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The Ecological Genetics of Plant-Pathogen Interactions in Natural Communities [and Discussion]

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The ecological genetics of plant–pathogen interactions in natural communities

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Plant–fungal pathogen interactions are among the most convenient systems in which to study the full complexity of co-evolved associations. Despite this, the potential ramifications of pathogen-induced damage in plant communities have yet to be widely recognized.

Pathogens affect host fitness by reducing fecundity or increasing mortality or both. The consequences of infection by pathogens that have an immediate effect on survival or that totally suppress reproduction are determined relatively easily. However, individual lesions of the type caused by many pathogens are not seriously damaging and their ultimate effect on fitness parameters is often separated temporally from the actual presence of disease symptoms. For annual plants this separation may be relatively short, whereas in perennials both reproduction and survival through periods of stress may be correlated with disease incidence in previous seasons.

A causal relation between pathogen-induced reductions in host fitness and demographic and genetic changes in plant populations has yet to be proven. However, circumstantial evidence strongly supports this hypothesis. Natural host populations have often been found to be variable for resistance. Presumably this reflects past selective pressures exerted by the pathogen, but the pattern of the variability can also be influenced by host features such as mating system. Recent work on populations of *Glycine* spp. (wild soybean) has documented the existence of race specific resistance, which is consistent with the hypothesis that gene-for-gene interactions are an important feature of natural plant–pathogen associations.

Pronounced spatial structuring also occurs between closely adjacent populations of both host and pathogen. The major factors which could influence this structuring are small-scale changes in the physical environment, founder effects and localized genetic differentiation in both the host and the pathogen.

Models based firmly on realistic ecological and epidemiological assumptions are beginning to provide a picture of the importance of population size and dispersal ability on the stability of natural plant–pathogen interactions.

Introduction

Plant biologists have a paradoxical view of the role of pathogens in plant communities. None would deny the devastating effect that the introduced pathogens causing Dutch elm disease (*Ceratocystis ulmi*) and chestnut blight (*Endothia parasitica*) have had on previously unexposed host populations of elm and American chestnut in Europe and North America. However, there is a tendency to ignore completely the relatively infrequent pustules, blotches and flecks that are more everyday signs of the presence of similar organisms exploiting most, if not all, plant species.

In this respect even plant pathologists, who know better than most the various ways in which pathogens may decrease the fitness of their hosts, have often concluded that a lack of frequent, devastating epidemics can be directly equated with a lack of effect or, at the very best,

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with a benign situation arising from prolonged exposure and selection. Indeed, many plant pathologists writing from an agricultural perspective seem to believe that some kind of *pax natura* exists between pathogens and their hosts in natural communities (Browning 1974).

We believe this view to be false. Rather, there is a continuing interaction between host and pathogen. Plants tend to remain relatively disease-free for much of any particular growing season. This is largely due to the host's genetic composition, growth pattern and spatial distribution, but is also aided by conditions of the physical environment. It is only when optimal environmental conditions for a pathogen coincide with its presence that disease rises to noticeable levels and epidemics of generally limited spatial scale and duration occur. Although these disease epidemics may occur for only a short time, the effect on the plant population may be dramatic.

In this paper we attempt to elucidate the ecological and genetic consequences of plant–pathogen associations for both hosts and fungal pathogens. The paper is divided into three sections. The first two have a heavy empirical bias, considering first the fitness consequences to the host of naturally occurring levels of disease and second the patterns of resistance or susceptibility and virulence or avirulence found in naturally occurring plant–pathogen associations. Because most studies have focused attention on the host an uneven treatment is inevitable. The third section addresses how host and pathogen may interact through time, pointing out some of the major unresolved biological requirements and consequences of current theoretical models.

The effect of pathogens on host fitness

For pathogens to play a role in shaping the size and structure of plant populations they must reduce individual host fitness. Certainly they can do this, but there are many ways in which disease can affect the fitness measures of fecundity and survival.

The simplest and most extreme cases of pathogens exacting a fitness penalty are those in which individual plants are killed before they reproduce. There are two major groups of disease that regularly have such an effect. Members of the first group strike at the seedling recruitment phase of the host's life cycle causing pre- and post-emergence damping-off syndromes. The actual contribution such diseases make to seedling mortality often goes undetected because infected individuals may disappear rapidly. One particularly dramatic exception is the well-documented role such damping-off diseases play in affecting the distribution and survival of seedlings of various tropical trees (Augspurger 1984; Augspurger & Kelly 1984). For six species, mortality induced by damping-off accounted for 12–74% of all seedlings although mortality around parent plants exceeded 80%. Several other examples of damping-off diseases causing seedling losses exceeding 10% are known (see, for example, Bloomberg 1973; King 1977).

