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Frequency-Dependent Selection in Plant-Fungal Interactions [and Discussion]

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Frequency-dependent selection in plant–fungal interactions

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A simple genetic model based on Haldane's arguments about the evolution of host–parasite interaction is described and its applicability and limitations with respect to plant–fungus interactions is discussed. The basic form of frequency-dependent selection envisaged by Haldane can be demonstrated in agricultural systems, but no data are available from natural systems.

Introduction

The rapid changes in the genetic constitution of populations of several economically important diseases of some of the world's major crops must rank among the more spectacular examples of natural selection in action. That different individuals or varieties of crop plants vary in their ability to resist infection by pathogenic organisms was recognized during the 19th century, even before the biological basis of plant pathogenesis had become firmly established (see Barrett 1981). The main impetus for breeding for disease resistance came soon after the 'rediscovery' of Mendel's work, with Biffen's demonstration that disease resistance in plants could be inherited in a simple Mendelian manner (Biffen 1907). The complementary phenomenon, that strains of pathogenic organisms varied in their specificity for different host genotypes and that the specificity was inherited stably, was established during the second decade of this century but it was not until the late 1920s and early 1930s that the genetic basis of this specificity began to be unravelled (Day 1974). The final step of simultaneously investigating the genetics of pathogenicity and resistance in a single host–parasite combination was finally done by H. H. Flor in the flax–flax-rust system (Flor 1942). From a series of crosses between host lines, which showed distinct resistance or susceptible reactions to a set of different strains of the fungus and between the fungal strains themselves, he was able to conclude that 'the range of pathogenicity of a physiologic race (strain) is determined by pathogenic factors specific for each resistance factor possessed by the host' (Flor 1942).

The period between 1915 and 1940 witnessed several disastrous crop failures in the wheat growing areas of North America, characterized by the widespread cultivation of new varieties that had quite suddenly succumbed to novel strains of the stem rust fungus, *Puccinia graminis*, which were able to overcome the resistance of these varieties. The same period also witnessed the decline of the banana growing industry based on the cultivar 'Gros Michel' in the West Indies and Central America because of the extreme susceptibility of this variety to the wilt fungus *Fusarium oxysporum* f.sp. *cubense*. These two examples of natural selection in action appear to have provided Haldane with part of the evidence that he drew upon in his 1949 paper to support his argument that diversity in host populations might be generated in response to infection by parasitic or pathogenic organisms: 'The most that the average species can achieve is to dodge its minute enemies by constantly producing new genotypes, as the agronomists are constantly producing new rust-resistant wheat varieties.'

Not only did he see that such a process was evolutionarily possible, but also that it would be strongly frequency dependent.

'Probably a very small biochemical change will give a host species a substantial degree of resistance to a highly adapted microorganism. This has an important evolutionary effect. It means that it is an advantage to the individual to possess a rare biochemical phenotype. For just because of its rarity, it will be resistant to the diseases which attack the majority of its fellows. And it means that it is an advantage to a species to be biochemically diverse... (which) will contain at least some members capable of resisting any particular pestilence.'

By the time of his 1954 essay 'The statics of evolution', Haldane had realized that what was sauce for the host was also sauce for the pathogen. Again using stem rust as his example: 'In a natural population of hosts and pathogens composed of many genotypes there will be selection for pathogens adapted to common biochemical types and against those only adapted to rare ones. The selection among the host plants will be in the opposite direction. Thus both host and pathogen will constantly alter their prevailing genotype, in so far as it affects the host–parasite relation. This process will favour a diversity of types in the host, and probably in the pathogen also.'

Despite quoting examples from plant pathology in developing the ideas outlined in both of these papers, Haldane gives no references to any papers in plant pathology. More surprising still is his omission of any reference to Flor's investigations but two factors suggest that he was unaware of this work:

(1) The complementarity of the genetic systems demonstrated in the flax–flax-rust system would have provided strong evidence for the type of evolutionary process for which he was arguing. So there is no reason for him to have left it out.

(2) With the evidence before him of a real case of genetic complementarity, it is hard to believe that he could have resisted the temptation to produce a mathematical model rather than somewhat vague verbal arguments.

