

The Current and Future Dynamics of Disease in Plant Communities

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Abstract

Pathogens are powerful evolutionary forces shaping the structure and dynamics of both individual species and of the communities of which they are part, at a broad range of genetic, ecological, spatial, and temporal scales. At all these levels their impact varies from the subtle and little recognized through to the most obvious destruction. Today the direct role of pathogens in natural plant communities is better recognized than at previous times, although the nuances of their interactions and the cascade of ramifications that can flow through changing biotic and abiotic effects are only now gaining recognition. However, as human influence on pathogens increases—either directly through enhanced if accidental dispersal, or through anthropogenic impacts on climate—we may expect to see increasing evidence of pathogens affecting plant species, community structure, and ecosystem function.

INTRODUCTION

Interest in the ecological and evolutionary role of parasites and pathogens in shaping populations, species, and community structure has grown dramatically over the past few decades (51), although little consideration has been given to the reverse issue, of how host community structure and composition influences pathogen population and community dynamics. Nevertheless, results emerging from studies of disease in both plant and animal populations suggest that broadly parallel processes operate in both types of interactions. Thus, in a recent review of animal—parasite interactions, Prenter and colleagues (110) showed that structuring may result from catastrophic disease outbreaks following invasions. However, they also argued that the predominant pressure exerted by parasites might be via the more cryptic consequences of sublethal effects whereby parasites simply shift the relative fitness of individuals within a species or of one species relative to another (e.g., apparent competition). Indeed, as Hudson points out (68), “the role of parasites in ecosystem functioning has usually been underestimated and poorly investigated because of their low biomass, low visibility, and small direct contribution to energy and material flows in natural ecosystems.” This message is just as pertinent for the interaction of plants with their pests and pathogens, and is one that has been implicitly or explicitly stated for some time (16, 17, 59). Through their impact on the dynamics of dominant or “keystone” species, pathogens control key processes (e.g., succession, nutrient cycling) that affect the diversity of communities and their long-term ecological and evolutionary trajectories.

This review provides a broad assessment of the impact of plant pathogens on their hosts and the communities of which they are part by sequentially examining the epidemiology of pathogens, the demography of their hosts, and, where pathogens have a fitness effect, the long-term consequences of those effects—

whether they are expressed genetically or numerically.

PATHOGENS AND THE DIVERSITY OF INDIVIDUAL HOST SPECIES

The basic building blocks of plant communities are individual species. While understanding of the functioning of complex multispecies communities is more than a simple addition of knowledge of the individual parts, recognition of the myriad of different ways in which individual hosts and pathogens interact provides a sensible starting point. Indeed, it has been considerations of the nature and strength of the evolutionary forces driving the evolution of resistance/susceptibility in hosts, and virulence/avirulence and aggressiveness in pathogens that have underlined the importance of the juxtaposition of host and pathogen life-history features. Thus, it is at the level of individual host-pathogen associations that the central importance of variation in mating system, longevity, demographic cycles, environmental responsiveness, and transmission mode (126) in determining the coevolutionary trajectories of individual host-pathogen associations and their genetic complexity is most clearly observed (24, 129).

Resistance diversity in plant populations evolves as a consequence of selective pressures imposed by pathogens fluctuating in abundance and severity of impact over time and space. The extent of such fluctuations may vary both within single host-pathogen associations, for example, due to environmental factors (119), or may range from endemic to epidemic in pathogens with distinctly different life histories. When resistance is controlled by a number of genes each with limited phenotypic effect (quantitative resistance) and pathogen lines show no differential specificity in their ability to attack individual host genotypes, host populations may still evolve resistance, although the differences between any two individuals may be

quite subtle (42, 111). On the other hand, when resistance is controlled by single genes with major phenotypic effects and pathogen lines show differential specificity (gene-for-gene-type resistance), major selective advantage/disadvantage turns on the particular combination of resistance and virulence in the population at any given time. Over time, many resistance genes may accumulate, leading to individual populations of considerable genotypic and phenotypic complexity.

The number of resistance genes detected in different populations and species varies dramatically. Although for quantitatively based systems no estimates of population resistance or virulence diversity are available, in species where clear distinctions in infection response between resistant and susceptible individuals have been demonstrated, some populations may contain many resistance genes whereas others only a few hundred meters away appear to carry none (73). Thus, estimated numbers of resistance genes in individual populations in natural plant-pathogen systems range from 1 (105) to 8 or more (18, 23, 44). Much of this variability stems from interactions between different intensity selective pressures applied by pathogens and genetic drift occurring in small, semi-isolated host populations. Theoretical studies also suggest that the diversity and distribution of resistance and virulence genes that evolve and persist may partly depend on aspects of life history and spatial structure that influence the degree of among-population asynchrony in disease dynamics (128). Differences in life history and spatial structure may also impact at the interspecific as well as the intraspecific level. However, at this stage it is impossible to attribute any real significance to interspecific differences in host resistance as they may simply reflect variation in investigative effort.

