Spitze, K. 1991. *Chaoborus* predation and life-history evolution in *Daphnia pulex*: Temporal pattern of population diversity, fitness, and mean life history. *Evolution* 45: 82–92.

Spitze, K., J. Burnson, and M. Lynch. 1991. The covariance structure of life-history characters in *Daphnia pulex*. *Evolution* 45: 1081–1090.

Taylor, D., and P.D.N. Hebert. 1992. *Daphnia galeata mendotae* as a cryptic species complex with interspecific hybrids. *Limnol. Oceanogr.* 37: 658–665.

Weider, L. J. 1985. Spatial and temporal genetic heterogeneity in a natural *Daphnia* population. *J. Plankton Res.* 7: 101–123.

Weir, B. S. 1990. *Genetic Data Analysis*. Sinauer, Inc. Sunderland, Mass.

Wolf, H. G. 1987. Interspecific hybridization between *Daphnia hyalina*, *D. galeata*, and *D. cucullata* and seasonal abundances of these species and their hybrids. *Hydrobiol.* 145: 213–217.

Wright, S. 1948. On the roles of directed and random changes in gene frequency in the genetics of populations. *Evolution* 2: 279–294.

Wright, S. 1951. The genetic structure of populations. *Annals of Eugenics* 15: 323–354.

Young, J.P.W. 1979a. Enzyme polymorphism and cyclic parthenogenesis in *Daphnia magna*. I. Selection and clonal diversity. *Genetics* 92: 953–970.

Young, J.P.W. 1979b. Enzyme polymorphism and cyclic parthenogenesis in *Daphnia magna*. I. Heterosis following sexual reproduction. *Genetics* 92: 971–982.

7

The Interplay of Numerical and Gene-Frequency Dynamics in Host-Pathogen Systems

Introduction

We know very little about how pathogens affect natural plant populations, but we have a vast body of literature on how pathogens affect crop plants. The neglect of pathogens by plant ecologists is curious, but it is consistent with the historical trend where, in the past, primary attention was given to examining the role of abiotic factors in plant distribution. Only within the past two decades has explicit focus been placed on biotic factors such as plant-plant competition and plant-insect interactions. The current surge of interest in plant-pathogen interactions (Burdon 1987; Fritz and Simms 1992) is therefore an extension of the realization that biotic forces, while perhaps of secondary consideration with regard to explaining differences among the major biomes and vegetation types, may be of primary importance in determining species abundance and diversity within those biomes. Unfortunately, the knowledge acquired in an agricultural setting has been gained in the context of a research tradition based on the applied goals of crop production and cannot be easily extrapolated to provide an understanding of natural systems. For example, agriculture has been concerned with disease control, with its emphasis on disease symptoms rather than on host and pathogen fitnesses; it has focused on situations where crops are planted at set densities and where the major dynamic processes are the epidemic spread of pathogens through preexisting monospecific, genetically uniform stands; and because agriculture is concerned with crop yield, a group property, there has been naive group-selection thinking about evolutionary outcomes (see Antonovics and Alexander 1989, for a discussion of these issues). In addition, at least in the United States, the lack of involvement of agricultural funding agencies (e.g., United States Department of Agriculture) in basic research combined with the concentration of plant pathologists in agricultural departments has severely aggravated the disjunction between the pure and applied aspects of the subject.

The applied study of plant pathogens has nevertheless provided two obvious but major generalizations that can serve as a starting point for

extrapolations to natural systems. The first general observation is that disease can have an enormous impact on the reproduction and survival of individual plants. The second general observation is that individual genotypes of both host and pathogen may differ greatly in resistance and virulence (Day 1974; Vanderplank 1984; Wolfe and Caten 1987). Given these two observations, we can expect host-pathogen dynamics to be driven by the interaction of strong ecological and genetic forces. We can expect dramatic impacts on survival and reproduction, unforgiving selection pressures, and consequently large changes in population size and gene frequency. In this and the following chapter, I explore these expectations further.