A simple genetic model

Given that there is genetic evidence of the type of genetic complementarity envisaged by Haldane a simple model based on his arguments can be built without having to invent the gene-for-gene relation (Flor 1956) to produce the model. The simplest model consists of two different phenotypes, R and r, in the host population and two different phenotypes V and v in the pathogen population. R is more resistant than r to phenotype v in the pathogen population but r is more resistant than R to infection by pathogen phenotype V. Conversely, v is less virulent than V on host genotype R but v is more virulent than V on host phenotype r.

The fitnesses of the host and parasite with respect to each other can be written as shown below.

host fitnesses

		host	
		R	r
pathogen	v	1	$1-s$
	V	$1-t$	1

parasite fitnesses

		host	
		R	r
pathogen	v	$1-a$	1
	V	1	$1-b$

Without too much loss of rigour these phenotypes can be treated as genotypes, i.e. assume that the organisms are haploid, clonally reproducing or predominantly self-fertilizing. This

may not be too far from the truth for many plant–fungus interactions. It then follows that the frequencies of the phenotypes in the next generation can be written as follows.

The same symbols for phenotypes and frequencies are used:

$$\left.\begin{aligned} R' &= R(1-Vt)/(1-RVt-rvs), \\ r' &= r(1-vs)/(1-RVt-rvs), \\ V' &= V(1-rb)/(1-Rva-rVb) \\ \text{and} \quad v' &= v(1-Ra)/(1-Rva-rVb). \end{aligned}\right\} \tag{1}$$

From this it is easy to show that $\Delta R = 0$ when $\hat{V} = s/(s+t)$ and $\Delta V = 0$ when $\hat{R} = b/(a+b)$. Below $\hat{V}$, R will tend to increase and, conversely, decrease when V is above $\hat{V}$. On the other hand, if R is below $\hat{R}$, V will tend to decrease and only increase if R is above $\hat{R}$. These conditions can be combined on a phase diagram (figure 1).

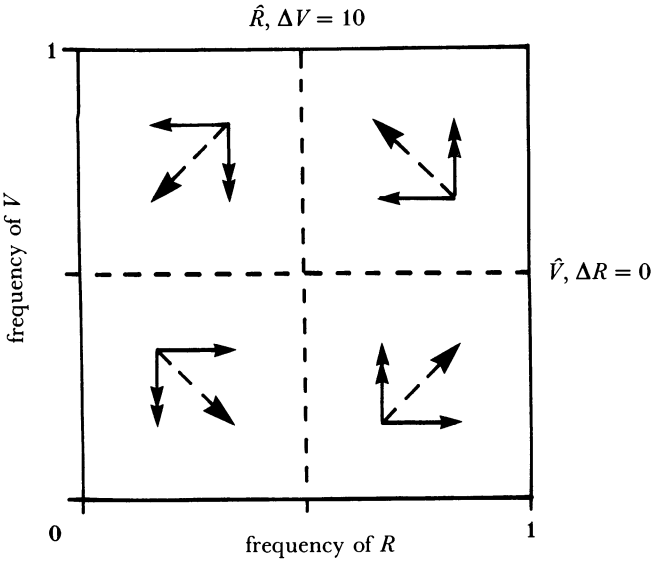

FIGURE 1. Phase diagram of the changes in frequency of the R phenotype in a host and V phenotype in a pathogen or parasite. ⟶, Direction of change of R. ⟶⟶, Direction of change of V. ⟶, Resultant. (For symbols, see text.) (Note that the position of the boundaries between the quadrants are dependent on the values of s and t, and a and b, but for clarity the quadrants have been drawn with equal areas.)

There is an important point to note here. Such a system of interaction does not show frequency-dependent selection in the conventional sense of the term (see also May & Anderson 1983). The recurrence equations have no fitness expressions in terms of a phenotype's own frequency but the fitnesses of the parasite depend on the frequencies of the different host phenotypes and the fitnesses of the host phenotypes depend on the frequencies of the pathogen phenotypes. In no case is the increase of any phenotype dependent on a fitness which is a function of its own frequency. Thus Haldane's assertion that novel resistant genotype will be at an advantage because of their rarity is seen to be false, but his error is easy to see. If a population of hosts consists only of the r-type and the pathogen population only of the v-type, then it can be seen that R-types will tend to increase in the host population because they are fitter in the presence of v than the r-types. Thus for the situation envisaged by Haldane, the apparent advantage of rarity is clear. However, in the top left-hand corner of the phase

diagram V is common in the pathogen population, and R will still decline despite its rarity. Genetic models of this type show a close affinity to the Lotka–Volterra equations describing the population dynamics of host–parasite or predator–prey systems, with gene or phenotype frequencies substituted for population numbers. It is also worth noting that this simple genetic model assumes that either all of the host individuals are infected or that a constant proportion of the host population is infected irrespective of phenotype or frequency. Similarly, all pathogen phenotypes are assumed to have an equal probability of successfully infecting a host.

