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The interaction between plant competition and disease

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Abstract

It is well documented that pathogens can affect the survival, reproduction, and growth of individual plants. Drawing together insights from diverse studies in ecology and agriculture, we evaluate the evidence for pathogens affecting competitive interactions between plants of both the same and different species. Our objective is to explore the potential ecological and evolutionary consequences of such interactions. First, we address how disease interacts with intraspecific competition and present a simple graphical model suggesting that diverse outcomes should be expected. We conclude that the presence of pathogens may have either large or minimal effects on population dynamics depending on many factors including the density-dependent compensatory ability of healthy plants and spatial patterns of infection. Second, we consider how disease can alter competitive abilities of genotypes, and thus may affect the genetic composition of populations. These genetic processes feed back on population dynamics given trade-offs between disease resistance and other fitness components. Third, we examine how the effect of disease on interspecific plant interactions may have potentially far-reaching effects on community composition. A host-specific pathogen, for example, may alter a competitive hierarchy that exists between host and non-host species. Generalist pathogens can also induce indirect competitive interactions between host species. We conclude by highlighting lacunae in our current understanding and suggest that future studies should (1) examine a broader taxonomic range of pathogens since work to date has largely focused on fungal pathogens; (2) increase the use of field competition studies; (3) follow interactions for multiple generations; (4) characterize density-dependent processes; and (5) quantify pathogen, as well as plant, population and community dynamics.

Key words: plant community composition, plant competition, disease, genetic variation, pathogen, plant population dynamics

Introduction

The prevalence of competitive interactions among plants is obvious to gardeners who thin their vegetables to increase the yield of individual plants. However, we remain surprisingly ignorant of the degree to which competition has altered the population sizes of plants, their genetic composition, or the composition of plant communities (Law & Watkinson 1989; Grace & Tilman 1990; Goldberg & Barton 1992; Tilman 1997). A major challenge is that competitive interactions are unlikely to be fixed properties of plant individuals or species but instead will vary with particular biotic and abiotic conditions (Ayres & Paul 1990; Clay 1990; Louda *et al.* 1990; Dunson & Travis 1991). In this perspective, we expand on the useful reviews of Ayres & Paul (1990) and Clay (1990) by discussing

the potential effects of pathogens on competitive interactions among plants. We address three general questions:

1. How does infectious disease alter intraspecific competitive interactions, and in what circumstances does this lead to changes in plant population dynamics?
2. If there is genetic variation in disease resistance, what are the implications of intraspecific competition for evolutionary changes in plant populations?
3. How does disease alter interspecific competitive interactions, and do these disease effects contribute to changes in plant community composition?

Ideally, a fourth issue should also be considered: the degree to which the above three aspects of plant competition in turn affect pathogen populations or communities. For example, competitive interactions among plants could reduce the strength of induced resistance (Karban *et al.* 1989) or alter susceptibility to infection. Unfortunately research on the fourth issue is limited and thus will not be addressed in any detail.

Before examining the possible interactions between plant disease and competition, we must highlight the fact that diverse definitions of competition exist (Law & Watkinson 1989; Grace & Tilman 1990). For the purposes of this paper, we define competition broadly as any type of interaction among individuals (either within or between species) which reduces fitness as measured by individuals' contributions to population growth. Our definition thus includes both resource-based exploitative competition as well as competitive interactions mediated indirectly by other species (apparent competition, Holt & Lawton 1994). In addition to problems of definition, a plethora of approaches for studying interspecific competition in the greenhouse and field have been introduced and debated (e.g. Grace & Tilman 1990; Snaydon 1991; Goldberg & Barton 1992; Watkinson & Freckleton 1997). For two species interactions, the most illuminating information is obtained from complex experimental designs that employ a wide range of total densities of both species in all combinations of relative densities (e.g. Law & Watkinson 1987). In contrast, the simpler "de Wit replacement series" or "substitutive designs" where total plant density is kept constant and the frequency of the species varies is difficult to interpret. In particular, these designs are problematic because intra- and inter-specific competitive interactions are confounded (Jolliffe *et al.* 1984; Snaydon 1991). However, as will be evident from the following pages, many studies of competition and disease are based on these simpler experimental designs. One explanation is simply logistics: it is difficult enough to have a sufficient number of treatments and replication to study competition, but this number must be at least doubled to study competitive interactions in the presence and absence of a pathogen. Since our goal in this perspective is to address a diversity of questions about disease and competition, we have chosen to discuss as many empirical studies as possible, despite the use of less desirable designs in some cases.

A perspective on "competition and disease" also must address what is meant by a plant pathogen. Disease-causing agents include organisms from diverse taxa including the fungi, viruses, bacteria, and nematodes. A more useful classification is focused on functional categories. For example, Burdon (1991) broadly arranges pathogens into classes defined by their effect on the demographic parameters of survival, fecundity, and individual growth. His categories are thus "killers" (for example, diseases of seeds and seedlings), "castrators" (such as sterilizing floral diseases), and "debilitators" (many diseases which retard plant growth). In considering just the fungi, Jarosz & Davelos (1995) suggested five categories defined by the physical site of pathogen activity: soil-borne pathogens (including everything from damping-off diseases of seeds and seedlings to persistent root diseases of trees); canker/wilt diseases such as chestnut blight; foliar, local lesion diseases such as many rusts; nonsystemic floral diseases such as the ergots; and finally the systemic diseases with fungal growth throughout the plant, as exemplified by some infections of grasses caused by the Clavicipitaceae and some smuts and rusts. Regardless of the classification scheme used, our knowledge of infectious diseases in nature is not evenly spread across pathogen groups: we know most about fungi, and within fungal diseases, there is far more work on the rusts and smuts than, for example, pathogens that cause soil-borne diseases.