Any host-pathogen system can vary in many ways: the disease may or may not cause changes in fecundity or survival, individuals may or may not recover from the disease, and there may or may not be induced resistance (i.e., the equivalent of an "immune class"). Because the possibilities are many, I focus here on models based on a real-world example that I have been studying, namely the anther-smut disease of white campion, *Silene alba* (= *S. latifolia*) caused by the fungus *Ustilago violacea*. I describe the biology of this system in more detail in the next chapter, but the salient features are (1) the disease sterilizes the host because both male and female plants develop anthers bearing spores and the ovary aborts in female flowers, (2) the disease is systemic such that the host usually does not recover, and (3) transmission of the disease is by pollinators. Because pollinators adjust their flight distances to compensate for plant density, it is likely that the probability of a healthy individual becoming diseased is a function of the frequency (and not absolute density) of diseased individuals in the population. In experimental arrays of diseased and healthy plants, spore deposition on healthy plants increased with increasing frequency of diseased plants but not with increasing population density (Antonovics and Alexander 1992). Some spore dispersal also occurs by spores falling from the parent plant, and seedlings placed in close vicinity to the parent can become infected (Alexander 1990). However, there is no evidence that the disease is transmitted through the seeds, even when those seeds are produced from partially diseased plants (Baker 1947). The pathogen may decrease plant longevity to some degree (Alexander and Antonovics 1988; Alexander 1989; Thrall, pers. comm.), but plants with the disease appear more or less normal vegetatively. The host has been shown to have genetic variation in resistance and susceptibility, whereas the fungus appears to be quite uniform genetically (Alexander, Antonovics, and Kelly 1993). There also appears to be some resistance cost; male plants that are more resistant have fewer flowers and flower later in the season (Alexander 1989).

Fortunately, these aspects of the biology of the *Silene-Ustilago* system can be encapsulated into fairly simple but general models. Thus throughout we will assume that there is no reproduction of diseased hosts, that hosts do not recover from the disease, and that there is no disease-induced mortality or immunity. With regard to the genetics, we will assume that the host is genetically variable for resistance but the pathogen is genetically uniform. We will also confine our attention to single isolated populations, realizing (as outlined in the following chapter) that this may inadequately describe the disease dynamics on a more regional basis.

Frequency, Density, and the Transmission Process

Population geneticists have traditionally focused on the dynamics of gene frequencies, whereas population ecologists have focused on the dynamics of numerical change. The numerical dynamics of the *Silene-Ustilago* system can be represented by simple difference equations:

$$X_{t+1} = X_t (1 + b - d) - X_t P_t \tag{7.1a}$$

$$Y_{t+1} = Y_t (1 - d) + X_t P_t \tag{7.1b}$$

where X,Y = number of healthy and diseased individuals

b = birth rate of healthy individuals

d = death rate of diseased and healthy individuals

P = probability a healthy individual becomes diseased

t = time interval.

For simplicity, we assume here that birth, death, and infection processes occur simultaneously, but we could easily modify the equations to represent situations where, for example, plants that become diseased early in the time interval do not subsequently reproduce.

The transmission parameter, P, can take two forms depending on whether one views disease transmission as a function of the absolute density of diseased plants,

$$P_t = \beta Y_t, \tag{7.2a}$$

or the frequency of diseased plants,

$$P_t = \beta Y_t / N_t. \tag{7.2b}$$

The density-dependent case would represent aerial transmission of spores where the per capita likelihood of a healthy individual becoming diseased increases as the density of diseased individuals increases. The frequency-dependent case would represent pollinator transmission of spores, where the pollinator increases its flight distance to compensate for increases in plant spacing: this is the predominant mode of transmission of anther-smut in *Silene*. This transmission mode is likely to be a general character-

istic of vector-borne and venereal diseases. A number of previous studies have shown that the transmission mode can have a large effect on the population dynamics (Getz and Pickering 1983; Thrall, Antonovics, and Hall 1992).

Extension of these cases to include genetic variation in host and pathogen is straightforward. This can be best illustrated by assuming the host is haploid and has two alleles at one locus:

$$X1_{t+1} = X1_t(1 + r1 - P1_t) \tag{7.3a}$$
$$X2_{t+1} = X2_t(1 + r2 - P2_t) \tag{7.3b}$$
$$Y1_{t+1} = Y1_t(1 - d) + X1_t P1_t \tag{7.3c}$$
$$Y2_{t+1} = Y2_t(1 - d) + X2_t P2_t, \tag{7.3d}$$

where $r = b - d$, and labels 1 and 2 refer to two different haploid genotypes of the host.