The effects of density

For many, if not all, pathogenic organisms, the probability of successful infection of a host individual is dependent on the density of the host population. Consequently there must be a threshold density below which a pathogen population cannot maintain itself because either contacts between infected and non-infected hosts are too infrequent or infected individuals die or become immune too quickly for effective transmission (see, for example, Black 1966). However, the importance of this density effect will vary according to the epidemiology of each pathogen and host combination. If a purely density-dependent component specific for each combination of phenotypes is added to the simple genetic model above, then the fitnesses will become genuinely frequency-dependent, because rarer phenotypes will also be present at lower densities (for discussion of this point see May & Anderson (1983)). It may be a general phenomenon that when genetic models are built with ecological assumptions, density-dependent ecological variables will translate into frequency-dependent genetic variables (for examples see Barrett (1976) and Levin, this symposium).

However, for plant host–pathogen systems such effects are likely to be weaker than in animal systems, not least because plants lack an immune response system. The importance of the relative density of host phenotypes on the development of an epidemic is strongest when incompatible interactions are lethal for the pathogen and compatible interactions are lethal for the host. From the perspective of the pathogen, a host that acquires immunity is equivalent to that host individual dying because it is no longer available for infection. On the other hand, if compatible and incompatible interactions are merely differences of degree, i.e. resistance or susceptibility and virulence or avirulence are relative rather than absolute measures, then the effects of the relative density of each phenotype on epidemic development is weakened. Although it must be true that at the limit there must be an effect of density in all host–pathogen systems, it is probably less strong in plant systems, particularly for aerially dispersed pathogens, crop plants and locally abundant plant species. Indeed, when doing laboratory experiments with plant pathogens, prevention of contamination from air spora is more of a problem than containment of the experimental material, particularly so for agriculturally important pathogens cf. pathogens of animals. So for an examination of the dynamics of the interactions between plants and their fungal pathogens, a simple genetic model may be less of an over-simplification than for animal systems.

Dynamic behaviour of the model, 1

From the phase diagram it can be seen that the resultant trajectories in each of the quadrants predict cyclical behaviour, and that this could give rise to a series of limit cycles with neutral stability, thus leading to the coexistence of polymorphism in host and parasite; such results

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have been obtained from several models based on variants of Haldane's arguments (see, for example, Jayakar (1970), Yu (1972), Clarke (1976) and Leonard (1977), the last of these being exclusively built on assumptions from plant pathology (see also Leonard & Czochor 1980)).

Evidence from the field

Despite May's stricture that anyone who needs data to convince themselves of the validity of a model shows a remarkable lack of self-confidence, it is always useful to see whether real biological systems behave in anything like the same way as the models predict. The first problem in looking for suitable data to test this type of model is that unless the selection coefficients are fairly large, the duration of the average research project is just not long enough to detect such changes in natural ecosystems. The second problem is that until recently nobody looked at the dynamics of the interaction in natural plant–fungus interactions. At best, the studies were mere catalogues of variation in host and parasite populations.