As Clay and his colleagues (34) have recently shown, many analogies can be drawn between the processes driving diversity in pair-wise host-pathogen interactions (e.g., the Red Queen hypothesis) and those affecting the diversity of whole communities. When ex-

amined from a genetic perspective, individual plant populations can be regarded as communities of clones, lineages, or types differing in their relative responsiveness to pathogen attack. Although individual resistance genes with major phenotypic effects and associated pathogen lines with differential specificity are typically thought of as characteristic of agricultural situations [and indeed have even been referred to as artifacts of plant breeding (8)], such resistance is widespread in a broad range of grasses (44, 89, 90), annual and perennial forbs (18, 19, 60, 61, 105), and shrubs and trees (77, 96, 130). As with the species composition of plant communities, the diversity or resistance gene richness of individual host populations varies considerably from population to population within a species, from species to species, and for that matter, between pathogens attacking the same species. A complex mix of individuals protected by different genes or alleles against a range of different pathogens is the likely consequence. The similarity of such structures to the diversity of species that comprise most plant communities is clear.

A growing body of evidence suggests a link between plant mating system and the severity of disease suffered by individual species (21, 27, 55). Within individual species the relevance of mating system is even more apparent (30, 76, 85) as, in most cases, the mating system of the plant species dictates the partitioning of resistance within and among individuals, thus playing a major role in determining the complexity of the resistance structure of local populations. For example, populations of apomictic species such as *Chondrilla juncea* may be composed of multiple clonal lines each with particular resistance genes in which all variation exists between individual lineages in the population as each parental line generates replicate copy daughters (45). In species in which recombination occurs, differences in the extent of selfing between different populations may result in dramatically different levels of population complexity and richness. Thus *Linum marginale* populations

from two geographically distinct areas (mountains and plains) showed marked differences in outcrossing rates (3% versus 30%, respectively), which, in turn, affected the pattern of distribution of resistance in the populations. The inbred nature of mountain populations resulted in most individuals being homozygous for the resistance they carried; on the other hand, a very significant proportion of individuals in the plains populations were heterozygous. This difference in distribution of resistance determines the overall diversity of the population and is likely to affect the rapidity with which the population responds to pathogen epidemics (23).

Intraspecific Genetic Diversity Affecting Disease Severity

In agricultural systems, genetic diversification of crops either through the use of intimate intraspecific mixtures or broader interspecific mixtures have been variously advocated as a means of reducing the depredations of pests and diseases. Here we briefly consider the relevance and potential impact of intraspecific mixtures on disease severity in agricultural systems where there has been extensive use of such approaches (largely because of the perceived importance of diversity in determining disease dynamics in wild plant-pathogen interactions), and then examine evidence from natural systems in more detail. The second type of genetic diversity—that of interspecific mixing—is considered in a later section.

Agricultural systems involving the use of varietal mixtures or multilines have repeatedly demonstrated the linkage between host and pathogen genetic structure and the capacity of a diversified community, albeit limited and artificial, to reduce disease severity and consequent pathogen-induced selection (139, 140, 144). Thus in a general review of the impact of multilines and varietal mixtures, Burdon found an overall ~8% drop in disease levels relative to monocultures (17), although reductions of >25% have been de-

tected in *Triticum aestivum* stands infected with *Pseudocercospora herpotrichoides* (95) or *Mycosphaerella graminicola* (40); and declines of 50% or more have been reported in interactions involving *Hordeum vulgare*—*Blumeria graminis* (141), and *T. aestivum*—*Puccinia striiformis* (46). Furthermore, as Newton and colleagues (97) have demonstrated with respect to 2- to 6-component barley mixtures challenged by *Rhynchosporium secalis*, there is evidence for a greater reduction in the size of the pathogen population as the diversity of the mixture, measured in terms of the number of components, increases. However, not all mixtures lead to disease reductions (54). This may especially be the case where host plant individuals are large and hence high levels of auto-infection may reduce the loss of inoculum on resistant individuals. Thus, in varietal mixtures of poplar clones the severity of *Melampsora larici-populina* infection was unaffected by host mixtures in one year, whereas reductions in infection were detected in a second year (92).

A long-standing perception is that disease loads in natural systems are much lower than those observed in agricultural systems. In fact, the rationale behind the use of mixture strategies in agriculture has been to reduce and manage disease levels by mimicking the diversity of natural populations. However, just what evidence is there that disease incidence is truly lower in comparable natural systems or that it relates to the genetic diversity of such systems (115, 127)? As discussed above, variation in resistance to pathogens both within and among local populations has been documented in natural plant-pathogen systems in a number of studies. In the latter case, consistent and absolute differences between different populations (i.e., all individuals resistant or susceptible) are frequently, although not exclusively (84), associated with broader geographic comparisons, reflecting differences in abiotic conditions that result in areas where disease is either consistently present or largely absent (119), or where mutual local adaptation of host and pathogen has resulted in

a marked divergence of the pathogenicity structure of the respective populations. On a more local (metapopulation) scale, chance events may lead to the establishment of populations with different gene frequencies, but the impact that this might have on disease severity summed across multiple populations within a given season is very difficult to estimate.