We can easily reformulate these equations in terms of frequency and not absolute number of the different types (setting $x, y = X/N, Y/N$):

$$Tx1_{t+1} = x1_t(1 + r1 - \beta 1\, y_t) \tag{7.4a}$$
$$Tx2_{t+1} = x2_t(1 + r2 - \beta 2\, y_t) \tag{7.4b}$$
$$Ty1_{t+1} = y1_t((1 - d) + \beta 1\, x1_t) \tag{7.4c}$$
$$Ty2_{t+1} = x2_t((1 - d) + \beta 2\, x2_t), \tag{7.4d}$$

where $T = 1 + r1\ x1_t + r2\ x2_t - d(y1_t + y2_t)$.

At least superficially, it may seem that by restricting ourselves to only gene-frequency dynamics, we are only "standardizing" frequencies by total population size, and therefore simplifying the models. However, it can be seen (eqs. 7.4a–d) that the distinction between the density- and frequency-dependent transmission modes is lost. Indeed, models that restrict themselves to consideration of only gene-frequency dynamics implicitly assume that the transmission mode is frequency-dependent. Such models can no longer directly address the dynamics of systems with density-dependent transmission. In using models based on gene frequency, we are also making implicit assumptions about population size and/or its regulation. Formally, these assumptions are either that the population size is constant (it is set to unity at each generation), or that it is changing in an unregulated manner (there is no density-dependent population regulation), or that population regulation acts proportionately on the healthy and diseased classes. An example of the latter may be where density dependence acts equally on, say, overwinter mortality (time interval $t + 1, t + \frac{1}{2}$) of both diseased and healthy individuals, but the remaining dynamics is determined in the summer period (time interval $t + \frac{1}{2}, t$) by

processes described using equations of the form (7.1) to (7.4) above. Therefore, models that do not incorporate numerical dynamics assume that density-dependent regulation is either not occurring or is proportionate among the types, and that the disease transmission process is frequency-dependent.

Population Regulation and Coexistence

It is commonly appreciated that the stability and dynamics of host-pathogen and predator-prey systems can be greatly affected by the presence of extrinsic factors limiting host and pathogen abundance (Hassell 1978; Anderson and May 1978). As mentioned above, for these extrinsic factors to have an impact, they must influence the rate of change of healthy and diseased individuals differentially. This can come about in two ways. First, the density-dependent processes may act equally on the birth and death rates of healthy and diseased individuals, but they may not affect the disease transmission dynamics. This introduces a nonproportionality in the rate of growth of the healthy and diseased classes. Second, the degree to which density alters the birth and death rates may differ among healthy and diseased individuals. Most generally we might expect diseased individuals to be more susceptible to the impact of density. A number of studies have shown that diseased individuals are competitively inferior to healthy individuals (Reestman 1946, cited in De Wit 1960; Burdon and Chilvers 1977) but I know of no explicit studies comparing the impact of density on diseased and healthy individuals. A number of other examples are known where the presence of a disease protects the host against predators and/or improves their competitive abilities (Clay 1991). If there is age or stage specificity in the probability of becoming diseased, but the impact of density occurs at an earlier or different stage, then healthy and diseased individuals may also have different density-dependent responses. This is likely to be the case in the *Silene-Ustilago* system as well as in other venereal disease systems where disease transmission is in the adult reproductive phase, but where density has the greatest impact on the juvenile phase.

The importance of differential density dependence is well illustrated in models of the type we have outlined above (eqs. 7.1, 7.2). Without differential density dependence, and given the assumptions of no host recovery and no reproduction of diseased individuals, density-dependent disease transmission predicts neutral stable limit cycles. With linear density dependence acting on reproductive rates or death rates, there is an oscillatory approach to equilibrium (Antonovics 1992). Because the fungus sterilizes the host and converts it morphologically entirely for its own