The other major group of pathogens that have an analogous fitness effect are the smuts, bunts and such fungal endophytes as *Epichloe typhina* (choke) that induce sterility in their hosts. Although plants infected by these pathogens frequently survive to maturity they fail to reproduce. Intriguingly, however, the growth and longevity of plants infected by these pathogens can be significantly greater than that of healthy individuals. Thus in the grass *Agrostis tenuis*, infection by *Epichloe typhina* is accompanied by greater vegetative vigour and preferential survival under harsh grazing régimes (Bradshaw 1959).

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The effect of pathogens on the fitness of hosts they kill before reproduction is relatively easy to determine. In contrast, the effects of pathogens that merely debilitate are much more difficult to assess. However, these partial reductions in survivorship and fecundity should not be overlooked as they represent a very real selection pressure on the host. The major problem that arises is that of linking the presence of disease at one stage in the host's life cycle with its ultimate effect on a fitness parameter later. In the broadest sense a positive correlation exists between the severity of disease and its effect on host survival and fecundity. However, the relation is rarely simple, with both the timing and duration of disease occurrence being critical variables in the equation. This is illustrated in the response of different members of a winter-annual population of the weed *Capsella bursa-pastoris* to infection by *Albugo candida* (white rust) and *Peronospora parasitica* (downy mildew) at different stages of the life cycle (Alexander & Burdon 1984). Individuals that were attacked as young seedlings soon after the autumn germination subsequently developed systemic infections and died before flowering. Plants infected early in the following spring survived to flowering but were less fecund than individuals that remained free of disease or that did not become infected until flowering began.

Here a combination of early infection and a period of environmental stress seems to be significant in exacting the maximum fitness penalty from those plants infected before winter. Recently Paul & Ayres (1986) have documented a similar phenomenon in populations of *Senecio vulgaris* (groundsel) infected with *Puccinia lagenophorae* (rust). In that association the pathogen has a major effect on the fitness of individuals in winter-annual populations but little effect on that of individuals from spring-annual populations.

The full complexity of the relation in annual plants between disease (with all its complicating variations in severity, duration and timing) and the fecundity and longevity of affected individuals has been most thoroughly explored in graminaceous hosts attacked by mildews and rusts. Disease early in the vegetative stage of many cultivated (e.g. *Hordeum vulgare*) and wild grasses (e.g. *Avena barbata* and *A. fatua*) tends to affect fecundity mainly through a reduction in the production of fertile tillers (Scott & Griffith (1980) and Burdon (1982), respectively). Similar levels of disease at, or just before, anthesis may reduce both the number of seeds per head as well as their individual size. In turn this may lead to further subtleties in the effects of pathogens on host plant fitness.

To date it has been generally assumed that for annual plants an accounting of fitness effects could be made at the end of each generation. However, we have found that this practical convenience may underestimate fitness penalties, as the depredations of pathogens in one generation have an effect on the number of descendants of such affected plants for up to two subsequent generations (A. M. Jarosz, J. J. Burdon & W. J. Müller, unpublished data). In an experimental wheat–powdery-mildew (*Erysiphe graminis tritici*) system we found that seed gathered from diseased and healthy plants at the end of one generation and classified, by weight, into 'large' and 'small' categories, gave rise to plants that showed significant differences in fecundity at the end of the first post-epidemic generation. In the absence of competition, plants derived from 'diseased' seed of either size category showed a mean 23% reduction in fecundity compared with plants derived from 'healthy' seed. In the presence of competition the situation became less clear, but 'healthy' and 'diseased' plants derived from seed taken from matched size categories still showed that disease in one generation gave rise to a carry-over effect on the fitness of offspring in the subsequent generation.

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In some ways the problem of an inter-generation fitness carry-over effect in annual plants is analogous to the extreme separation of pathogen attack and its subsequent expression in altered host fitness parameters that may occur in perennial plant species. Recently we have followed the fate of individual members of a natural population of the perennial flax species *Linum marginale* growing at Kiandra in the Snowy Mountains of southern New South Wales, Australia. During the first season an epidemic of the rust pathogen *Melampsora lini* occurred, and by monitoring the severity of rust on individual plants and their subsequent reproductive performance the pathogen was found to have no apparent effect on fecundity (table 1).

TABLE 1. REPRODUCTION AND SURVIVAL OVER WINTER OF A POPULATION OF *LINUM MARGINALE* GROWING AT KIANDRA, NEW SOUTH WALES, IN RELATION TO DISEASE CAUSED BY THE RUST PATHOGEN *MELAMPSORA LINI*

(A. M. Jarosz & J. J. Burdon, unpublished data.)

plant group	average disease severity[a]	number of seed capsules per plant: normal	number of seed capsules per plant: aborted	survival over winter
high disease	6.3%	11.5	7.3	21% (13/62)
medium disease	3.0%	11.5	6.1	36% (23/64)
low disease	1.1%	13.5	5.0	42% (37/88)

[a]Average area of green tissue infected per day over the 154 day growing season.