By the 1960s the widespread cultivation of resistant varieties of crop plants, the evolution of pathogen strains adapted to these resistant varieties ('resistance breakdown') and the subsequent decline in popularity of these varieties had become so commonplace that it had acquired a name: 'the boom-and-bust cycle' (Suneson 1960). By the end of the decade a new generation of plant pathologists had realized that the problems of 'resistance breakdown' and the 'boom-and-bust cycle' were problems in population genetics and that merely cataloguing the occurrence of different 'races' of pathogens was not satisfactory. Surveys now began to move towards to collecting data on the frequencies of different 'races' or phenotypes within pathogen populations. The cultivation of barley in the U.K. since 1960 has shown a classic 'boom-and-bust' cycle. New, resistant, varieties have only had an effective life of 3–5 years before the evolution of the powdery mildew fungus, *Erysiphe graminis*, has rendered the resistance ineffective. If the area planted with varieties carrying the resistance gene *Mla12* and the prevalence of the corresponding pathogenicity in the powdery mildew populations is plotted on a suitable pair of axes to correspond to relative frequencies, cyclical behaviour is obtained (figure 2). These data are very useful because they show not one but two overlapping cycles when the resistance gene was recycled in combination with other resistance genes as breeders increased the number of resistance genes in their later varieties (Wolfe 1984).

To some extent this is not a true analogue of genetic feedback in that what is driving the changes in the host frequencies is not natural selection but the growers' perceived economic advantages and disadvantages of the varieties carrying this resistance gene. However, because this is in part a reflection of the loss in yield due to 'resistance breakdown', it reflects the lost reproductive output of the affected plants, although the magnitude of the changes in the host population from year to year are far larger than natural selection would produce, e.g. a farmer losing 15% yield on one variety would not grow it in the following year, thereby rendering a 15% reduction in reproductive output effectively lethal.

However, these data do demonstrate that the advantage of resistance within the context that we have defined is strongly dependent on the composition of the pathogen population and that given Haldane's frame of reference it could be seen as frequency dependent. That the only data available for illustrating this idea come from agriculture, with all of the possible artefacts that this could generate, only serves to highlight the lack of data from natural systems.

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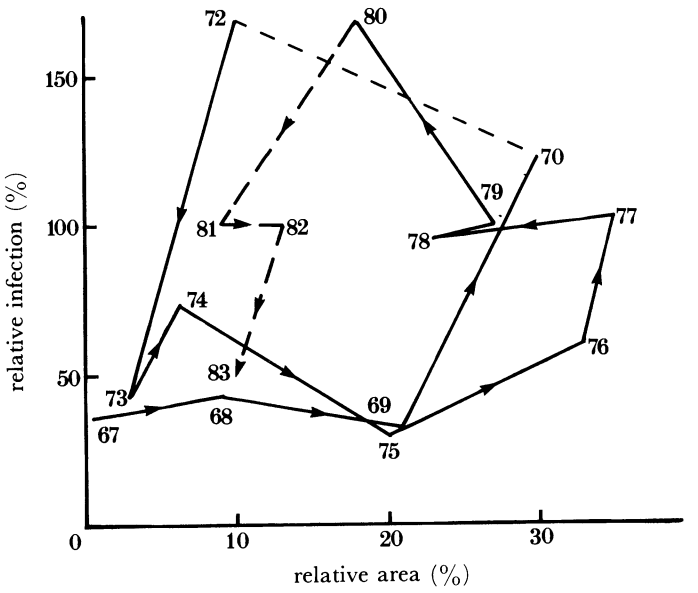

FIGURE 2. Relative infection of barley varieties carrying the *Mla12* mildew resistance gene plotted against the relative area planted with these varieties between 1967 and 1983 (the point for 1971 is missing). Between 1973 and 1975 varieties carrying *Mla12* resistance alone were supplanted by varieties carrying *Mla12* with other resistance genes. (Data mostly from the Agricultural Development and Advisory Service (1981) and M. S. Wolfe, personal communication.)

Although there have been several studies of variation in natural plant host–pathogen systems, particularly in systems related to crop species (see Barrett (1985) for examples; Parker (1985)), no study has yet followed genetic changes over a long period of time.

DYNAMIC BEHAVIOUR OF THE MODEL, 2

The simple model that is described above is essentially symmetrical because it more closely resembles the sort of idea that Haldane was attempting to promote. However, this is not necessarily realistic. For many fungal diseases, particularly those of cultivated plants, the fitness arrays are far from symmetrical. This slight change of emphasis can be illustrated as follows:

host fitnesses

pathogen	host: R	host: r
v	1	$1-s$
V	$1-t'$	$1-t$

parasite fitnesses

pathogen	host: R	host: r
v	$1-a$	1
V	1	$1-b$

In general, $t' \leqslant t \leqslant s$ and $b \ll a$.