Several broad issues must be addressed to explore the interaction between plant competition and disease, regardless of whether the focus is on population dynamics, evolution, or

community composition. For example, one needs to understand the degree to which pathogen population dynamics directly depends on the abundance and other characteristics of focal host populations. Some specialist pathogens, for instance, can infect only a single plant species and thus their population sizes may be directly dependent on their host's numbers. In contrast, the population size of a generalist soil pathogen which also obtains nutrition from nonliving organic matter may be independent of the abundance of a particular host species under study. This distinction between specialists and generalists has been addressed in modeling population dynamics of predator-prey and host-parasitoid interactions and also should be considered for host-pathogen interactions (Hassell & May 1986; Holt & Lawton 1994). A related issue is that plant competitive interactions in the presence of disease may be affected by the nature of disease transmission, such as whether the rate of disease spread is greater in dense plant populations. Similarly, the temporal nature of disease development in the plant population in relation to key demographic events may affect competitive interactions among individuals. For example, are primarily seedlings or reproductive plants infected, and does disease occur before or after density-dependent effects due to other factors? Spatial questions to consider include whether diseased plants are highly clumped or randomly located in the plant population. Overlaid on top of temporal and spatial patterns of infection is the issue of how disease affects individual plants: to what degree does disease alter plant survival, growth, or reproduction? For diseases that have a debilatory effect on plant growth, a crucial issue is the extent to which the individual plant (or its component parts) can compensate for losses due to disease. Finally, since it is unlikely that all plants in the population or community will have equal levels of disease, one must consider the compensatory ability of neighbouring plants of the same or different species, both in terms of growth of currently existing individuals and new recruitment.

Disease and intraspecific competition: significance for population dynamics

Initially, we focus on ecological effects of disease within a single plant population and how this alters intraspecific competition, and potentially population dynamics. Clearly, few species exist in monospecific stands. However, an understanding of the simpler interactions in a single-species stand is useful before we introduce the more complex situation of interspecific competitive interactions. We must be aware, however, that the presence or absence of other plant species in the community can greatly alter the effects of disease on intraspecific plant interactions.

To examine how a pathogen could alter its host's population dynamics, we begin with a conceptual model that has been used to explore the combined effects of plant competition and herbivory (Louda *et al.* 1990) and that can be modified to examine disease effects. The basic idea is that the impact of disease on plant population size involves "overlaying" disease effects on pre-existing patterns of density-dependence in per-capita growth rates. For simplicity, we assume that the population grows continuously and that age- and stage-structure can be ignored. In the absence of the disease, the growth dynamics of the plant population is given by $\frac{dN}{dt} = N\,f(N)$ (N, population size; $f(N)$, per capita growth rate, i.e. per capita births minus per capita deaths), as a function of N. For example, if $f(N) = r\left(\frac{K-N}{K}\right)$ (r, intrinsic rate of growth; K, carrying capacity), the plant population shows logistic growth. One can envision effects of disease as a reduction in growth rates by an amount D (e.g. a "killer" pathogen increases mortality). The population growth equation is then modified to $\frac{dN}{dt} = N\,f(N) - D$. Such reductions could at times be density-independent (for example a soil saprophyte will occasionally act as a pathogen, with a per-capita reduction in growth of a constant d', i.e. $D = d'N$), but often infectious disease may be more prevalent or effective at higher host density, leading to $D = d(N)N$, where $d(N)$ depicts density-dependence in the per-capita expression or impact of the disease. In turn, $d(N)$ can be written as the product of two components, i.e. $d(N) = p(N)i(N)$, where $p(N)$ is the probability that an individual will be infected at all, and $i(N)$ is the average reduction in growth rate (i.e. a measure of the reduced contribution to population growth) of infected individuals.

Density-dependence in disease thus could include density-dependence in *prevalence* (the *p(N)*) and/or density-dependence in individual *effects* (the *i(N)*).

In Fig. 1, the solid lines depict how the per capita growth rate of a plant population might decrease with increasing plant density, in the absence of disease. For a given carrying capacity, density-dependence can be linear (line a and b), as in the logistic model, but with different slopes (strengths of density-dependence) depending on the intrinsic rate of increase (the y intercept). The qualitative form of density-dependence can also be nonlinear and fit other forms. In line c, for example, crowding effects occur strongly at low densities, but further increases in density have little additional effect on per capita growth rates. By contrast, line d refers to a situation where per capita growth rates are not altered by density up to some threshold density, beyond which there is a sharp reduction in growth rates. The equilibrium population size of the plant in the absence of the pathogen is that value of *N* where the per capita growth rate is 0, the "carrying capacity" of the population (determined by resources, for example). To explore the effects of a pathogen, the dashed lines illustrate the superimposed reduction in the per capita growth rate due to disease. In the example shown, this impact could be either independent of host density [$D = d'N$] (line e) or density-dependent [$D = d(N)N$], with increasing impact at higher host density (line f). The equilibrium density of the plant population in the presence of the pathogen is determined by the intersection between the solid and dashed lines. Finally, we note that we have not illustrated one other possibility: inverse density-dependence, where pathogen impact declines with plant population size ($d(N)$ declining with N). For instance, if an animal vector disseminates the pathogen from one plant to another, and the vector population size is fixed and its activity is limited, an increase in host population size may dilute the per capita rate of infection (analogous to predator satiation).