However, continuing assessments of the host population the following spring showed clearly that the disease markedly influenced the over-winter survival of infected plants. Although the risk of winter mortality was high for all plants, those that had been heavily infected with rust during the previous summer were twice as likely to succumb as those only lightly infected (table 1). Both immediate and delayed fitness effects have been observed in populations of *Phlox* spp. infected with powdery mildew (*Erysiphe cichoracearum*), where the current season's fecundity and subsequent winter survival were lowered following infection. Moreover, the effect of the disease epidemic carried over into the following year when the reproductive output of the previously infected plants was reduced relative to disease-free clones (M. Levy, A. M. Jarosz & B. Pugesek, unpublished data).

THE GENETIC AND DEMOGRAPHIC CONSEQUENCES OF HOST-PATHOGEN INTERACTIONS

The potential and actual effects of pathogens on the fitness of individual hosts growing in natural communities having been established, it would be pleasing to be able to point to specific empirical examples where the changes wrought by a pathogen have been closely followed over the early stages of particular associations. It is at this stage that the effects of selection are likely to be most obvious. Unfortunately, at both the demographic and the genetic level, such data do not currently exist.

Massive demographic changes in individual plant populations and in the structure of whole communities have been noted occasionally (for example, the total elimination of a population of *Echinocystis lobata* (wild cucumber) by an unidentified disease (Silvertown 1985); and changes from a *Eucalyptus*/*Banksia* woodland with a thick shrub layer to an open woodland

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dominated by sedges that follow epidemics of *Phytophthora cinnamomi* (die-back) in southeast Australia (Weste 1981)). However, although these examples document extreme cases of the effects of pathogens, they give little indication of their effects under more normal circumstances. What is needed are controlled field experiments in which disease levels are manipulated.

At a genetic level the quality of examples is similarly deficient. For the most part, we must resort to observations of the occurrence of resistance in host populations and assume that these reflect past episodes of selection. From this we can speculate as to the extent to which pathogen-induced damage may favour the emergence of resistant genotypes within a species or encourage the invasion of non-host species.

Before going on to such considerations it is necessary to touch briefly on the phenotypic expression and genetic basis of the major types of resistance that are found, and in particular those that have been monitored in wild plant populations.

Disease resistance in plants

Genetically based disease resistance in plants can be divided into two broad categories. Passive resistance can result from past interactions and encompass mechanisms whereby the detrimental effects of disease are at least partly absorbed by infected plants or where they lead to an avoidance of contact between host and pathogen (e.g. precocious germination or the closed flower habit of many cereals). By contrast, active disease resistance mechanisms occur as a result of continuing interactive responses between hosts and pathogens, and may be subdivided into resistance that is recognized by differential interactions between host and pathogen genotypes (race specific resistance) and that which is apparently expressed against all pathogen genotypes (race non-specific resistance).

Race non-specific resistance may be expressed in various ways including reductions in the frequency of successful host penetration, colony size and spore production. This form of resistance reduces the rate of pathogen increase during an epidemic. The genetic control of race non-specific resistance is poorly documented but, in most cases, appears to be under polygenic control with each gene having only a small phenotypic effect.

Race specific resistance, by contrast, is expressed as differences in the visual appearance of disease symptoms. In a susceptible host, a completely compatible reaction results in disease symptoms characterized by large, profusely sporulating lesions. Resistant or incompatible reactions may range from a complete lack of microscopically visible symptoms through non-sporulating lesions to sporulating lesions with restricted fecundity. In general, race specific resistance is controlled by single dominant genes that are inherited according to simple Mendelian principles.

The procedures involved in the evaluation of race specific and race non-specific resistance reflect their differences. Quantification of race non-specific resistance is complicated by its expression in features that require detailed microscopic examination, or that can only be determined by multiple monitoring. Race specific resistance, by contrast, can usually be determined on one occasion by assessing visually the infection type response of plants to specific pathogen races. Typically, such determinations are made on seedling plants and, although changes may occur as the ontogenetic age of plants increases, such variations are comparatively rare. It is this kind of resistance for which there are the most data and upon which we shall concentrate here.

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The genetic structure of individual host populations

The extent of variation for disease resistance that can be detected in wild populations is influenced by several factors (Burdon 1987*a*). However, because resistance genes are only expressed phenotypically when challenged by avirulent pathogen isolates, no factor is more important than the number and pathogenicity of the races involved in the assessment. Studies involving only one or two isolates of the pathogen, or those of extreme pathogenicity, are unlikely to provide as detailed a picture of the resistance structure of the population as are studies involving many races of varying levels of pathogenicity. However, the former examples are not only commoner (Dinoor 1970; Zimmer & Rehder 1976) (see figure 1) but also still serve a useful purpose. Indeed, the results of one such restricted study involving the response of a population of *Trifolium repens* (white clover) to single isolates of two foliar pathogens led to the suggestion that the frequency distribution of infection types in a population may reflect the relative importance of particular pathogens as selective forces acting upon the host population in the recent past (Burdon 1980) (see figure 1).

