies of disease in natural systems is exploring how a changing global environment could alter the incidence and impact of diseases on natural communities. Effects of global change are challenging to study: numerous attributes of the abiotic and biotic environment are projected to change (i.e., temperature, CO_2 levels, nutrient levels, species diversity) at various rates across the earth, and the impacts of these changes are likely to be species and location dependent. Garrett et al. (52) argues persuasively that consideration of these diverse issues requires a broad research agenda that ranges from the genome to the ecosystem.

Global warming could alter disease processes in many ways, including reduced mortality of pathogens in winter, increased number of pathogen generations per year, or shifting of host or pathogen geographic ranges (40,52,62). Roy et al. (111) took advantage of a meadow warming study in a montane community to examine pathogen and herbivore damage on plant species exposed to higher temperatures. When snow melted early in the season, due to either the warming treatment or plot aspect, the number of pathogen and herbivore species and their damage was generally higher. Individual pathogens and herbivores, however, often had species-specific effects, making it difficult to make general predictions of the effects of warming temperatures on disease. Field studies that manipulate temperature levels are understandably rare. Another approach focuses on whether plant–microbe interactions change as plants from warmer climates invade more northern habitats. Recent work, for example, suggested reduced soil pathogen effects (or, alternatively, increased soil mutualist activity) for plant species that moved into northwest Europe compared to plant species native to this region (133).

Studying the effect of increased CO_2 levels on disease levels of individual plants can be done in the laboratory, but understanding effects of enhanced CO_2 on populations and communities requires large-scale field manipulations. Mitchell et al. (87), for example, studied foliar fungal pathogens in the BioCON facility in Minnesota, a site where field plots differed in the number of species per plot, CO_2 levels (ambient versus increased levels), and nitrogen levels (ambient versus increased levels) (Fig. 10). They found that C_3 grasses had more pathogens with elevated CO_2. Disease also increased in plots with reduced species diversity, most likely because the plant species found in low-diversity plots were more locally abundant, allowing greater disease spread. This phenomenon is worthy of more study, given its agricultural implications for intercropping (28,53) and the linkage it provides between biodiversity research and disease ecology (39,71,88).

Soil nutrient levels in natural ecosystems can also affect disease levels. Springer et al. (119) showed that calcium levels and rust