The continuous nature of population growth in this model means it cannot be directly applied to most plant populations in nature, where population growth occurs in discrete time intervals and age- and stage-structure are important. However, the model does highlight several important features. First, the

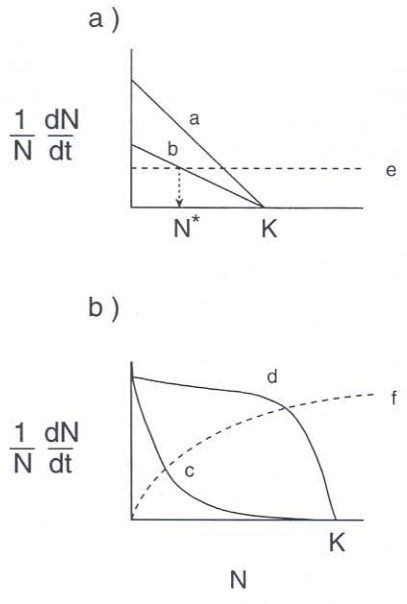

Fig. 1. Effects of intraspecific density dependence and pathogenic infection on equilibrium plant population size. Solid lines in each graph depict per-capita growth rates as a function of density. The equilibrium population size in the absence of disease is shown as "K". (a) Linear density-dependence; lines a and b refer to logistic density-dependence; the dashed line e depicts the reduction in per-capita growth rate due to density-independent infection. (b) Nonlinear density-dependence. With line c, there is an initial negative effect of increasing plant density on per-capita growth, but subsequent additions in density have little effect on per-capita growth rates. In contrast, line d shows no effect of density on per-capita growth rates until above a threshold density. The dashed line f depicts density-dependent infection. With the assumption that the effects of intraspecific competition and pathogen infection are additive, the density at which a given pair of solid and dashed lines intersect (in either graph a or graph b) indicates the equilibrium plant population size in the presence of disease, e.g. in graph a, N* denotes equilibrium abundance for a species with the inherent growth properties of line b, as reduced by density-independent infection (e). For simplicity, the figures depict density-independent infection overlaying logistic growth and density-dependent infection with non-logistic growth; more generally, one might also observe cases of density-dependent infection (f) combined with logistic growth (a or b), or density-independent infection (e) with nonlinear growth (c or d). See text for additional details. This figure is modified from Louda *et al.* (1990); the model is based on continuous population growth and does not incorporate age or stage structure.

greatest numerical effect of a consumer (including pathogens) on equilibrium sizes of plant populations will be when intrinsic growth rates are low (compare line b to a) and when plants are unable to compensate at the population level for pathogen-induced losses (e.g. line c). Second, density-dependent effects of disease on per capita growth rate (e.g. line f) means that the disease has a proportionately higher impact at high density plant populations, but that the pathogen may not cause extinction of the plant population because of the latter's ability to increase when rare. This scenario may be likely when considering highly-specific pathogens whose own abundance would tend to be low when their specific hosts are rare.

Empirical studies are generally supportive of the conclusions of this model, though many examples do involve age/stage-structure effects that are not included in the graphical model. For example, the importance of plant compensation at the population level has long been recognized. De Wit, in his classic 1960 treatise, discussed the "compensation power" of plants: the yield depression in a field crop with a certain percentage of diseased plants was often less than would be predicted based on yield of fields with 100% diseased individuals (de Wit 1960). The compensatory "power" could be quite high, as in de Wit's description of a study where healthy potato plants adjacent to plants with a leaf roll disease had increased growth relative to growth of healthy plants in a totally healthy population. However, for pathogens that only caused high levels of disease late in the season, yield decreases due to the presence of disease were not compensated by increased growth of healthy plants. Compensation can be studied with various plant characters: for example, both biomass and number of flowering capsules of *Portulaca oleracea* were greatly reduced in stands of 100% virally infected plants, but there were few differences among stands that were either 1:1 mixtures of healthy and diseased plants, or all healthy (Friess & Maillet 1996).

Compensatory responses can vary for the same system under different environmental conditions. For example, Mihail *et al.* (1998) sowed seeds of a legume, *Kummerowia stipulacea*, at a wide range of densities in greenhouse pots that either were, or were not, exposed to either *Pythium irregulare* or *Rhizoctonia solani* isolates. Higher mortality was caused by *R. solani*, but subsequent growth of surviving plants led to similar total plant biomass regardless of pathogen species. However, for both pathogen treatments, individual plants could not fully compensate for disease losses. In a field study of the same plant and similar pathogens (Alexander & Mihail, unpubl. data), by contrast, dramatic compensatory effects were observed. The percentage of sown seeds that survived to be seedlings was very low in pots to which fungal spores were added, but was independent of sowing density. Hence, disease mortality was density-independent, as in line e (Fig. 1). In spring, the few survivors in the fungal-addition pots were also small. However, by fall, the average plant size was actually highest in pots with added fungi, apparently due to the release of surviving plants from intraspecific competition.