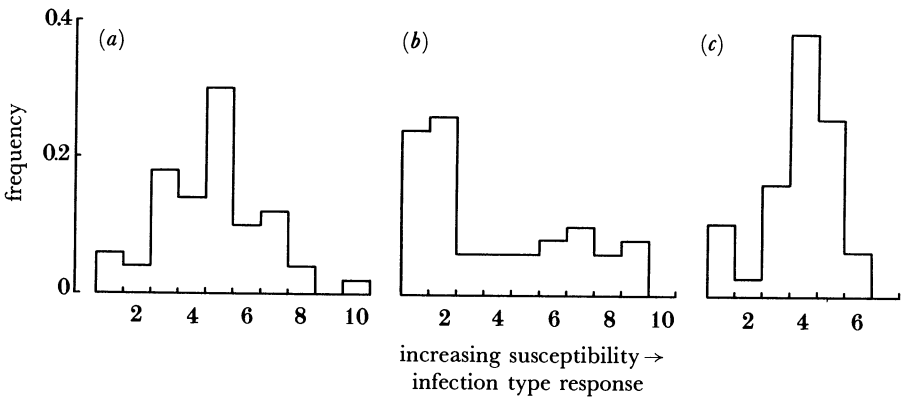

FIGURE 1. Frequency distributions for infection type responses to the pathogens: (*a*) *Cymadothea trifolii*; (*b*) *Pseudopeziza trifolii* in a population of *Trifolium repens* (Burdon 1980); (*c*) *Puccinia coronata* in a population of *Avena fatua* (Burdon *et al.* 1983). For details of infection type categories see original publications.

Recent criticisms of this hypothesis (Dinoor & Eshed 1984, 1987) have confused the frequency distribution of disease incidence with that of infection type responses. Moreover, as recently pointed out (Burdon 1987*a*) the basic relation between selective pressure and frequency distribution is likely to be modified by the history of pathogen attack, the genetic basis of the resistance involved, the genetic load associated with resistance and the nature of the host breeding system. The importance of this last factor in shaping host–pathogen associations seems to have been underestimated.

The full complexity of the pattern of resistance and susceptibility that may occur in plant populations is illustrated by two examples involving the response of closely related species of *Glycine* to nine races of the rust pathogen *Phakopsora pachyrhizi*. The disease resistance structures revealed by these analyses were radically different. In one population of *G. canescens* eleven different phenotypic resistance patterns were observed (Burdon 1987*b*). No individuals were susceptible to all the pathogen races and half the population was resistant to all the isolates. Subsequent genetic analysis of these lines indicated the presence of one, two or three resistance

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genes in each line. It was estimated that twelve resistance factors occurred in that population. In contrast, the resistance structure of the only known population of *G. argyrea* was very different (A. M. Jarosz & J. J. Burdon, unpublished data). Thirty of the 100 plants tested were susceptible to all nine races of *P. pachyrhizi*. Moreover, only four patterns of resistance and susceptibility were detected in the population. One of these patterns was represented by only a single individual. Overall there were only two resistance genes in the whole population, and no individual carried more than one resistance gene.

These studies of resistance in wild *Glycine* populations have concentrated on race specific resistance. In part this certainly reflects the relative ease with which such resistance is detected (Day *et al.* 1983). However, the high frequency of single resistance genes with major phenotypic effects in *G. canescens* and *G. argyrea* (Burdon 1987*b*, 1988; A. M. Jarosz & J. J. Burdon, unpublished data) is a clear indication that race specific resistance is a common feature of at least some wild plant–pathogen interactions. It is probable that many other similar examples will accumulate as detailed analyses are made of other wild systems.

We have not attempted to measure temporal changes in the frequency of resistance in these populations. However, a major problem that arises in the interpretation of such changes in populations of inbreeding species such as *G. canescens* is in determining whether they truly reflect selection for resistance *per se* or selection for other genomic features. Perhaps the most obvious example of this phenomenon is found in the changes that have occurred in Australian populations of the Mediterranean weed, *Chondrilla juncea*. This herbaceous species is obligately agamospermous and three distinct forms occur in Australia. In 1971 a single strain of the rust pathogen *Puccinia chondrillina* capable of attacking one form of *C. juncea* (morphologically distinguishable by its narrow leaves) was released and rapidly exerted a major effect on the fitness of this form. The other two forms of the host were uninfected and have since spread rapidly. Here selection for disease resistance in the entire *C. juncea* population has also produced widespread changes in the frequency of a range of unrelated electrophoretic and morphological markers (Burdon *et al.* 1981).