reproduction, it is in a sense a predator or parasitoid, and the models developed above are similar in form to those developed for predator-prey or host-parasitoid systems (Hassell 1978). In frequency-dependent transmission systems (e.g., venereal diseases), equilibrium coexistence of host and pathogen is not possible unless one invokes some external density-dependent factor limiting host and pathogen mortality or reproduction (Getz and Pickering 1983). However, if there is no host recovery and no reproduction of diseased individuals, as we assume in our *Silene-Ustilago* system, then host-pathogen coexistence further requires that the degree of density dependence be different in the healthy and diseased class (Thrall, Antonovics, and Hall 1992). In the case of sterilizing venereal diseases such as the anther-smut of *Silene*, unequal density-dependent effects greatly expand the range of parameters (disease transmission rate, death rate, and reproductive rate) over which host-pathogen coexistence is possible (Thrall, Biere, and Uyenoyama 1992; Thrall, Antonovics, and Hall 1992). In the models that follow, I include differential density-dependent regulation by assuming that it acts on the growth rate of the healthy class (e.g., via its effects on juvenile recruitment, or adult fecundity), but has no effect on the diseased class (death rates of diseased and healthy individuals are density independent).

Joint Numerical and Gene Frequency Dynamics

It is possible to develop theoretical models of host-pathogen systems that incorporate numerical and gene-frequency dynamics to differing degrees. Of particular interest are three special cases representing variation in gene frequencies only (no numerical dynamics), variation in numbers only (no gene frequency dynamics), and variation in both frequencies and numbers of diseased and healthy individuals/genotypes. For each, there may be both gene-frequency equilibrium and numerical equilibrium, there may be one or the other, or there may be neither. I also focus on particular equilibria, and ask whether they are approached in different ways given different assumptions about the genetic and numerical dynamics.

Unregulated vs. Regulated Populations

We begin by comparing the gene-frequency dynamics in a population that is unregulated (or where healthy and diseased individuals are proportionately regulated) with dynamics in a population where there is differential response of the two types of individuals to density. To again reflect the situation in the *Silene-Ustilago* system, we will consider the case

where reproduction of (or recruitment into) the healthy class is a function of plant density, whereas the diseased class is unaffected by density. Throughout we assume that populations follow the "reciprocal yield law" characteristic of many plant populations (Harper 1977), and which reflects what is often termed pure "contest" competition in animal populations (Hassell 1975). This assumes that at very high densities the number of individuals emerging in the next generation asymptotes to some level, rather than decreases. We include population regulation by setting birth rate,

$$b = b_0/(b_1 N_t + 1), \tag{7.5}$$

such that the carrying capacity,

$$K = (b_0 - d)/b_1 d, \tag{7.6}$$

where b_0 = birth rate at low density

b_1 = a constant determining the intensity of density dependence

N_t = total population size ($= X_t + Y_t$)

d = death rate.

It can be shown that without some form of density dependence a stable genetic polymorphism is not possible. Typically the resistance gene either does not spread, or allele frequencies show cycles of ever-increasing amplitude. It can be seen from eqs. (7.3a–d) that the fitnesses of the resistant and susceptible genotypes do not depend on their relative frequencies, but only on the frequency of diseased individuals.

An example of an unregulated population is shown in figure 7.1a: here gene-frequency dynamics shows ever-increasing oscillations, leading eventually to extinction of one or the other genotype. However, when population regulation is included in the model but otherwise the parameters are unchanged, stable gene frequency (fig. 7.1b) and numerical dynamics are possible (fig. 7.1c).

Genetically Uniform vs. Genetically Variable Populations

At the simplest level, the importance of genetic variation in a host-pathogen system is obvious. Thus if a population is initially composed of only very susceptible individuals, we predict the certain and rapid demise of the whole population. However, the subsequent spread of a few resistant individuals can save such a population from extinction. As in all host-pathogen systems, it is obvious that in the presence of the disease, and with no fitness cost to resistance, the resistant genotype will spread in the population. However, if there is a cost to the resistance, then there is the