We often are ignorant of the degree to which the per capita impact of disease is dependent or independent of host population density (i.e. the "dashed" lines in Fig. 1). In several studies, such data are lacking because plants were deliberately inoculated, leading to a constant disease level across all density treatments. Under situations with natural disease spread, it is often unrecognized that a density dependent effect of disease on per capita growth (i.e. line f) can occur in two different ways: either there is a greater proportion of diseased plants at high densities due to density-dependent disease transmission (the $p(N)$ term), or the same incidence of disease has a greater per capita effect at high plant densities (the $i(N)$ term). Density-dependent disease transmission has been demonstrated in a few experiments (e.g. Burdon & Chilvers 1975) and is a possible explanation pertaining to studies where a higher proportion of diseased plants are found as host density increases (Long & Cooke 1969; Burdon & Chilvers 1982; Augspurger & Kelly 1984; Gilbert *et al.* 1994). Very strong density-dependent transmission could have interesting effects on plant population dynamics, leading to a "humped" recruitment curve where total seed production per population is actually highest at intermediate plant densities. Although theoretically interesting because it could contribute to cyclic fluctuations in plant population size (Symonides *et al.* 1986), this phenomenon appears generally to be unlikely in plant populations (Crawley 1990) and has only rarely been explored with

regard to diseased plant populations (Mihail *et al.* 1998). An example where the same level of disease had a greater per capita effect at high plant densities is provided by Lively *et al.*'s (1995) study of rust infection of the annual *Impatiens pallida*. A field survey revealed that the proportion of plants that were infected was not related to density, but the effect of infection on plant growth was higher under the naturally high densities, than when plots were experimentally clipped to achieve a lower density. It is likely that variance in pathogen effects among host individuals also increases with host density.

The simplicity of the Louda *et al.* (1990) model of course does not capture the detailed nature of interactions between healthy and diseased plants, which has been the subject of considerable study using rust infection of groundsel, *Senecio vulgaris* (Paul & Ayres 1986, 1987b). In their work, plant populations were established with various densities or frequencies of healthy and/or diseased plants. Diseased plants were produced by inoculation of individual plants. In general, the effect of disease on infected plants was enhanced with increased intraspecific competition. For example, using de Wit replacement designs, Paul & Ayres (1986) demonstrated that size hierarchies of populations with 100% healthy or 100% rusted plants were similar, but in mixtures of healthy and diseased plants, the healthy plants were in the largest size categories whereas diseased plants were smaller. Moreover, suppression of plants due to the combined effect of disease and competition varies with environmental conditions. Using sunken tubs in the field exposed to different watering regimes, Paul & Ayres (1987b) found disease altered competitive interactions only under drought conditions.

The presence of infection does not always alter competitive interactions. With the systemic anther smut infection of *Silene dioica*, Carlsson & Elmqvist (1992) found no differences in individual growth of healthy compared to diseased plants grown in monoculture or mixtures. Their conclusion is, however, statistically weak because mortality in the field experiment reduced sample sizes. Garcia-Guzman *et al.* (1996) also reported no effect of disease on competitive ability in grasses in a replacement series study with grasses infected by a systemic floral smut over a range of densities. As is true of all studies using a de Wit design, it is difficult to separate competition between plants of the same infection class and competition between plants of different infection classes. For example, because treatments with either only healthy or only diseased plants were not established over a range of densities, one cannot distinguish the absence of competition from a situation of equal competitive abilities of healthy and diseased plants (Snaydon 1991; Watkinson, pers. comm.). A reduction in competitive ability of floral smut infected grasses was reported at a combination of high density and low nutrients (Garcia-Guzman *et al.* 1996). Infections in grasses caused by fungi in the Clavicipitaceae present another interesting counter-example, since infected plants can be *more* competitive than healthy plants of the same or different species. These patterns are strongest when herbivores are present, since infected plants have toxic effects on many kinds of consumers (Clay *et al.* 1993; Clay 1997a). Evidence of the importance of endophytic interactions to plant-plant interactions comes from competition studies, but also from research documenting the increase in frequency of infection over a period of several years in planted grasslands (Clay 1997a). In many studies, the increase in frequency of endophytic infection must be explained by the increased competitive ability of infected plants coupled with vertical transmission of the endophyte via clonal vegetative growth or seeds since contagious spread of the infection is rare.

Besides the more obvious resources of water or nutrients, pollen or pollinators may also limit plant populations and thus lead to competitive interactions. With the systemic anther-smut disease of plants in the genus *Silene* caused by *Microbotryum violaceum* (formerly *Ustilago violacea*), sterilized flowers on diseased plants produce spores instead of pollen. Consequently, the presence of disease should reduce pollen production in the population and potentially mean that seed production is limited by pollen availability. To test this hypothesis, Carlsson-Graner *et al.* (1998) performed hand pollinations of the dioecious plant *Silene dioica* in populations with different levels of disease, as well as in populations where they experimentally created a skewed sex ratio. Pollen limitation, as demonstrated by an increase in seed production on a whole plant basis with hand-pollina-

tion, was only demonstrated in the most extremely female biased populations and did not differ among populations with low *vs.* high disease incidence. Floral diseases could also alter pollination if the presence of spores on healthy flowers inhibited seed production. This phenomenon has been demonstrated with anther-smut disease in three different *Silene* species (Alexander 1987; Marr 1997, Carlsson-Graner *et al.* 1998). Even if these effects do not directly alter population dynamics, they could have important consequences for relative fitness, and the relative importance of gene flow from external pollen sources.

For all types of pathogens, the nature of intra- (and inter)specific competitive interactions in the presence *vs.* absence of disease will depend on spatial patterns of disease incidence and how this patterning compares to the distances at which plants compete with each other. Disease is often spatially aggregated in plant populations as might be expected due to both the mechanisms of spatially limited disease transmission and aggregation of similar host genotypes and microsites conducive to disease development (Real & McElhany 1996). Theoretical (Watve & Jog 1997) and empirical (Burdon & Chilvers 1976) studies have examined the consequences of host clumping for disease transmission but we are unaware of attempts to consider plant competitive interactions in the context of clumped spatial patterns of disease. For example, if diseased plants are aggregated with respect to the distribution of the entire plant population, diseased plants will primarily compete with each other and there may be thus less opportunity for compensatory responses of healthy plants. Similar ideas have been considered for herbivores (Crawley 1983).