The critical point to note here is that the V-type able to attack the R phenotype of the host can attack the r, or susceptible, phenotype, almost equally as well and that the r phenotype is almost equally as susceptible to the V-type as it is to the v-type. That is, although there is a large difference in fitness between the two host genotypes when the pathogen population consists only of the v-type, both host genotypes have almost the same susceptibility to V. Conversely, although there is a large fitness difference between the two pathogen genotypes on the

R-type, there are only small fitness differences on the susceptible phenotype. Although such asymmetries in the fitnesses may have no effect on the overall oscillatory behaviour of the system, the rate at which the system passes through the four quadrants of the phase diagram are quite different. Starting from a similar situation to that envisaged by Haldane, i.e. a susceptible population of hosts in which a novel resistant genotype has just arisen and an essentially monomorphic pathogen population, the resistant phenotype will begin to spread (figure 3). Once $\hat{R}$ is exceeded, the pathogen population responds so the process is essentially as before. When the system enters the upper right quadrant, the *R*-type will start to decline, but the fitness differences are small so that the rate of decline is slow, because the fitness differences between the two host genotypes is small. *V*, however, continues to increase to the point where it is effectively fixed in the population. However, given sufficient time, *V* eventually begins to decline once $\hat{R}$ has been passed. There is, therefore, a long period in which *R* slowly declines, and *V* remains high. By comparison, the time taken for the substitution of *V* by *v* and *v* by *V* is relatively short. With this lengthy period when nothing much appears to be happening and *V* is very close to fixation, *v* may be lost by stochastic variation. Even if this does not happen, there is ample opportunity for a new resistance mutant to arise so that instead of the constant cycling of the system, the host population could move into another resistance substitution. In this case, what is superficially a cycling system may move into a cascade process of successive resistance substitutions followed by the response of the pathogen; in other words, the cycling behaviour could degenerate into an 'arms race'.

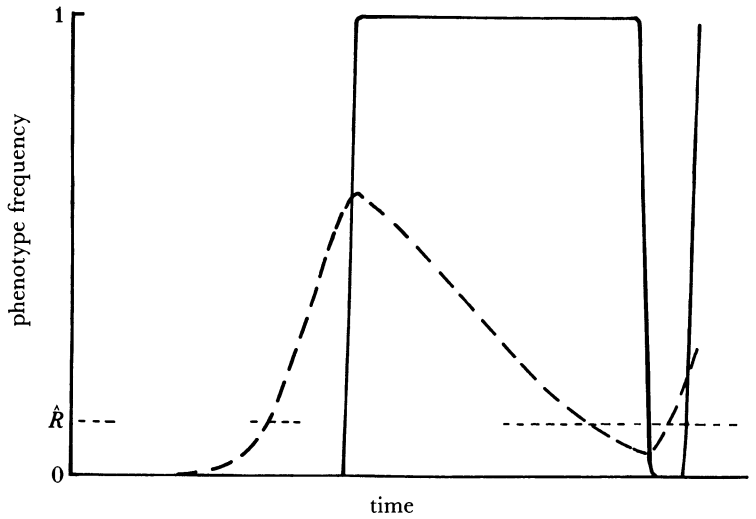

FIGURE 3. An example of changes in the frequency of the *R* phenotype (– – –) in the host and the *V* phenotype in the parasite (——) over time when the fitness matrix is aymmetrical and both phenotypes are initially rare. For symbols see text. (S. P. H. Johnson & J. A. Barrett, unpublished data.)

A further interesting phenomenon follows from examination of model: because the fitness differences between the host phenotypes tend to be smaller than those between the pathogen genotypes, any oscillations generated in the host phenotype frequencies will have a proportionately larger effect on the pathogen phenotypes. This can easily be demonstrated by an example in which the host frequencies start at values close to the point at which their 'frequency-dependent' fitnesses cancel out and similarly for the pathogen frequencies (figure 4). The host populations show oscillations of small amplitude and the pathogen populations