So far the studies we have considered have examined random samples of individual host populations. These provide an overall picture of the resistance structure but do not allow investigation of the spatial distribution of particular host phenotypes within a population. Data we are currently collecting in the *Linum marginale* – *Melampsora lini* host–pathogen association are beginning to enable us to overcome this problem.

At Kiandra in the Snowy Mountains *L. marginale* is distributed across a hillside in a continuous, although variable, density population extending over a hectare. Two parallel transect lines were laid out diagonally across the site and individual *Linum* plants were sampled at 3 m intervals along each line. If no plant was found within 1 m of each point, no sample was taken. A further sample of 23 plants was taken in a permanent plot established within the same population but approximately 25 m east of the transects. These plants were derived from seed collected from a random sample of the plants involved in the data given in table 1.

The resistance or susceptibility of all these plants to two distinct races (N and AF) of *Melampsora lini* collected from the same site has been determined (figure 2). Although particular host plants showed distinct differential reactions to these two races (all four possible combinations of resistance and susceptibility being detected), over the population as a whole the frequency of resistant individuals was very similar (29 % and 32 %, respectively). However, on the permanent plot there was a significantly higher proportion of individuals susceptible to

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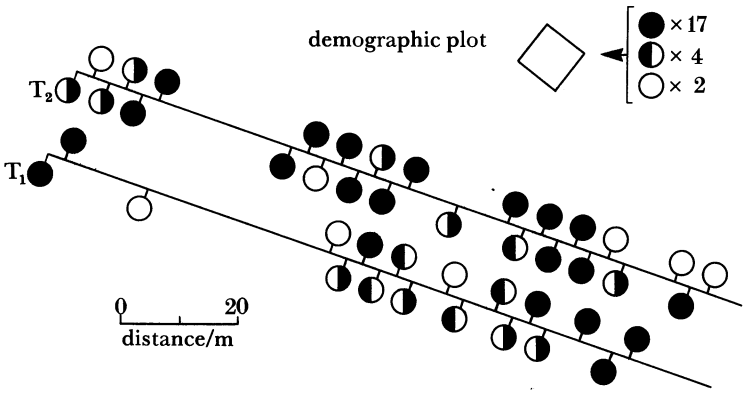

FIGURE 2. Spatial distribution of resistance in a *Linum marginale* population growing at Kiandra, New South Wales to races N and AF of *Melampsora lini*. ●, Plant susceptible to both races; ○, plant resistant to both races; ◑, plant susceptible to race AF and resistant to race N; ◐, plant resistant to race AF and susceptible to race N; T_1 and T_2, transects 1 and 2 respectively (A. M. Jarosz & J. J. Burdon, unpublished data).

race N than was found along the entire transect. The distribution of resistance to race AF between these two areas showed no significant difference, although there was again a greater frequency of hosts susceptible to this pathogen race on the permanent plot. Along the transect lines, nearest neighbour analyses failed to detect any significant departure from a random dispersion of resistant and susceptible individuals.

More closely spaced transect samples (1 m intervals) were taken for several *Hordeum spontaneum* (wild barley) populations which were then tested against a mixed culture of a mildew *Erysiphe graminis hordei* (Wahl *et al.* 1978; Segal *et al.* 1980). Our analysis of the infection type data for three of these populations indicated a random pattern for two populations (R.S. 205 (Wahl *et al.* 1978) and R.S. 207 (Segal *et al.* 1980)) whereas in the third (R.S. 217 (Wahl *et al.* 1978)) both susceptible (type 3) and highly resistant (type 1) plants were clumped.

Overall, these results suggest that pathogens select against large clumps of similar individuals as clumping would be expected in highly self-fertilized species such as *Linum marginale* and *Hordeum spontaneum*. However, a more definitive conclusion on this subject must be deferred until the hypothesis is tested.

Host and pathogen variability between adjacent populations

Marked differences in the susceptibility of closely adjacent populations often occur even within areas where particular pathogens are generally present and gross physical environmental conditions are reasonably conducive to their increase and spread (Burdon 1987*a*). A population at one site may be fully susceptible whereas others nearby may be entirely resistant or be composed of a mixture of resistant and susceptible individuals.

There are many possible causes for this variability, but three warrant special consideration. The first, and one which is frequently overlooked, is the extremely small spatial scale at which the physical environment may change sufficiently to affect materially the incidence and severity of disease and hence its potency as a selective agent. Such variation may be associated with obvious topographic features, with more subtle qualities such as changes in soil nutrients (Snaydon & Davies 1972), or even with the amount of shading by tree canopies. In the latter

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case we recently demonstrated very marked differences in the incidence and severity of disease caused by *Rhynchosporium secalis* (leaf scald) on *Hordeum leporinum* (barley grass) plants growing in full sunlight and those growing under tree canopies only a metre or so away (Jarosz & Burdon 1988). The extreme sensitivity of this host–pathogen interaction was demonstrated clearly by the tendency for disease levels to decline more rapidly on the sunny side of trees than on the shaded side.