In contrast to the extensive information available regarding the performance of mixtures in agricultural systems, in natural plant-pathogen associations evidence that intrapopulational diversity for disease resistance contributes to lowered disease severity is rather limited and conflicting. Thus a study of disease severity in populations of *Linum marginale* attacked by the rust fungus *Melampsora lini*, found that the efficacy of disease resistance diversity was greatest in epidemic years (although not quite significant; $P = 0.09$ in two years) and nonexistent in years when pathogen incidence was low (127). [Rather intriguingly, this result runs counter to a study of the epidemiology of barley mildew where mixtures reduced infection more at low inoculum levels (98)]. In contrast, in populations of the apomictic crucifer *Arabis holboellii* attacked by the systemic rust pathogens, *Puccinia monoica* and *P. thlaspeos*, there was no consistent positive relationship between host clone frequency and disease incidence (number of individuals infected) within three populations. Clonally diverse populations did not necessarily have decreased disease incidence. Indeed, the population with the lowest disease incidence also had the smallest number of host clones (112). The complexity of the potential impact of host population structure on pathogen epidemiology was underlined in an experimental study of the effect of genetic diversity in *Solidago altissima* where levels of disease caused by *Erysiphe cichoracearum* varied through the epidemic cycle (115). At the beginning of the season disease severity levels tended to be higher in more diverse populations; however, as the growing season progressed, disease severity

in the more uniform populations became higher.

An important factor in considerations of the effects of resistance diversity is the extent to which that diversity correlates with overall resistance in host populations. In the *Linum* example discussed above, although resistance diversity had only low explanatory power, there was a clear negative relationship between the average resistance of host populations and disease (127). The observation that resistance-disease severity relationships may change within (115) or among seasons (127) clearly indicates that such relationships need to be investigated more generally in conjunction with multiyear epidemiological studies.

PATHOGENS AND THE DIVERSITY OF COMMUNITIES

Despite the significant upsurge in research on natural host-pathogen interactions over the past two decades, our understanding of the ramifications of plant-pathogen interactions at the community level is still rudimentary, although their importance in plant community structure and composition is becoming more widely recognized (51). The extensive experimentation using exclusion trials that uncovered the impact of animal grazing on community structure (58, 65, 74) has no equivalent in pathogen experimentation. Indeed, even the use of pesticide sprays on whole communities to control insect herbivory (13, 14, 29) has parallel experimentation in host-pathogen interactions. In trying to control the broad effects of multiple fungal pathogens in whole communities, a significant complicating effect has been the nature of fungicides and their impact beyond their intended targets (106). Thus potential phytotoxic or nutrient effects have provided significant control definition issues while soil-drench fungicides may affect beneficial fungi such as mycorrhiza (49) just as much as they affect soil pathogens. Indeed, where pesticides have been used to reduce fungal attack in whole communities,

treatment effects are often inconsistent. A study of the composition of a regenerating grassland detected transient changes associated with the diversity of the community or specific components (for example, perennial herbs) fluctuating both within and across seasons as disease was controlled (108).

As a consequence, to flesh out our understanding of the role that diseases play in shaping community structure, appeals have been made to a range of natural “experiments”—the impact of invasive pathogens on previously unexposed naïve host populations (and through these, impacts on the broader plant community), and the impact of pathogens deliberately introduced to control plant populations exploding outside their native range. These have been supplemented by field studies of natural pathogen-host interactions involving woody species where visible effects are less ephemeral than in situations involving herbaceous species, and a limited number of mixed species and artificially constructed community experiments (81, 94, 102, 109, 113).

Interspecific Genetic Diversity Affecting Disease Severity

In the agricultural arena, the use of interspecific mixtures to control insect populations in traditional low-input systems has been shown to reduce population sizes (2, 3). Although similar claims have been made with respect to the control of fungal diseases in traditional coarse-grain cereal mixtures (e.g., rye/wheat), specific testing of this hypothesis has been very limited. Not surprisingly though, given the effect of varietal mixtures, mixtures of barley and wheat also reduced the rate of increase of barley mildew (26). In more extensive studies, Vilichmeller (135, 136) showed the ability of rye/wheat and barley/oat mixtures to reduce the severity of the biotrophic fungi *Blumeria graminis* and *Puccinia recondita*, and to a lesser extent the necrotrophic pathogens *Drechslera avenae* and *Rhynchosporium secalis*. Barley/oat and barley/wheat mix-

tures also affected the incidence of the stem and root diseases *Pseudocercospora herpotrichoides*, *Fusarium* spp., and *Rhizoctonia cerealis*, although the results were strongly dependent on mixture composition. However, mixtures do not always lead to disease reductions. Thus in Californian grasslands, the alien species *Avena fatua* acts as a highly susceptible reservoir host for *Barley yellow dwarf virus*, significantly increasing the incidence and severity of the virus in a range of native grasses (87, 109) above that encountered in its absence.