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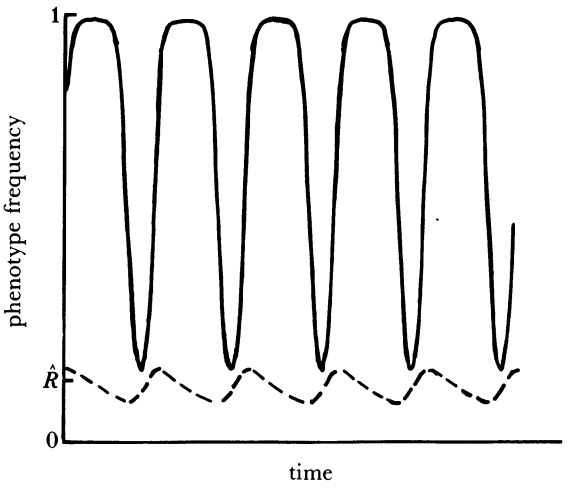

FIGURE 4. An example of the changes in the frequency of the R phenotype (– – –) in the host and the V phenotype in the parasite (——) over time when both phenotypes start near their 'equilibrium' and the fitness matrix is asymmetrical. For symbols see text. (S. P. H. Johnson & J. A. Barrett, unpublished data.)

show oscillations of a considerably larger amplitude. In this case the oscillations in the host phenotypes could be within sampling error of most survey methods. Alternatively, the pathogen oscillations would be easily detectable. So if this type of behaviour were observed in a natural system it would probably be interpreted as showing an apparently stable polymorphism in the host, and large but unaccountable fluctuations in the composition of the pathogen population.

GENETICS OF THE INTERACTION

Not much attention has been paid to the explicit genetics of the system so far. This is because whether the host and pathogen species are haploid, diploid or even polyploid, and which characters are dominant or recessive, makes little difference to the general form of the evolutionary dynamics. All of the models reduce to essentially a one-locus–two-allele system in host and parasite, so long as overdominance or co-dominance is not introduced. Similarly, for 'single-locus' models, it makes little difference whether the species are sexual or asexual (inbreeding); only the rates are changed. However, more realistic models must deal with more than two loci and once the models move beyond single loci, all of the complexities of the genetic and breeding systems must be introduced (see Seger, this symposium).

However, the genetics of fungal host–parasite systems are often not as simple as the one-locus–two-allele basic model. Examples can be found with simple resistance or susceptibility and virulence or avirulence dimorphism at single loci in both host and parasite respectively but, in the more extensively worked systems, effectively multiallelic systems for resistance can be found. These are matched not by multi-allelic systems in the parasite but by independently inherited virulence characters, e.g. the *Mla* locus in barley. The consequence of this on the dynamics of the systems is that what are mutually exclusive phenotypes in the host can be overcome by single genotypes in the parasite. In a dynamic system, therefore, polymorphism in the host could give rise to monomorphism in the parasite.

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Epidemiology

Perhaps the severest criticism of the basically genetic models of this kind is that they take no account of the ecology and epidemiology of the interactions. It is implicit in this model that each host is only infected by one pathogen genotype at a time. This may be a reasonable assumption for some seed-borne pathogens but is certainly not reasonable for aerially dispersed pathogens which may go through several asexual generations for each host generation. To the best of my knowledge no-one has yet produced a comprehensive model that combines the evolutionary dynamics of a plant host–pathogen system with epidemiologically realistic assumptions. However, some indication of the possible complexity that might arise can be drawn from attempts to investigate some components of the interaction. For example, there has been much discussion about the advantages or disadvantages of growing heterogenous mixtures of crop plants to control disease. Barrett (1978, 1980) has examined the effects that such mixtures could have on the evolution of a parasite. In this case the proportions of different genotypes in the host population are fixed and the changes in the pathogen population followed. The models attempted to incorporate elements of the epidemiology of foliar pathogens with several to many parasite generations per host generation.