These diverse examples suggest that diseases can alter intraspecific competitive interactions in a variety of ways. Largely unanswered, however, is the degree to which these processes lead to changes in population dynamics of host plants. From a theoretical perspective, more attention needs to be paid to the population dynamic consequences of compensation for pathogen damage; the insights of Crawley (1983) on compensation for herbivore damage are likely to be particularly relevant to this issue. Empirical multi-generation studies are also often lacking (with the exception of agricultural mixtures; see later section). Quite simple field studies can provide a lot of information: for example, Carlsson & Elmqvist's (1992) seed addition experiment is an elegant demonstration of how seedling recruitment can be limited by availability of seeds at sites with high levels of disease. Such studies are important, as the effects of disease on individual plant attributes is not necessarily the same as its effect on population size in subsequent years (Crawley 1990). Long-term studies are also needed to detect delayed density-dependence: for instance, the compensatory ability of neighbouring plants could potentially lead to the absence of numerical effects of disease within a single generation, but the build up of the pathogen population through time could impact the size of later host generations.

Disease and intraspecific competition: significance for evolution

The above discussion on intraspecific competition and disease did not consider why some plants are healthy and others are diseased within populations. Differential infection of plants can be due to numerous factors including the vagaries of plant microsite, plant size, and plant location with regard to conspecifics. Of interest here is when genetic differences among plants affect the incidence and severity of disease and the degree to which this phenomenon interacts with competition to determine the genetic composition of plant populations. Genetic variation in disease levels among plants (both due to traits affecting probability of infection as well as more classical variation in disease resistance) is often found in natural plant populations (reviewed in Alexander 1992). Potential interactions between resistance variation and competitive ability were first suggested by biocontrol studies of the apomictic plant, *Chrondrilla juncea* (skeleton weed), in Australia. Australian populations of *C. juncea* consist of different frequencies of three biotypes. In the 1960's, populations of *C. juncea* were dominated by biotype A. With introduction of a rust isolate specific to biotype A, biotype A numbers decreased dramatically, followed by expansion of the numbers of biotypes B and C (Burdon *et al.* 1981). These

field observations led to greenhouse studies on the effect of the rust on competitive interactions between biotypes. Using a replacement series design, Burdon *et al.* (1984) grew biotype A (susceptible) and biotype C (resistant) at a range of frequencies; plants were either inoculated or not inoculated with the rust fungus. By the second harvest, data on size hierarchies revealed that plants of the susceptible biotype dominated the larger size classes in the absence of the disease but in the presence of disease, the resistant biotype was competitively superior. This switch of competitive ability due to the presence of disease was central to Burdon's (1982) conceptual model of the interaction of disease and competition. Burdon (1982) argued that when susceptible plants were found at a low frequency in a mixture, disease levels should be low. If susceptible plants are competitively superior in the absence of disease (as suggested by the *C. juncea* system), the susceptible component should increase in frequency through time. Eventually, however, disease levels should rise as the frequency of the susceptible component increases. Under such conditions, the resistant component should subsequently increase in frequency. Burdon (1982) argued that this frequency-dependent shift in competitive hierarchy arising from host genotype-specific infection should contribute to the maintenance of resistance variation in a population. Burdon's conceptual model was developed into a mathematical model by Gates *et al.* (1986) who concluded that certain combinations of model parameters could lead to long-term maintenance of both susceptible and resistant components, but that the maintenance of diversity was not an inevitable outcome. Similar effects emerge from analogous models of herbivores attacking mixtures of susceptible and resistant plants (Holt *et al.* 1994).

No direct tests of the Gates *et al.* (1986) model have been published. Many of the essential components of the model are, however, likely to be important in any consideration of the evolutionary consequences of disease and competition. For example, the frequency or density (these terms are equivalent when total density is fixed) of susceptible plants affects disease levels in the Gates *et al.* (1986) model; specifically, disease levels will be higher with a greater density of susceptible plants. If disease levels are higher with a greater density of susceptible plants, it follows that susceptible plants will have low disease levels when they are at low abundance in a population. The strongest evidence for this relationship comes from the agricultural literature on "multilines" or cultivar mixtures, where a mixture of varieties of the same crop species are planted in a field. Commonly, susceptible components in such mixtures have lower disease than when planted in monoculture (e.g. Finckh & Mundt 1992). Explanations for this reduction include the lower density of susceptible plants in the mixture (as in the Gates *et al.* 1986 model), but also other processes such as trapping of spores by resistant plants and induced resistance (Mundt & Browning 1985). Analogous studies in natural systems, however, provide less compelling evidence for density- or frequency- dependent infection. Jarosz & Levy (1988), for example, established experimental field populations with different frequencies of susceptible and resistant *Phlox* plants. The proportion of resistant plants in the population did not alter the development of mildew epidemics. A negative result was also found by Alexander (1991), who found similar rust levels on sunflower plants (*Helianthus annuus*) grown either with plants of the same half sib family or with a mixture of plants from many different families that differed in susceptibility. Roy (1998) performed a reciprocal transplant study that involved planting clones of the crucifer *Arabis holboellii.* Clones that were "rare" at the transplant site did not have lower rust levels; since fungal isolates infect more than one clone, a rare clone may not be perceived as an uncommon type to a pathogen. Only Schmid's (1994) study is consistent with the general concept of lower disease in more genetically diverse plant populations. At the end of a season, plots of *Solidago altissima* composed of replicate plants of the same clone had higher mildew levels than plots composed of increasingly more diverse genetic composition. Thus, despite the intuitive appeal of the idea that susceptible genotypes should have low disease levels when found at low density within a population, little empirical evidence is available from natural systems. An added complication is that susceptible genotypes may be rare because of disease.