An intermediate step between assessments of artificial mixtures in agricultural settings and the full complexity of uncontrolled natural communities is provided by a series of studies at the Cedar Creek Natural History Area in Minnesota, where fungal disease severity has been examined in small communities containing between 1 and 24 species—the species richness of the latter approximating to that of the natural grasslands of the area. An initial evaluation of four particular host-pathogen associations (*Liatris-Septoria*, *Lespedeza-Uromyces*, *Monarda-Erysiphe*, and *Schizachyrium-Colletotrichum*) showed highly significant negative correlations between disease severity and community complexity. However, in all cases, multiple regression analyses indicated that disease severity was highly dependent on host plant density, suggesting that reduced levels of disease in complex communities reflected reduced host density rather than an effect of species richness per se (81). When this approach was expanded to include the effect of diversity on the overall levels of disease suffered by all species in the community, a similar outcome eventuated. Thus the mean disease level on all species in the community was approximately three times as great in monocultural stands as in the most complex 24-species communities (94). Eleven individual diseases increased in severity as the richness of communities declined, but again in six of these, species richness had no effect on disease severity once the effects of host abundance were factored out. Interestingly, these experiments also

demonstrate that changes in plant species richness can impact on pathogen community composition as well as abundance—increased disease levels in the less diverse plots were associated with concomitant increases in the relative prevalence of host-specific foliar pathogens (94). Such feedbacks are likely to have further implications for disease dynamics and hence the potential for pathogens to influence plant communities properties.

The mechanisms whereby plant mixtures reduce overall disease loads has been the subject of much discussion, with declining host density and the interception of inoculum by nonhost individuals generally being recognized as the two most potent factors (15, 50). The architecture of individual cereal plants growing in high-density stands inevitably tends to favor spore dispersal away from the individual (i.e., reduced auto-infection) and hence sets up conditions conducive to inoculum interception by nonhosts. Despite this, evidence for significant reduction in disease from this source is very limited and seems to be detected only under high disease pressures. In contrast, in most nonagricultural systems variations in the numbers of one species may result in an increase in the size and or numbers of others, leading to a potential confounding of density and tissue abundance. It is not entirely clear what, if any, effect this may have had on the outcomes of the Cedar Creek experiments.

Natural Epidemics Changing Community Structure

The most compelling examples of native pathogens affecting the structure and diversity of plant communities are, not surprisingly, found among interactions involving pathogen-induced reductions in the density and abundance of large, dominant keystone species. Without doubt, the relatively small size of many herbaceous species and the speed at which all traces of an individual may disappear after death have not encouraged investigation of more modestly sized species.

However, the differential death of individuals of large dominant species while still at the seedling stage may be responsible for structural changes that are superficially impossible to identify. Thus for some time the Janzen-Connell hypothesis for tropical forest diversity (36, 72)—in essence that diversity is promoted by the failure of self-recruitment in the immediate vicinity of individual dominant trees because of species-specific, density, and distance-dependent seedling mortality—was little more than an attractive hypothesis. However, a series of studies of increasing sophistication have clearly demonstrated not only density and distance-dependent mortality of a range of tropical (5, 6) and temperate trees (104) but also the pathogenicity of fungi recovered from infected individuals (104).

In a similar way, Van der Putten and colleagues have made a strong case supporting the concept that sand dune succession on sea shores is at least partially driven by the action of pathogenic fungi and nematodes in the sand (131–133). At the front of the dunes where sand is being continuously replenished by the sea, there is virtually no evidence of the presence of pathogenic soil organisms and plant growth is vigorous. However, as dunes become increasingly stabilized and colonized by the grass *Ammophila arenaria*, this pathogen load increases substantially and new species encroach. Controlled experiments including the use of pesticides support the contention that successional processes are being encouraged by the detrimental impact of the soil pathogens on *A. arenaria*. Interestingly, in South Africa where *A. arenaria* is an exotic dune invasive, it appears to be limited by soil pathogens associated with native plant hosts (80).

More recently, a series of workers have investigated aspects of the interaction between soil communities, both pathogenic and mutualistic, and the growth of plants (9, 79, 91, 102). These diverse studies have all come to the same general conclusion, namely that feedback between soil organisms (for example, the soilborne pathogens *Cylindrocarpum*,

Fusarium, and *Verticillium*; 79) and plants is likely to be an important mechanism facilitating plant species coexistence and the diversity of communities. Another compelling example of the importance of plant-soil interactions in shaping community structure comes from work on spotted knapweed (*Centaurea maculosa*), which is a serious invasive weed in North America. Studies of this species in its native range indicate that soil pathogens there have negative effects on plant growth that may control abundance (thus promoting diversity). In contrast, *C. maculosa* appears to cultivate soil organisms in the exotic environment that result in positive feedbacks contributing to greater success and high abundance of the weed. This, in turn, results in overall reductions in plant community diversity, and may have further implications for feedbacks to the soil community. Thus in the native range of *C. maculosa*, coevolved soil communities may be tolerant of antimicrobial root exudates produced by the plant and so pathogens are less suppressed (28). This further underlines a general issue to do with feedbacks between plant and pathogen communities; not only do pathogens affect plant community dynamics, but the reverse is clearly also true.