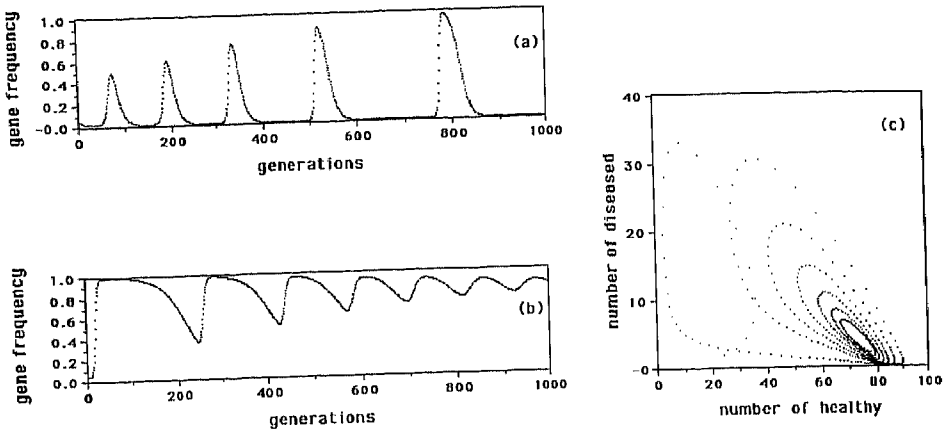

FIG. 7.1. (a) Gene frequency dynamics of a host-pathogen system assuming no population regulation. Graph shows change in frequency of the resistant allele over generations. Model is based on eqs. (7.4a–c) (see text) with parameter values: $b1$, $b2$ = 0.8, 0.64; d = 0.2; β1, β2 = 0.9, 0.1. (b) Gene frequency dynamics when there is population regulation by density dependence acting on the healthy class. Model is based on eqs. (7.3) and (7.5) (see text) with parameter values as above, except that in addition $K1$, $K2$ = 100, 80. (c) Numerical dynamics for case (b), shown as a phase plane plot. Here and in all subsequent figures, starting numbers (or proportions) were 18 susceptible, 1 resistant, and 1 diseased.

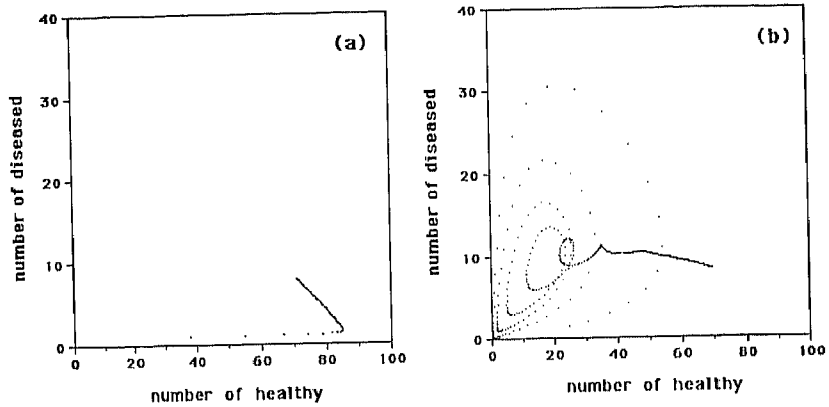

FIG. 7.2. Numerical dynamics of two host-pathogen systems that have similar numerical equilibria, but where (a) the host population is genetically uniform, and (b) the host population is genetically variable. Model is based on eqs. (7.3) and (7.5) (see text) with parameter values: (a) b = 4.4; d = 0.45; β = 0.5; K = 88.6. (b) $b1$, $b2$ = 0.9, 0.6; d = 0.4; β1, β2 = 0.9, 0.35; $K1$, $K2$ = 120, 105.

possibility that the population may maintain a genetic polymorphism for resistance. Perhaps less obvious is the fact that even though the numerical equilibrium of a genetically variable host-pathogen system may be identical to one that is genetically invariant, the approach to equilibrium may be quite different. This is illustrated in figure 7.2, where it can be seen that in the case of a genetically uniform population, approach to equilibrium involves an initial overshoot on numbers of the healthy, but then a relatively direct and rapid convergence on the equilibrium (fig. 7.2a); in the genetically variable population, approach to numerical equilibrium is oscillatory and takes an extremely long time (fig. 7.2b). It is particularly noteworthy that in the genetically variable case, identical points in the phase plane can have quite different trajectories (also in fig. 7.1c). The reason is that the phase plane, as depicted, represents numerical composition and not genetic composition: thus populations represented by singular points on this plane can have different genetic compositions and can therefore change in different directions depending on this composition. To fully represent the dynamics, the phase plane should be in three dimensions, with gene frequency as a third axis: the dynamics shown in figure 7.2b would then be a path that takes the shape of an ever-narrowing helix. Any phase-plane analysis of a host-pathogen system will therefore be misleading if there is underlying genetic variation in the responses of the host and/or pathogen.