Another assumption of the Gates *et al.* (1986) model is that susceptible plants have a higher competitive ability in the absence of the disease. This assumption exists in the

model for at least two reasons: first, Burdon *et al.* (1981) did find this relationship in the *C. juncea* system and second, in the absence of this assumption, resistance is always favoured and thus the question of maintenance of genetic variation in the model would be moot (except as maintained by mutation or gene flow). To examine the empirical basis for this assumption requires an entry into the rather controversial subject of "costs of resistance", since a restatement of Gates *et al.*'s (1986) assumption is that resistant plants have a fitness cost of resistance expressed in reduced competitive ability. Bergelson & Purrington (1996) recently reviewed the ecological and agricultural literature to document the extent of costs of resistance. Studies included in their review had to meet several criteria, including adequate control of genetic backgrounds so that any fitness costs could be directly attributed to the resistance genes. Burdon *et al.*'s (1981) study of *C. juncea* was, for example, not included in Bergelson & Purrington's (1996) review because apomictic biotypes have complete association of all genes and thus the reduced competitive ability of the resistant biotype cannot be assumed to be due to the resistance trait. The net result of the review is that 56% of the pathogen resistance studies did find evidence for costs of resistance. However, most studies of resistance costs are based on plants grown singly or in stands of constant densities, and thus do not measure fitness costs in terms of competitive ability. Unfortunately, it is dangerous to conclude that decreased fitness of plants grown singly or in monoculture is indicative of poorer competitive ability in a mixture (for example, a tall variety may have relatively low yield in monoculture but have high yield in a mixture with a shorter variety).

Disease resistance costs as measured in competitive ability thus have not yet been conclusively demonstrated in the empirical literature. Recent theoretical work also suggests that purely genetic models may overestimate the need for very large resistance costs for maintenance of genetic variation in resistance (Antonovics & Thrall 1994). Consideration of spatially structured populations further alters interpretations of resistance costs. For example, when resistance costs are absent, the spread of resistance genes to fixation is much slower in a spatially structured population than in a single panmictic population (Thrall & Antonovics 1995). A focus on spatially structured populations is particularly relevant to consider with pathogens, because disease outbreaks are likely to be sporadic in occurrence and highly localized in space.

We conclude that the evolutionary consequences of disease and competition in host populations is in need of a fresh look. From a theoretical perspective, models that incorporate both a numerical and gene frequency component seem promising (Antonovics 1994). Additionally, quantitative genetics approaches may be required given the nature of traits such as "competitive ability"; see Salonieni (1993) and Hochberg & Holt (1995) for quantitative genetics approaches to predator-prey interactions. Models could have built into them transmission parameters that depend on density or frequency of susceptible plants as desired and preferably should incorporate spatial structuring in both competition and disease transmission (Antonovics *et al.* 1997). Finally, in contrast to the skeletonweed example and Gates *et al.* (1986) model that focuses on selfing lineages, new approaches will need to account for multi-locus effects in species with free recombination.

Disease and interspecific competition: significance for community composition

Some of the earliest discussions of plant disease and competition in natural systems focused on potential implications for community composition. For example, Chilvers & Brittain (1972) proposed a model for the coexistence of two hosts that depended on the existence of host-specific pathogens for each host type. Although one host species could competitively exclude the other in the absence of disease, competitive dominance of one host could be countered by the build-up of the pathogen population on that host, leading to long-term coexistence. These results are intuitively appealing, but many dynamical outcomes are possible when multiple species of pathogens and plants interact. For instance, Seger's (1992) two-host, two-parasite, model leads to stable limit cycles. Similarly, Gates *et al.*'s (1986) model, which can be applied to community as well as population genetic questions, has a variety of out-

comes depending on choice of initial parameter values. The above models can be considered in the context of a rich theoretical literature on impacts of predators and other natural enemies on species coexistence (Yodzis 1986; Holt *et al.* 1994; Chesson & Huntly 1997), which in a general way can be reinterpreted to apply to host plant-pathogen interactions. Rather few models to date, however, incorporate important realistic features of plant-pathogen systems, such as spatial localization of interactions.

Empirical studies of the effects of pathogens on plant community composition follow two general approaches (Law & Watkinson 1989). One approach involves establishing communities with different compositions and following changes in species abundances over time. Ecologists rarely use this approach, partly because of the long life time of the species they study. However, agricultural biologists have established cultivar mixtures of wheat and followed them for multiple generations in the presence or absence of disease. Because of the high rates of selfing, these cultivars essentially act as distinct species in a community. In Finkch & Mundt's (1993) study, four cultivars were initially sown at equal proportions, but over multiple generations their frequencies changed in the absence of disease due to differences in competitive ability. Such changes were accentuated in the presence of disease because in this particular case, resistant cultivars were also better competitors (i.e. no cost of resistance). Long-term studies are also underway to explore the effects of endophytic infection on plant communities. Clay (1997a) developed a conceptual model, in which the frequency of infection first increases within grass species over time due to enhanced vigour of infected individuals. These intraspecific interactions in turn have community effects, as species with high levels of endophytic infection increase as a consequence of their enhanced resistance to herbivores and increased competitive ability. The predicted result is a decline in species diversity and dominance by a few endophyte infected species. Clay's ongoing experimental study of the effect of endophytic infection on vegetation in old-field communities is providing results that are generally consistent with this model (Clay 1997b).