Although such examples provide evidence of the complexity of pathogen-community interactions, none has the same persuasive power provided naturally by the broader patterns of disease and changing plant community dynamics generated by a range of soil-borne forest tree pathogens responsible for slowly expanding disease foci that create gaps in forest canopies (56, 116). Thus in the conifer forests of the Pacific Northwest of the United States, the laminated root-rot fungus *Phellinus weirii* acts as a keystone species influencing the speed and direction of successional events (37, 43, 56) as well as the complexity of the plant community as a whole (63, 64).

The conifer forests of the Pacific Northwest are dominated by *Tsuga mertensiana* and a range of other species including *Abies mon-*

ticola, *Pinus contorta*, and *P. monticola*. When the pathogen is absent for long periods of time, local microclimatic factors, particularly moisture availability, determine successional outcomes with a shift from a common *Pinus-Tsuga* early stage situation to *Abies*-dominated forests in mesic habitats and *Tsuga*-dominated ones in drier habitats (37, 43). None of the conifers composing these communities are immune to attack from *P. weirii* but the relative differences in susceptibility, especially of *T. mertensiana* and *Abies* species, are such that, when coupled with inherent differences in the environmental preferences of these two groups of host species, marked changes occur. On moist sites the diversity of the community within expanding disease foci remains essentially the same as that of unaffected forest, but successional processes are accelerated as later-stage *Abies* species become relatively more competitive and establish preferentially. *Abies* species are less suited to drier sites, however, and when infection foci establish in such situations, the death of *T. mertensiana* individuals precipitates a renewed cycle of establishment of *Pinus* species (37). The resulting cascading impact of such changes in the dominant tree layer on other members of the plant community in the shrub and herbaceous layers can be dramatic (64).

Similar to the impact of *P. weirii*, in Western Australia, *Armillaria luteobubalina* is responsible for a range of community patch dynamic effects as a consequence of the creation of disease centers in *Eucalyptus wandoo* woodland (116) and in coastal dune vegetation (117). In the *E. wandoo* case, however, infection centers were marked by greater vegetation cover than surrounding healthy forest, with a positive correlation occurring between overstory mortality and the occurrence of living understory plants. However, although plant numbers increased in infection centers, neither species richness nor diversity was affected (116). In contrast, the same pathogen attacking susceptible hosts from the Proteaceae, Mimosaceae, and Myrtaceae growing on ancient sand dune systems generated

infection foci with significantly less plant cover than in surrounding disease-free areas. Again, species richness was relatively unchanged but infection centers were dominated by a different suite of understory species (117).

Invasive Pathogens

In extant natural communities we see the subtle interplay of host-pathogen associations that have evolved over millennia. Pathogens still exert selective pressures on hosts and hosts on pathogens but, even where individuals die, the overall outward appearance of this reciprocal struggle is usually limited. There is at least the superficial appearance of a general homeostatic balance. In contrast, when pathogens encounter new hosts the consequences may be devastating with, in the extreme, major widespread and severe epidemics leading to rapid declines in host numbers and consequent changes to the structure of the community. Such emerging pathogens arise from one of two types of host jump. The first represents a true evolutionary change in which pathogens gain pathogenicity to host species already present in the community through some genetically based change such as chromosome loss (134), somatic hybridization, or recombination. Although such genetically based events have to date generally been less spectacular than those afforded by first encounters, they are nonetheless important and potentially more difficult to detect. Recent examples include the appearance of hybrids of *Puccinia graminis tritici* and *P. graminis secalis*. These hybrids are capable of attacking lines of *Hordeum vulgare* that neither of the parents can attack (22, 138). Similarly, in recent years the co-occurrence of *Melampsora medusae* (North American origin) and *M. larici-populina* (European origin) in New Zealand has resulted in the appearance of a somatic hybrid with a broader host range within *Populus* than either parent (121), whereas *Phytophthora alni* attacking *Alnus* spp. in north-western Europe appears to be of hybrid origin (11, 12).