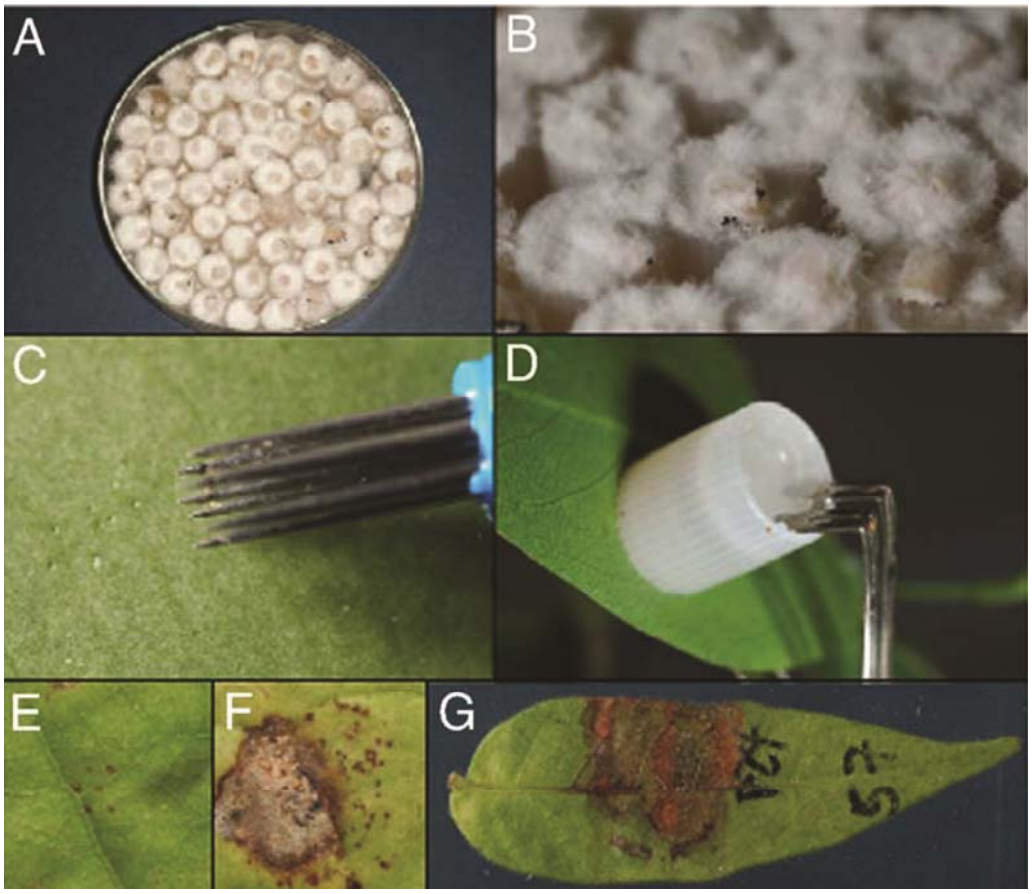

Fig. 8. Method for *in situ* inoculations of foliar fungal pathogens in a tropical forest community. Fungi isolated from diseased leaves were grown in agar-filled cryovial caps (top left; close-up, top right). Leaves were wounded with pins (middle left), and the inoculum in the cap was attached to leaves in the field (middle right). The bottom row indicates a wound response (left, resistant), a necrosis response (middle, susceptible), and a diseased leaf 7 days after inoculation (right). Figure from Gilbert and Webb (56). Copyright 2007 National Academy of Sciences, USA.

disease are inversely related; unusual plant communities associated with serpentine outcrops may thus be particularly vulnerable to disease due to low calcium levels. In contrast, disease levels often increase as nitrogen is added to an environment (87,91,120); such patterns are also known with agricultural crops. Strengbom et al. (120) explored the relationship between nitrogen addition and disease in boreal forests known to be nitrogen limited (Fig. 11). In experimental plots where nitrogen was added, infection of the fungus *Valdensia heterodoxa* increased on the dominant shrub, *Vaccinium myrtillus*. The increased disease on the shrub was also associated with increased abundance of the grass *Deschampsia,* suggesting that effects of nutrient additions on vegetation composition could depend on disease-induced shifts in competitive ability.

Future work on global change phenomena also must not ignore evolutionary processes: Chakraborty and Datta (38), for example, have demonstrated increases in pathogen fecundity over 25 infection cycles in an elevated CO_2 environment. In studies of powdery mildew, Laine (74) found that fungal genotypes differed greatly in the degree to which spore production was altered by nutrient and temperature treatments. The discovery of such genotype × environment interactions emphasizes that environmental changes will not occur independently of genetic changes in host and pathogen.

Finally, in addition to disease levels being potentially altered by changes in global variables, pathogens may play a role in ecosystem-level response to such changes. Mitchell (83) compared field plots of grasses that were or were not given fungicide treatments. He found that reduction of foliar diseases led to large increases in belowground carbon allocation, suggesting that the presence of disease reduces plants' ability to sequester carbon. A thrust of future work will be to explicitly examine how diseases affect ecosystem processes (46). As an example, Lovett et al. (77) summarized the potential effects of exotic pathogens and pests on forest ecosystem structure and function. They highlighted how key features of host and pathogen, including host specificity, mode of action of the disease, virulence, and dominance of the hosts in the forest may allow general prediction of future impacts. For beech bark disease, for instance, Lovett et al. (77) predicted that the combination of host specificity and a moderately lethal disease would alter carbon and nitrogen cycling and food web dynamics.

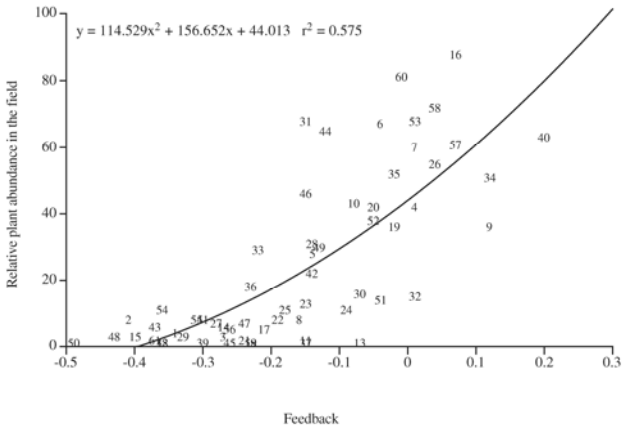

Fig. 9. Positive relationship between relative abundance of plant species in an early successional plant community and their soil feedback scores. Each number refers to a different plant species. To obtain soil feedback scores, seeds of each plant species were grown in soil that had a history of growth of either the same or different plant species. Net feedback scores can be negative (reduced growth of a species when grown in its own soil relative to soil from other plant species) or positive (increased growth of a species in its own soil relative to soil from other plant species). Reprinted by permission from Macmillan Publishers Ltd: Klironomos (70).