An alternative approach for studying competition is to do single-generation experiments that incorporate a range of plant densities and use these results to infer long term changes in community composition (e.g. Law & Watkinson 1987). Most empirical studies of disease and interspecific competition, for example, consist of growing mixtures and monocultures of two species (a host and nonhost) in the presence or absence of the pathogen for a single generation (e.g. Paul & Ayres 1987a; Paul 1989; Ayres & Paul 1990; Chen *et al.* 1995; Ditommaso & Watson 1995). A crucial issue to address in the future in such experiments is the performance of a species when it is rare: are results consistent with an increase in its numbers over time? If this pattern exists for each species, maintenance of multiple species in a community might be expected. The conceptual model presented earlier (Fig. 1) further suggests that components of density-dependence (i.e. density-dependence in host intrinsic growth rates as well as in disease incidence and impact) should be explicitly examined in experimental studies.

To review the current literature, we focus on studies of the groundsel, *Senecio vulgaris*, and the host specific rust fungus *Puccinia lagenophorae*. In work by Paul & Ayres (1987a), populations of *S. vulgaris* and lettuce were grown in the presence or absence of the rust fungus. Importantly, populations of single species were also grown over a range of densities in addition to the mixture plantings. Thus, the yield of each species in mixture could be compared to its yield in a single-species stand planted at the same density. Due to this experimental design, it was evident that the effect of the disease was primarily to release the nonhost (lettuce) from competition; yield of lettuce in monocultures was equivalent to the yield of lettuce in mixtures of lettuce and rusted groundsel for comparable lettuce densities. In contrast, the degree to which groundsel yield in competition differed from groundsel yield in monoculture was not greatly altered by the presence or absence of the rust fungus. Parallel results (i.e. presence of the disease leading to enhanced growth of the nonhost but little effect on the host) were also found in a similar study with groundsel and *Euphorbia peplus* (Paul 1989), even though *E. peplus* (in contrast to lettuce) is a poor competitor against groundsel. The *E. peplus* study further revealed that the enhancement of growth of the non-host was not experienced by all individuals; a minority of nonhost individuals of greater than average size were

released from competition when disease was present. In field studies, Paul & Ayres (1987a) found that if lettuce was grown with increased densities of healthy groundsel, yield was decreased. However, if groundsel was rusted, lettuce yield was unchanged up to a threshold groundsel density, and then reduced, suggesting relevance of the rust to modulating competition in weed/crop interactions.

Researchers working with soil-borne diseases have also explored the question of disease and interspecific competition. Van der Putten & Peters (1997) considered the role of disease and competition in a successional setting. On sand dunes, *Ammophila arenaria* is replaced in a successional sequence by *Festuca rubra*. Previous studies suggested that host-specific nematodes and fungi that accumulate in the roots of *A. arenaria* lead to the decline of this species. Using a replacement series design, van der Putten & Peters (1997) found that growth of *A. arenaria* was relatively poorer in nonsterile compared to sterile soil, while *F. rubra* performance was either not affected or enhanced. Bever *et al.* (1997) further discuss the prevalence of negative feedback interactions between plants and their associated soil communities and suggest that this phenomenon is one explanation for coexistence of plants with similar competitive ability.

Yet another way pathogens may alter interspecific plant interactions is demonstrated by Roy (1996) working with systemic rusts of the crucifer *Arabis holboellii* that cause the development of "pseudoflowers", clusters of rust infected leaves that look and smell like flowers. Roy examined whether the presence of pseudoflowers led to facilitation or competition for insect visitation to a co-occurring species of *Anemone patens*. In general, the presence of pseudoflowers enhanced insect visitation to *A. patens*. However, the pseudoflowers also produce spermatia (effectively fungal gametes) that can be deposited on stigmas through insect visits. Roy found that flowers of *A. patens* which were experimentally augmented with a mixture of pollen and spermatia had lower seed production than did flowers that were only hand-pollinated. This latter phenomenon suggests an unusual mechanism by which infected individuals of one plant species can compete with a second plant species.

Finally, pathogens can influence competitive interactions between plant species in a very different way. We noted earlier the importance of the degree of host specificity of a pathogen: non-specialist pathogens can link the dynamics of two or more host populations. Even if two plant species are not directly competing for a limiting resource, each plant species may have an indirect negative effect on each other's population growth if they share a common pathogen. This "apparent competition" (Holt & Pickering 1985; Holt & Lawton 1994) develops because increased densities of one host species leads to increases in the pathogen population, which in turn can have a detrimental effect on population growth of the other host species (and vice versa). The existence of such indirect interactions can complicate interpretations of field studies: removal of one plant species followed by increased performance of another plant species could be due to apparent competition as opposed to greater availability of a resource (for examples see Connell 1990; Reader 1992).