The second type of host jump is represented by situations where pathogens are brought into contact with a susceptible host species for the first time (usually through some external anthropogenic event) (118), as occurred with the introduction of the Asian chestnut disease *Cryphonectria parasitica* into the naïve North American chestnut population. The most obvious effect of this invasion has been the simple replacement of chestnut by one of several codominant species (122, 142). However, where the forest was dominated by *Castanea dentata*, the loss of chestnut was often accompanied by an overall increase in diversity of trees and shrubs. Furthermore, interplay with changed microclimatic conditions has resulted in an acceleration of successional processes in moist sites, and the development of more xeric communities in drier ridge situations. Other examples of exotic pathogens inducing major, on-going change in plant communities include *Cronartium ribicola*, cause of rust of Asian pines, which has spread to European and Northern American species (78). By killing adult trees, the pathogen is opening up natural forests and thereby changing local environments. Similarly, *Fusarium circinatum* has spread from Mexican pines to cause pitch canker disease on *Pinus radiata* in California (52); *Phytophthora ramorum* has emerged as a generalist pathogen causing lethal infections on *Quercus* spp. and *Lithocarpus densiflorus*, thus changing redwood forest dynamics (88); while attack by the native South American rust *Puccinia psidii* on a range of plantation-grown eucalypts in Brazil provides an example of the introduction of a host species with pre-existing vulnerability into the native range of a pathogen (38).

Pathogens and Nutrient Flows

To this point we have considered the effects of direct interactions between pathogens and their hosts. However, this fails to recognize the broader potential ramifications for nutrient flows through communities that may result when species with large ecological

footprints are abruptly removed. All plant species have the capacity to affect the dynamics of nutrient flows through ecosystems. However, the extent to which they do is determined by interplay between the position they typically hold in the community, their population size and biomass, their longevity, and any biogeochemical processes typical of that species (for example, phytate excretion by the roots of lupins, affecting the solubility of phosphorus in the soil). Some of the most obvious and perhaps ubiquitous of such effects are seen in the impact of nitrogen-fixing plant-microbe associations. Thus, multiple examples exist of natural successional invasions, or of accidentally or deliberately introduced N-fixing legumes or actinorrhizal plants spreading and substantially changing nitrogen inputs in pastures and plant communities. In pastures and grasslands, changes in the relative dominance of grasses and legumes may lead to changes in soil pH, which, in turn, may affect the incidence and severity of soil-borne diseases (143). Similarly, in Hawaiian plant communities invaded by *Myrica faya*, soil nitrogen rose more than 300%, which, in turn, was associated with floristic changes as grasses became more abundant (137). It is not difficult to envisage that this could lead to a shift in the dynamics of natural host-pathogen interactions as increased foliar nitrogen levels can result in an immediate increase in the incidence of mildew and rust diseases (41, 101); similar effects have been documented with regard to the severity of outbreaks of insect herbivores (100). However, paradoxically, over time such increased disease may favor selection within the host population for resistance and ultimately lead to a negative correlation between total soil nitrogen levels and disease severity. Such an outcome is consistent with results obtained for the *Anthoxanthum odoratum*-*Blumeria graminis* interaction in the Rothamsted Park Grass Experiment (120).

The ultimate expression of the complex interaction between pathogens, host species, nutrient flows, and community composition

is particularly well illustrated by the cascading and unpredictable effects stemming from accidental introductions of pathogens or herbivorous insects of dominant plant species, especially where almost complete destruction occurs in diseased areas. One such example is afforded by the spread of beech bark disease (caused by *Neonectria* spp.) into mixed beech-hemlock-red oak-sugar maple forests. Studies of the movement of nitrogen through the soil, microbial, and plant pools of forested catchments in the Catskill Mountains, New York, have demonstrated that forest types individually dominated by these species vary considerably in the amount of nitrogen retained in the forest floor (86, 125). Forest floors under beech and hemlock take up and retain larger amounts of nitrogen than do those under maple stands. Yet maple may well largely replace beech and hemlock as the impact of the exotic beech bark disease and hemlock woolly adelgid (*Adelges tsugae*) destroy existing beech (53, 67) and hemlock (103) populations, respectively. A consequence of this interplay of differences between dominant species in the community and pathogen-induced mortality of a subset of those species is the possibility of very significant and widespread knock-on effects not only to the immediate community but also via changes in stream water NO_3^- composition (86) to river communities relatively distant from the disease epidemic.

Here we have focused on one specific example of the broad-scale ramifications that can flow from the introduction of a pathogen of a potential keystone species. Many other species fall within this category: *Phytophthora cinnamomi* dieback in Western Australia, America chestnut blight, oak wilt caused by *Phytophthora ramorum* in Europe and North America, and *Fusarium* dieback of *Acacia koa* in Hawaii (4). In all these cases, diseases of plants are bringing ecosystem changes (e.g., increased soil-water content and declining pH; 4) far in excess of the simple death of individual trees. Indeed, the consequences of these pathogen-induced changes are long-term, if not essentially permanent, and

because their final expression is often temporally or spatially separated from the pathogen epidemic, may not even be recognized as being due to such biotic influences.

PATHOGENS AND GLOBAL CHANGE—EFFECTS ON SPECIES AND COMMUNITIES

Invasive pathogens provide us with a series of examples of the potential power of these organisms to alter the structure of plant communities and, consequently, a range of physico-chemical variables. In recent years, the frequency of such invasion events has apparently increased with the increasing globalization of economic activity and the mobility of people. Although we should not expect the rate of change from this source to decline, changes in climate variables have the potential to unleash a new wave of major alterations in the dynamics of existing associations (114).