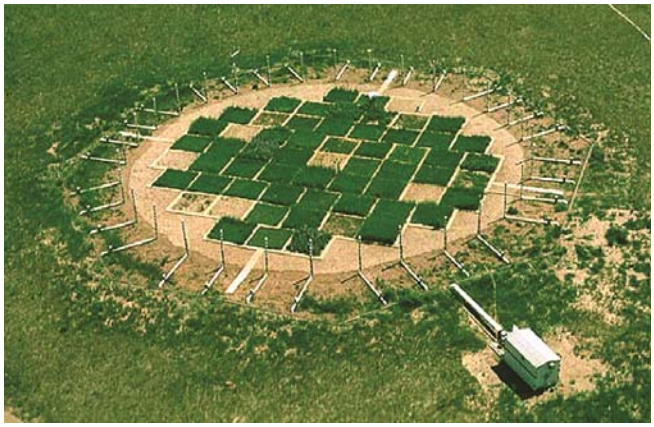

Fig. 10. The BioCON facility at the University of Minnesota can be used to study the effects of enhanced CO_2 on plants and pathogens under field conditions, such as reported by Mitchell et al. (87). There are six fumigation circles; each circle can be fumigated with either CO_2-enhanced air or air with ambient CO_2 concentrations. Individual plots are 4 m^2. Photograph provided by Cedar Creek Ecosystem Science Reserve at the University of Minnesota.

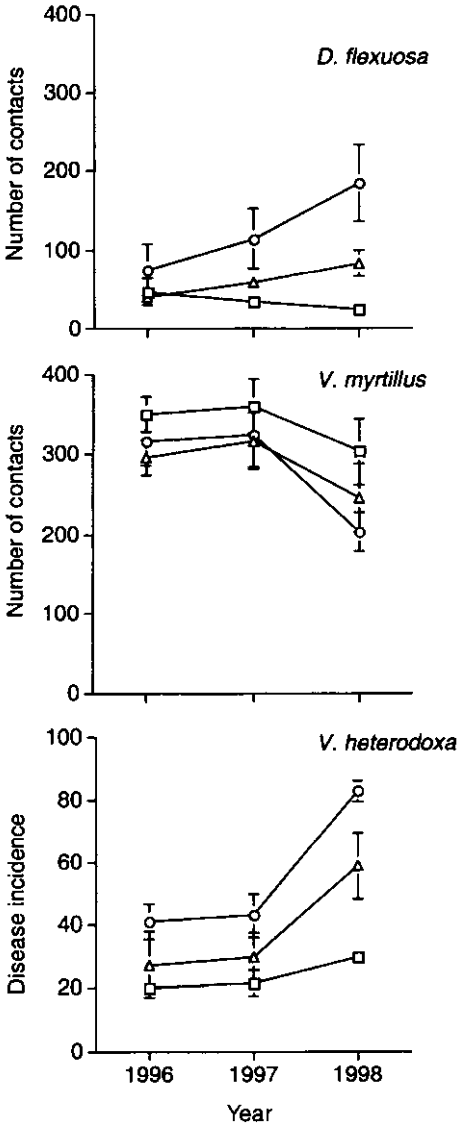

Fig. 11. Experimental addition of nitrogen to boreal forest plots led to an increase in fungal disease caused by *Valdensia heterodoxa* on the dominant shrub, *Vaccinium myrtillus* (bottom figure). Reduced abundance of the shrub (middle figure) and premature leaf-drop due to the disease led to increased abundance of the grass *Deschampsia flexuosa* (top figure). Square symbols refer to controls, triangles to low nitrogen addition treatments, and circles to high nitrogen addition treatments. Reprinted by permission from John Wiley & Sons, Inc.: Strengbom et al. (120).

Summary

Given the diversity of both pathogens and plants, generalizations are challenging. However, it is clear that pathogens can reduce survival and reproduction of individual plants, that disease can limit plant population growth, and that host–pathogen interactions at larger spatial scales (metapopulations, geographic distributions) are not necessarily predictable from small-scale studies. These ecological studies cannot be divorced from genetic investigations, given both the prevalence of genetic variation for resistance and virulence and the need for an evolutionary ecology approach to understand phenomena such as host shifts.