Explicit studies of apparent competition in plants due to shared pathogens have not been done, to our knowledge. However, many well-known phenomenon in phytopathology clearly relate to this concept, such as the reduction of wheat stem rust in North America following eradication of barberry, the alternate host (Stakman 1934) or concerns about weed hosts being "reservoirs" for viruses that infect crops (Duffus 1971). A possible example of apparent competition outside of agriculture concerns the generalist fungus *Phytophthora cinnamomi* (Weste 1986). This pathogen has led to major changes in the *Eucalypt* forests in Australia, by converting mixed woodlands to areas dominated by sedges. Since plant species differ in the degree to which their growth is reduced by the pathogen, sedge invasion may result not only because of its probable greater competitive ability but also because it can harbor a pathogen that has a greater impact on other species in the community. Similar ideas have been proposed by Rice & Westoby (1982) who speculate whether there is an evolutionary advantage for host species to be infected by pathogens that also infect alternate host species which are their competitors. Exploration of the ideas of apparent competition emphasizes that disease "tolerance" requires more attention. Although difficult to define and measure (Clarke 1986), strong tolerance refers to a phenomenon

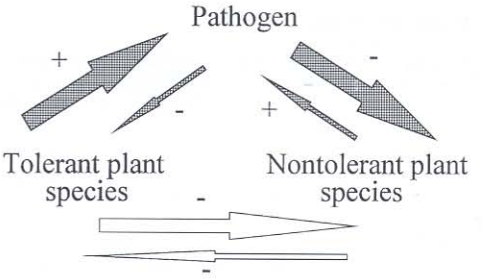

Fig. 2. A conceptual model of asymmetrical apparent competition due to shared pathogens. A pathogen infects two hosts; one is tolerant (infection has little effect on plant fitness) while the other is not tolerant (infection has large effects on plant fitness). Arrows indicate the effect of one species on the population growth of another species. The "+" and "–" signs depict whether a species has a positive or negative effect on each other, with the size of the arrow indicating the magnitude of the effect. Shaded arrows are direct interactions while clear arrows illustrate indirect interactions that result from the direct interactions. Asymmetry of the indirect interactions results because the positive effect of the tolerant species on pathogen population growth is large whereas the negative effect of the pathogen on this plant species is modest. In contrast, the pathogen has a large negative effect on the population of the nontolerant species. The nontolerant species can have a small positive effect on the pathogen population (as shown here) if infection of the nontolerant species leads to stunting, for example, and thus few pathogen propagules emerge from the nontolerant species. Alternatively, the positive effect of the nontolerant species on the pathogen may be equivalent to that of the tolerant host. The latter situation would reduce, but not eliminate, the asymmetry of the indirect interaction.

where there is little or no reduction in plant fitness despite infection. Tolerance may play an important role in community dynamics because apparent competition between tolerant and non-tolerant species will tend to be asymmetrical (Fig. 2); additionally, it is a subject of increased interest for evolutionary ecologists (Simms & Triplett 1994; Roy & Kirchner, unpub. data). Note that tolerance is not necessarily an "all or nothing" phenomenon but may occur at different levels or degrees (i.e. infection reduces plant fitness for two different strains or species, but one is more affected than the other). Finally, the importance of apparent competition is modulated by density-dependence in plant growth (Holt 1977); host species with low carrying capacity and/or intrinsic growth rates are not likely to exert strong effects via this mechanism on an alternate host species (Holt & Lawton 1994).

Conclusions

Despite several empirical studies, we cannot provide truly satisfying answers to any of the three questions on how disease and competition affect plant population dynamics, genetic composition, or community composition. In particular, five points need to be stressed in light of future research.

1. *The need to expand our knowledge of different taxonomic and life history groups.* Our current studies are highly biased towards studies of fungi, with little knowledge of viruses, bacteria, or nematodes. A true understanding of the interaction between disease and competition needs a broader range of species. Similarly, work has been heavily biased towards studies of short-lived plant species. Competitive interactions are certainly important in disease interactions in forests (Gilbert & Hubbell 1996), but the long-term consequences are understandably more difficult to study.
2. *The need for more field research.* More studies must be conducted under field conditions, so that plants experience natural patterns of disease transmission. To investigate how disease levels and competition interact, as a first step the constant levels of disease that result from artificial inoculation make sense. However, our actual focus is on how the disease process interacts with competition, and thus experiments with natural disease levels are needed. Field experiments of all kinds are important: in addition to experimental designs that allow measurement of competitive ability (Watkinson & Freckleton 1997), simpler experiments are also of value. Lively *et al.*'s (1995) thinning experiments on disease impacts or Morrison's (1996) study on the effect of neighbouring plants on disease incidence in *Juncus dichotomus* are examples. Field studies also will allow apparent competition and many other indirect interactions (e.g. Dobson & Crawley 1994) to be explored. Apparent competition is open to experimentation, for example, by establishing treatments where

species co-occur but resource competition is prevented (perhaps by growing plants in pots in the field). Studies in the field will also emphasize the need to expand our competition focus from two species to multi-species communities.

3. *The need for long-term experiments.*
   Our research must follow the lead of agriculturists in including experimental treatments that persist for more than a single generation. Besides providing a true test of changes in population size or community composition over time, long-term studies allow detection of "carry-over" effects of infection that impact multiple generations, such as infected plants producing smaller seeds than healthy plants (Jarosz *et al.* 1989).
4. *The need to explicitly examine density-dependence.*
   As emphasized by the graphical model (Fig. 1), density-dependent processes in both host and pathogen populations can have important consequences for ecological processes. However, we largely lack experimental studies of these phenomena.
5. *The need for consideration of pathogen population and community dynamics.*
   We must not forget that pathogen populations are themselves potentially changing in abundance and frequency. De Wit (1960) noted this problem in his interpretation of reduced competitive ability of infected potato plants. He stated, "as far as competition goes it should be concluded that the percentage of leaf roll diseased plants decreases rapidly in time which is of course not true because leaf roll is an infectious disease"! We also must realize that "disease" is not a fixed treatment in an experimental design, but instead recognize it as a potentially dynamic entity.

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