In gross terms, climate change is predicted to lead to increasing temperature and atmospheric carbon dioxide concentrations, and significant changes in aridity in both a positive and negative direction. Mean global surface air temperatures are predicted to increase in the range 1.4°—5.8°C by the end of the current century, although patterns of temperature change and the certainty of rainfall are likely to differ across the globe (66, 71). Thus both night-day and winter-summer mean temperature ranges are likely to reduce as minimum temperatures increase more than maximum ones; and continental and high-latitude areas will tend to warm more than coastal and lower-latitude ones. On average, global evaporation and precipitation are expected to increase, but distinct spatial differences should lead to areas of intensifying aridity as well areas of increased humidity and precipitation. Simultaneously, in many parts of the globe, particularly in the northern hemisphere, the effects of climate change on plant growth and pathogen activity will be further confounded by anthropogenic emissions of nitrogenous air pollutants and their

subsequent deposition (47, 48). As nitrogen is the most limiting nutrient in most terrestrial ecosystems, increased nitrogen availability will have, and has already had, profound effects on biodiversity in a number of ecosystems (99). The relative importance of pathogens in these vegetational changes has rarely been studied. However, as increased foliar nitrogen levels can result in an immediate increase in the incidence of a number of biotrophic pathogens (41, 124), the potential role of disease in nitrogen-induced vegetation change should be assessed. One example is provided by the vegetation shift induced by *Valdensia heterodoxa*, a common polyphagous pathogen, attacking *Vaccinium myrtillus*, the dominant plant of the understory in boreal coniferous forests. In this case, increased nitrogen availability favors the pathogen, resulting in premature leaf-shedding that, in turn, encourages a shift from *V. myrtillus* dominance to grass dominance (124). This increased grass dominance was caused by the increased light availability in patches severely defoliated by *Valdensia heterodoxa* (123).

The predictability of the impact of these factors on whole communities, their individual constituents—the plants, pathogens, vectors and so forth, and their interaction—is, not surprisingly, even more uncertain with both indirect and direct effects of varying magnitude being likely.

Indirect Effects

When climatic change has a significant and direct effect on plants, changes in competition may occur, which, given differential responses across plant species, may lead to relative changes in community composition, or when coupled with range extensions or contractions of individual species will lead to increased or decreased diversity of whole plant communities. In turn, such changes generate new opportunities or threats for associated pathogens. Thus pathogens of one host species⁹ may be brought into more intimate contact with previously unencountered

naïve hosts (although the likelihood of the spatial movements necessary for this to occur is perhaps low in the immediate future); may benefit from increasing overlap of obligate alternate host distributions; or may suffer significant reductions in population size as a consequence of the generation of allopatric distributions or incomplete congruence in the distribution of obligate alternate hosts. A potential example of changes that could result from variations in the relative distribution of alternate hosts is found in the distribution of *Pinus contorta* and its alternate host *Myrica gale* in North America. Currently, the distributions of these species do not completely overlap and the host-specific alternating rust fungus *Cronartium comptoniae* is found only in areas where both hosts occur. However, *P. contorta* populations from those areas show considerable resistance with, typically, less than 10% of trees being infected. In contrast, when samples from *P. contorta* populations from beyond the range of *Myrica gale* are challenged with *C. comptoniae*, up to 30% or more of trees are susceptible (69). Clearly, further expansion of the range of *Myrica gale* into that of *P. contorta* should result in increasing disease severity.

Increasing CO₂ concentration in the atmosphere may also affect individual plant-pathogen interactions through an increase in the efficiency of carbon fixation and a concomitant increase in growth and improvement in the carbon status of plants (70). Increases in leaf area and thickness, branching and tillering behavior, and stem and root length are all well-established CO₂ enrichment effects (35). The flow-on effects of such changes to other aspects of the nutritional status of plants—nitrogen, potassium, phosphorus levels and so forth, is less clear but the changed carbon-nitrogen ratio of tissues typically results in slower decomposition rates (7). Each of these morphological and nutritional changes has a clear capacity to affect the suitability of plants as hosts to a range of pathogens. Thus elevated CO₂ increased the pathogen load on C-3 grasses, possibly by decreasing water stress

and increasing leaf longevity and photosynthetic rate (93).

Similarly, pathogens that rely on biotic vectors for dispersal may see significant shifts in their distribution and or intensity if environmental changes affect the behavior and or viability of their vector. When keystone members of the plant community (for example, some tree species) are the host target, the knock-on effect may be large. Thus drier, warmer years that favor the growth and survival of *Scolytus* bark beetles that transmit Dutch elm disease (*Ophiostoma novoulmi*) may favor disease spread (57). The same scenario also applies to herbaceous species in which aphid-transmitted viral diseases tend to vary in severity from year to year, with outbreaks of *Barley yellow dwarf virus* being associated with mild winters (31).