The initial establishment of host populations from very limited initial seed inputs may also be a cause of variability in the disease resistance structure of adjacent populations. Small populations established by a few fully susceptible individuals are most likely to remain susceptible or may even become extinct (if selection pressures are sufficiently great), unless further gene flow introduces alleles for resistance. Variability between adjacent populations due to founder effects is likely to be particularly important in close inbreeders or agamosperms where the chances of recombination with other individuals are low or non-existent.

A third, and as yet totally unexplored, potential source of variability in the resistance or susceptibility spectrum of adjacent populations is that caused by local differences in the virulence and length of association of pathogen populations with those of their host. The extent of the complications that may arise can be seen in comparisons of the virulence structure of *Melampsora lini* populations with the resistance structure of their associated host (*Linum marginale*) populations occurring at the Kiandra site (considered earlier) and at two adjacent sites, P_1 (only 300 m away) and P_2 (2.7 km away).

At all three sites the pathogen populations were variable (table 2). The site at Kiandra has been studied most intensively and is dominated by race A. Twelve other races have been found there but only two constitute more than 5% of the population. The pathogen population at P_2 had several races in common with that at Kiandra including the same two dominant ones (A and N), although the frequency of these was very different. In contrast the pathogen population at P_1 was remarkably different despite its much closer proximity to Kiandra; it had only one race in common with the population at Kiandra and two with that at P_2. The commonest race at P_1 (44%) was not present at either Kiandra or P_2, while the dominant race at Kiandra (A) was not present at P_1.

TABLE 2. FREQUENCY OF MAJOR RACES OF *MELAMPSORA LINI* OCCURRING IN THREE POPULATIONS OF *LINUM MARGINALE* IN THE SNOWY MOUNTAINS OF NEW SOUTH WALES

(J. J. Burdon & A. M. Jarosz, unpublished data.)

pathogen race	host site: Kiandra	P_1	P_2
A	0.725	–	0.278
E	–	0.444	–
H	0.666	–	–
K	–	0.167	0.167
N	0.077	0.167	0.278
U	–	0.167	–
all others	0.132 (13)[a]	0.055 (1)[a]	0.275 (5)[a]
no. rust isolates	98	18	19
distance from Kiandra/m	–	300	2700

[a]Number of additional races.

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Some of these differences can be at least partly explained by the resistance structure of the three host populations (table 3). The *Linum marginale* population at Kiandra is almost totally resistant to race E, which is the dominant race at P_1, whereas the host population there is fully susceptible. The two races held in common by sites P_1 and P_2 (races K and N) were both virulent on 90% or more of those host populations. Their lower frequency at Kiandra may well reflect the higher frequency of resistance (30%) at that site. The greatest anomaly in this comparison is caused by race A to which all three host populations are totally susceptible. However, although this race is present at high frequency at Kiandra it is totally absent at P_1 just 300 m away!

TABLE 3. THE FREQUENCY OF INDIVIDUAL PLANTS IN THREE POPULATIONS OF *LINUM MARGINALE* THAT ARE RESISTANT (R) OR SUSCEPTIBLE (S) TO PARTICULAR RACES OF *MELAMPSORA LINI*
(J. J. Burdon & A. M. Jarosz, unpublished data.)

	frequency of infection type response					
pathogen	Kiandra		P_1		P_2	
race	R	S	R	S	R	S
A	0.00	1.00	0.00	1.00	0.00	1.00
E	0.99	0.01	0.00	1.00	0.00	1.00
N	0.29	0.71	0.05	0.95	0.10	0.90
AF	0.32	0.68	0.15	0.85	0.20	0.80

At present such results are posing as many, if not more, questions than they answer. To date we have been able to look at the resistance structure of host populations and the virulence structure of pathogen populations only on single occasions. Inevitably, many of the questions that arise have a major temporal component because host–pathogen interactions are likely to be dynamic associations where the size and genetic constitution of host and pathogen populations fluctuate through time.

How rapidly do host populations respond to pathogen pressure? Just what relation does the resistance structure of the present host population have to the past virulence structure of the pathogen population and vice versa? Can resistance or virulence that is present but no longer effective be viewed as some kind of reflection of previous selection? What does variability in the virulence structure of the pathogen population represent? This may be explained if no one race is capable of overcoming all resistance in the population. However, when a local race is highly virulent why do less virulent forms survive? Finally, do host and pathogen populations interact and evolve on the same spatial scale or does that of the pathogen far exceed the spatial scale of host populations?

Empirically based answers to many of these questions are not likely in the short term and we must turn to theoretical models for assistance.