At the community level of organization, diseases can lead to increases or decreases in plant diversity. Research on generalist pathogens is particularly important, with recent work exploring both the ecological consequences of shared pathogens (e.g., pathogen spillover, apparent competition) and the role of plant phylogenetic relationships in host ranges. Studies of feedback between soil microbes (including pathogens) and plant species has been significant, in part because it provides new ways of explaining the coexistence of multiple species in plant communities. Community level research, in turn, often has links to ecosystem level studies. Recent work suggests not only that changes in temperature, greenhouse gases, and nutrients can alter disease levels but also that plant diseases and their effects can alter ecosystem properties. Studies on plant disease are thus an important component of global climate change research.

Future Directions

In 1977, John Harper stated, "The stage is ripe for bringing the ideas and techniques of plant pathology from its traditional area of concern with crops and man-managed forests to the exploration of natural vegetation" (61). More than 30 years later, numerous researchers have explored disease in natural plant populations, communities, and ecosystems, and made considerable progress in understanding their effects. This article has barely scratched the surface of this rich literature; interested readers will want to consult the original literature and recent reviews (33,54,86,93). As research continues to diversify, it is useful to reflect on this discipline, including asking those basic questions of *who, what, why, where,* and *how*.

Who? Obviously, researchers interested in disease in natural systems focus on pathogens, but this term includes a diversity of types of organisms. Early work often emphasized rust and smut pathogens that had obvious symptoms; in recent decades, research has broadened to include generalist foliar fungal pathogens, soil fungi and bacteria, nematodes, and viruses. We also must remember that a pathogen is not a species but a particular ecological role (C. M. Malmstrom, *personal communications*), and that many microbes can vary in their effects on plants. For example, recent work suggests that viral infection may be common in asymptomatic plants (90) in nature and that viruses may sometimes have positive effects on plant hosts (139). A current focus in research considers disease in the context of diverse symbiotic interactions (mutualism to parasitism), and postulates that coevolutionary trajectories may relate to the complexity and productivity of the environment (128).

What? Researchers tend to define their research disciplines with labels (i.e., ecology, genetics), potentially impeding important research at the interface of disciplines. For example, at the population level, there are still few studies that truly integrate population dynamics and genetic variation (129), and community ecologists are just starting to add knowledge of phylogenetic relationships among hosts to our understanding of multispecies interactions (56). Similarly, ecosystem research in disease ecology must be done with recognition that plants and pathogens will likely evolve in response to a changing environment (38).

Why? The ubiquitous nature of plant–pathogen interactions and the importance of disease to agriculture and forestry both justify interest in disease. Research on noncrop plants, however, may seem esoteric to plant pathologists working on real-world problems of crop health. However, many research questions in agricultural plant pathology have ties to studies of disease in natural systems (i.e., predicting the role of noncrop plants as pathogen reservoirs, understanding the evolution and function of resistance genes, using pathogens in weed biocontrol). Recent work has emphasized the landscape-level connections between managed and unmanaged plant populations and communities (32). For example, Fabiszewski et al. (47) included both crop landscapes and patches of unmanaged vegetation in models of soybean rust epidemiology and illustrated that noncrop plants (such as kudzu, *Pueraria montana* var. *lobata*) could play an important role in overwintering survival of the pathogen. Diseases in natural systems can also have immense economic effects, as may occur in the nursery industry due to sudden oak death. Finally, research on disease in natural systems can solidify important research questions and approaches that have practical benefit. Antonovics et al. (13), for example, illustrated how studies of fungal infection of a roadside weed allowed them to explore fundamental questions about emerging diseases that would be impossible to address in human or agricultural situations.

Where? To gain a truly global understanding of disease processes, we need research on plant–pathogen interactions across diverse biomes, and at a variety of spatial scales. Such research should occur not only in pristine natural communities, but also in the increasingly large proportion of the world where agricultural and natural communities coexist (44). Burdon and Thrall (32) emphasize the importance of the agroecological interface in generating pathogen diversity and in acting as a reservoir for crop diseases.

How? As always, integration of observational, experimental, and modeling approaches is needed, especially to address questions across multiple spatial scales. Collaborative teams of researchers are essential, and there are ample opportunities for interactions between scientists who identify themselves as plant pathologists and those who use terms such as ecologist or evolutionary biologist. Often the biggest challenges are not the biology of the organisms or the scientific questions, but our scientific cultures. Researchers working with plant disease in diverse systems need to meet each other, attend each other's meetings, and read each other's journals to increase the likelihood of future collaborations.

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