In one model (Barrett 1980) there are three resistant host genotypes and an array of the eight possible pathogen genotypes that can attack respectively none, or one, or combinations of two or all three, of the host genotypes. The model was based on the cultivated barley–powdery mildew interaction in which the pathogen must match each of the resistance genes carried by a host plant in order for infection to be successful. If a pathogen 'race' carries any virulence genes not required for successful infection, its fitness is reduced. Therefore maximum fitness in the pathogen is where each pathogen genotype exactly matches the genotype of the host line. To add reality, a proportion of the spores is dispersed between host plants in each generation. If there is no plant-to-plant transmission in each generation (i.e. all spores or propagules fall back onto the plants on which they were produced, or none are transferred to neighbouring plants), then one of the pathogen genotypes able to attack all of the host genotypes can show a net increase at the beginning of the cycle because on average it is fitter than those pathogen genotypes specialized on just one host, even though it is less fit than the more specialized types on any individual host genotype. As the season progresses, the more specialized genotypes replace it. On the other hand, if plant-to-plant transmission is high, then the flexibility of the multiply virulent form offsets any disadvantage that it may have on any particular host and it becomes the predominant genotype over the season. It is important to note that both of these simulations are genetically identical, and that the only difference is that population dynamics have been changed slightly, but the effect on the evolutionary outcome is dramatic. With little plant-to-plant transmission a polymorphism persists in the pathogen, but with higher levels of plant-to-plant transmission, the pathogen population becomes monomorphic.

Although this is a very simple model of just part of a host–parasite system, it shows that the interaction between just two epidemiologically based parameters, i.e. duration of the epidemic and the amount of plant to plant transmission, can determine whether the pathogen population ultimately consists of specialists or generalists on the different host genotypes. (For an example of the dynamics of multiple infection, see Bremermann & Pickering (1983) and for a more extensive general discussion of the effects of combining epidemiology and genetics into evolutionary models, see May & Anderson (1983)).

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Epilogue

The past few years have witnessed an awakening of interest in the population biology of systems in which two or more species interact. From both a practical and academic perspective, plant host–fungal pathogen interactions offer a fascinating insight into how species interact. Haldane's assertion that host–parasite interactions are frequency-dependent is likely to be his lasting endowment to studies in this field and despite the passage of nearly 40 years, the last paragraph of his 1949 paper is just as apposite today.

> In this brief communication I have no more than attempted to suggest some lines of thought. Many or all of them may prove to be sterile. Few of them can be followed profitably except on the basis of much field work.

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Discussion

J. Antonovics (*Botany Department, Duke University, U.S.A.*). Although it may be true that plant–pathogen interactions are not truly frequency dependent (i.e. depend on interspecific frequency dependence) when both plant and pathogen have equivalent generation times, this is surely not the case when the generation time of the pathogen is much shorter than that of the host? In that case, build up of the pathogen on abundant host genotypes will generate true frequency-dependent fitnesses when the latter are measured over the lifetime of the host. A model demonstrating this has been presented by May & Anderson (1983).

J. A. Barrett. As I understand the models of May & Anderson, hosts are either infected or not infected; they did not consider the case of hosts having different levels of infection. From epidemiological considerations, they showed that there is a 'threshold' density of each host phenotype below which the matching pathogen phenotype cannot maintain itself and that the host fitnesses are dependent on the ratio between the actual number of each host phenotype and its threshold density. For the special case where the total host-population size is constant, i.e. the pathogen is not regulating the population size of the host, the threshold density translates into a 'threshold frequency', and the host fitnesses become frequency-dependent. However, if the net reproductive value, R_0 of the pathogen is sufficiently high, as it may be for locally abundant hosts and pathogens with high reproductive output, the threshold density becomes zero and all potential hosts can be infected in each host generation; in this case the host fitnesses become independent of density or frequency. Although the type of frequency-dependent fitnesses obtained by May & Anderson are derived for a polycyclic pathogen, '...the infectious diseases spread through each generation of hosts in an epidemic fashion...', the critical factor is the proportion of each host phenotype that is ultimately infected in each generation. A monocyclic pathogen, would produce similar dynamics, but in this case the epidemic would be measured over host generations. Under the assumptions of May & Anderson, if a polycyclic pathogen is capable of infecting all potential hosts in the population, frequency-dependent selection, *sensu stricto*, will not arise; whether fitnesses are truly frequency dependent or not depends critically on the biology of the pathogen and the ecology of the host.

My own work on polycyclic pathogens in genetically heterogeneous host populations certainly suggests that if plant-to-plant transmission is high, so that disease build-up on individual plants is primarily due to propagules from other plants (allo-infection), the frequency-dependent effect that you describe is observed. On the other hand, if disease build-up on individual plants arises primarily from self-infection (auto-infection), then the host fitnesses are independent of frequency. Again, polycyclic pathogens do not necessarily give rise to true frequency dependence; the critical factors are the biology and ecology of the host–pathogen system.

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