MODELS OF HOST–PATHOGEN INTERACTIONS

In contrast to animal–parasite population models, which have treated ecological and life history parameters in a sophisticated manner (see, for example, Anderson & May 1981; May & Anderson 1983), those investigating plants and their pathogens are much more limited. Indeed, nearly all existing models have ignored ecological aspects of the interaction and have concentrated exclusively on genetic aspects of the plant–pathogen association. These models

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involve the interactions that occur at a single diallelic locus in both host and pathogen and assume that infection, occurring at a fixed probability for susceptible hosts, leads to reproduction for the pathogen and death for the host. In this extremely simplified state the frequency of the virulence gene in the pathogen population rapidly approaches unity; a balanced polymorphism is not sustained between resistance and susceptibility in the host population, or virulence and avirulence in the pathogen population (Jayakar 1970; Leonard 1977).

This result runs counter to various heuristic arguments (Person 1966; Burdon 1987*a*); to the general recognition that highly virulent races of pathogens rarely dominate their populations even under agricultural conditions; and to the observed pattern of distribution of resistance and susceptibility on a regional scale (Leonard 1984). It is only by introducing fitness penalties associated with resistance in the host and virulence in the pathogen that a polymorphic balance is attained (Jayakar 1970; Leonard 1977).

These models have been valuable in focusing attention on the question of how a polymorphic balance for resistance and susceptibility may be maintained, but their basis in genetic theory is unfettered by ecological or epidemiological realism! The underlying assumptions, of host and pathogen populations of infinite size and of random mixing of both populations at each generation, make it very difficult to interpret these models with respect to reality. There host and pathogen populations are usually small and fragmented and, particularly in the case of pathogens, fluctuate greatly in size from season to season. The magnitude of the fitness penalties associated with resistance and virulence in natural systems remains unknown.

The consequences of divorcing the genetics of host–pathogen systems from their ecological context are illustrated by a model developed for a more limited purpose, that of investigating changes in pathogen populations in agricultural mixtures of cereal varieties. This model examined changes in the efficiency of spore dispersal and its effect on the structure of the pathogen population (Barrett 1980). By partitioning the pathogen spores produced each generation into those that reinfect the same plant (autoinfection) and those that are distributed at random across the whole host population, Barrett found that the relative success of simple and complex pathogen races was radically altered by changes in the proportion of autoinfection. When this was high, relatively avirulent pathogen races predominated. By contrast, when most spores were distributed at random, complexly virulent races dominated the pathogen population.

In contrast to the need to view all genetic interactions in their ecological context, models developed from an ecological standpoint can provide insights into the numerical dynamics of host and pathogen populations without incorporating a genetic component. Indeed by doing so, and by investigating the effects of factors such as population size and degree of fragmentation on the basic interaction, this approach develops the ideal basis on which to build genetic aspects of such associations.

The only such model that we know of is currently under development and investigates the temporal and spatial effects of a pathogenic association between a generalized smut fungus and its grass host (G. C. Kirby, personal communication). It does not incorporate the possibility of variation either for resistance or susceptibility in the host, or for virulence or avirulence in the pathogen. In its basic form this model simulates the dynamics of a single host–pathogen system founded on the same basic premises as the earlier genetic models, i.e. an infinitely large host population with random mixing of host and pathogen in each generation. This framework is

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modified sequentially by restrictions on the size of the host population (hence implying an increasing importance of stochastic processes) and by assuming limited dispersal until, in its most complex form, the model simulates the dynamics of both host and pathogen population when they are limited in size and subdivided into a series of discrete colonies.

In its simplest state the equilibrium properties of Kirby's grass–smut interaction model depend upon the reproductive rate of host and pathogen. Four zones of interaction can be identified. These are: where neither host nor pathogen have sufficient fecundity to persist; where only the host is present; where host and pathogen reach a stable equilibrium point; and finally where host and pathogen show regular cyclical changes in abundance. The restriction of host populations to a finite size has a marked effect on these predictions, as combinations that were previously at a steady equilibrium show cyclical fluctuations. The amplitude of these fluctuations becomes more pronounced as the host population declines in size (figure 3). Indeed, in small host populations the frequency of the pathogen may become so low that random extinction occurs, a phenomenon not encountered in very large populations.