Direct Effects

When we turn to considerations of the impact of the more unpredictable aspects of global climate change—drier/wetter, warmer/cooler environments—on pathogens themselves, it is again likely that over time we will see significant changes in host-pathogen interactions. Such changes are likely to be in both directions with, perhaps, increases in disease in mountain and high-latitude communities as the number of epidemic cycles/season increases, and declines in others as conditions become less favorable. In both cases this will result in changed selection pressures. Simple examples of these effects are provided by many rust fungi, which toward northern latitudes either fail to form overwintering teliospores, or show a gradual reduction or even suppression of the uredo stage (75). For these pathogens, any extension of the growing season is likely to result in an increased probability of teliospore formation, or larger pathogen population sizes following an increased number of uredinal cycles, respectively. However, among obligate host-alternating rusts such as *Melampsora pinotorqua*, alternating between *Pinus sylvestris* and *Populus tremula*, even minor

changes in the amount and timing of precipitation during the short period of shoot elongation when the pine is susceptible to infection in early summer, or during the spread to the alternate host (82), may drastically affect the disease dynamics.

Similar changes in disease occurrence and severity may reasonably be expected along altitudinal gradients. In southeast Australia, the overall severity of foliar disease caused by a suite of pathogens attacking *Eucalyptus pauciflora* showed a distinct decline with increasing altitude (20). In this and many other instances, it is easy to see how more benign environmental conditions (favoring greater off-season survival and faster reproductive rates) will increase the basic reproductive rate for pathogens (R_0) above one and allow pathogens to spread with potentially devastating consequences into host refuges that are currently free of disease. Indeed, for any pathogens that are vulnerable to experiencing severe population bottlenecks during off-season periods (many rusts and other biotrophic fungi), increases in survival during those periods (e.g., through greater host survival), may induce significant changes in both the intensity of disease and its predictability. As Harvell and colleagues (62) point out, there is a broad range of examples of plant pathogens that tend to induce more severe disease epidemics following mild winters. This increase in selection pressure and its greater predictability is predicted to favor the evolution of quantitatively based rather than qualitatively based resistance (24). The rate at which this occurs and the subsequent evolutionary interplay between host and pathogen could be critical to the ultimate impact of disease on species and genetic diversity (62).

At present the literature addressing the potential impact of global change on plant pathogens has a strong focus on increasing disease problems. However, at northern latitudes and at higher altitudes, snow-blight pathogens in which extensive mycelial growth occurs beneath thick winter snow pack consti-

tute an important part of the pathogen community on evergreen conifers [e.g., *Phacidium infestans* on *Pinus sylvestris* (10, 25)], and dwarf-shrubs [e.g., *Eupropelella vaccinii* on *Vaccinium vitis-idaea* (83)], where they affect both host survival and community structure. Any reduction in either the thickness or duration of snow cover is likely to have a negative impact on these important pathogens with subsequent effects on host survival and community structure.

While changes in the intensity and predictability of pathogen-induced selection may cause marked changes in host resistance, increases in CO_2 have also been correlated with changes in the aggressiveness (=area infected) of some isolates of the necrotrophic fungus *Colletotrichum gloeosporioides* when attacking lines of the host plant *Stylosanthes scabra* (32, 33). In comparisons involving growth at ambient and twice-ambient levels of CO_2 , the aggressiveness of pathogen isolates grown at the higher CO_2 level was significantly increased after 25 successive infection cycles but only following a marked decline during an initial lag period that lasted 10 generations (32). Interestingly, the susceptible variety of host developed a level of resistance to *C. gloeosporioides* at elevated CO_2 , but that of the partially resistant line remained unchanged (33). In contrast, in the controls (infection under ambient CO_2), aggressivity increased on both the resistant and susceptible host lines.

The balance that is struck in asexually reproducing fungi between increasing aggressiveness when grown on the same host over multiple generations (1, 39) and increased host resistance resulting from morphological and biochemical changes in plants growing at high CO_2 levels is one that is not immediately predictable. Indeed, we should perhaps take the results obtained by Roy and her colleagues, in a study of links between increasing temperature and disease, as a general warning for all general considerations of the impact of global change on plant disease—that while there was a general tendency toward increasing disease with increasing temperature,

some pathogens showed the opposite trend, suggesting unique individual host-pathogen responses (113).

SUMMARY

For millennia, pathogens have shaped the structure of individual plant populations, of whole species and of entire communities. While many of the processes involved are cryptic and unapparent, their legacy is the complex pattern of resistance/susceptibility, and virulence/aggressiveness found in virtually all host-pathogen associations today. During this process of evolution, periodic host jumps or movements of pathogens from one

spatial arena to another must also have resulted in occasional dramatic and uncontrolled epidemics [see elm decline (107)]. More recently, with increasing global movement of humans, such invasion events appear to have increased in frequency, being marked by classic examples of wholesale community change caused by chestnut blight in North America and *Phytophthora*-dieback in Western Australia. In coming decades, native plant communities will continue to be challenged by such invasions, to which will be added the unpredictable effects of changes to the dynamics of individual host-pathogen interactions resulting from global atmospheric changes.

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ERRATA

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