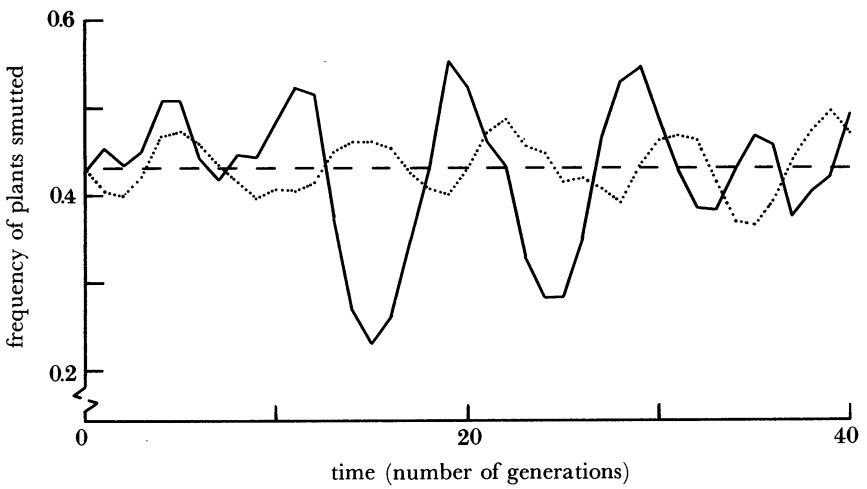

FIGURE 3. Variations in smut abundance in successive generations in an infinitely large and two finite host populations (500 (unbroken line) and 2000 (dotted line) individuals) as predicted by a model developed by G. C. Kirby (unpublished).

The elimination of the random mixing assumption by the introduction of limited dispersal again effects the dynamics of the host–pathogen system. The reproductive rates of both host and pathogen must be higher than those in the random dispersal model before a pathogen population can become established. Moreover, the total size of the pathogen population remains at about half that occurring when dispersal is random.

When this model is used to investigate the effects of subdividing the host population into a number of discrete patches, the dynamics of the overall host and pathogen populations is greatly affected by the amount of dispersal between patches. The behaviour of host and pathogen in each patch is like that of a small version of the random dispersal model. However, because the entire host or pathogen population may include many patches and each patch cycles almost independently they may no longer follow a regular cycle but show only long-term changes in abundance.

Kirby's model requires considerable development. Even at an ecological level it does not

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account for competition between healthy and infected individuals. Moreover, it does not address problems that arise when considering pathogens that reduce host fitness, often at different stages of the host's life cycle, rather than those that prevent reproduction totally. Nevertheless, the firm ecological and epidemiological basis of the present model stresses the importance of some of these ecological factors, particularly population structure and dispersal, to the dynamics of the host–pathogen interaction. It also provides a strong biological base upon which a thorough analysis of genetic interactions between host and pathogen can be developed.

These complex models will give a more realistic view of host–pathogen co-evolution than has previously been available. They will allow us to determine which ecological factors have the greatest effect on host and pathogen populations and to make predictions about the long-term co-evolutionary dynamics of these associations, which is impossible from field studies alone.

Most of the elements needed to develop such a model are now available. Fitness costs associated with pathogen attack; competitive effects between infected and healthy individuals; the types and genetic control of resistance and their distribution in natural plant populations; fluctuations in the size of pathogen populations and their virulence structure: for all of these variables some information exists. Quantification of the size of the fitness costs associated with resistance and virulence remains a challenge for empirical assessment.

We are extremely grateful to Dr G. C. Kirby for making his host–pathogen model freely available and to Dr A. H. D. Brown, Dr R. H. Groves and Dr G. C. Kirby for comments on the manuscript.

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Discussion

B. D. Harrison, F.R.S. (*Scottish Crop Research Institute, Dundee, U.K.*). Can Dr Burdon give any estimate of the relative importance in populations of wild plants of resistance to pathogens controlled by major genes, such as those he has described, and resistance controlled by multiple genes? Is there any evidence that the relative importance of these two kinds of resistance differs between annual and perennial wild plants?

J. J. Burdon. It has often been suggested that one of the main reasons for the apparently limited nature of disease epidemics in wild plant populations is that they are mainly shielded by resistance based on many genes that together give protection against all races of a pathogen. Indeed, it has even been argued that major gene resistance is virtually an artifact of agriculture. We now know that these are rather biased views of the resistance structure of wild plant populations. Not only has the formal basis of resistance been clearly assigned to major genes in wild *Glycine* species (challenged with *Phakopsora pachyrhizi*) but also in *Senecio vulgaris* (challenged with *Erysiphe fischeri*) and *Amphicarpaea bracteata* (challenged with *Synchytrium decipiens*). However, assigning an actual percentage value to such major resistance against that provided by multiple genes remains an extremely difficult task. Clearly, it is likely to vary

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markedly between different species, and even between different populations of a single species. Thus in the two *Glycine* species discussed here, major gene resistance is a very important feature of the *G. canescens* population whereas it is a more minor one of the *G. argyrea* population. In fact, in the latter population we are now following up apparent differences in the length of the latent period. It might well be that the relatively low incidence of major gene resistance in that population is offset by a high incidence of resistance controlled by many more minor genes.

The question of whether there is a difference between annual and perennial plants in the relative importance of these two kinds of resistance has yet to be resolved. This is particularly so if we are to consider the entire age spectrum presented by annual and perennial plants. Certainly, major genes for resistance are present in both annuals and herbaceous perennials, but in woody perennials such as forest trees one might suspect that resistance controlled by multiple genes may well be the most common. It is also quite probable that such patterns will be confounded by various features of the biology of particular host–pathogen associations.

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