Demographic Form of the Resistance Cost

To illustrate how the ecological and demographic expression of the cost of resistance can affect the dynamics, we will compare the outcomes of three cases (fig. 7.3a–c). In all three cases the model parameters are identical except in the way the cost is included. Thus in figure 7.3a we assume that the cost affects the reproductive rate of the individuals, but has no effect on the carrying capacity. This might be a situation where the resistance gene affects reproductive output (e.g., more resistant individuals reproduce less), but that population size is a function of adult density and that density acts equally on resistant and susceptible individuals. In this case, a polymorphism cannot be maintained. The reason is that, as the resistance gene spreads, population size increases; at high densities, because resistant and susceptible genotypes have equal carrying capacities the fitness differential between them is reduced, and so resistant individuals further increase in frequency, again increasing population size, and so on. In figure 7.3b we assume that the reproductive rate of resistant and susceptible genotypes is the same, but resistant genotypes are more affected by increasing density. This might be a situation where the resis-

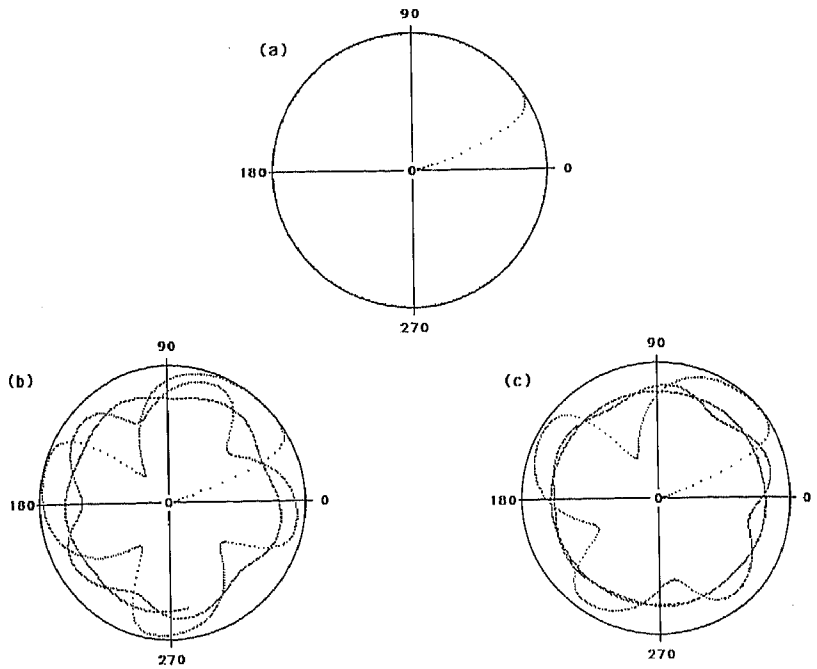

FIG. 7.3. Gene-frequency dynamics of a host-pathogen system assuming different forms of the resistance costs: (a) different reproductive rates, identical carrying capacities; (b) identical reproductive rates, different carrying capacities; (c) different reproductive rates and carrying capacities. Change in frequency of the resistant allele is plotted on polar coordinates, where distance from the center of the circle represents allele frequency (scale 0–1), and where generations are plotted in units of degrees starting at zero and increasing counterclockwise around the circle. Model is based on eqs. (7.3) and (7.5) (see text) with parameter values: for all cases, $d = 0.2$, $\beta 1$, $\beta 2 = 0.9$, 0.1. (a) $b1$, $b2 = 0.8$, 0.48; $K1$, $K2 = 100$. (b) $b1$, $b2 = 0.8$; $K1$, $K2 = 100$, 60. (c) $b1$, $b2 = 0.8$, 0.48; $K1$, $K2 = 100$, 60.

tance gene affects resource-use efficiency, such that resistant individuals require more resources for survival. Here a polymorphism can be maintained. However, under these conditions, imposing a fitness cost in terms of the reproductive rate is not without effect: the dynamics are again different when the cost affects not only the carrying capacity but also the reproductive rate of resistant genotypes (fig. 7.3c).

Another possibility is that the fitness cost is expressed in only one sex. This may well be the case in *Silene*, where resistant males have fewer flowers and flower earlier, but where no such trend is seen in females (Alexander 1989). If this is the case, then a polymorphism is maintained even though the resistant and susceptibles have identical carrying capaci-

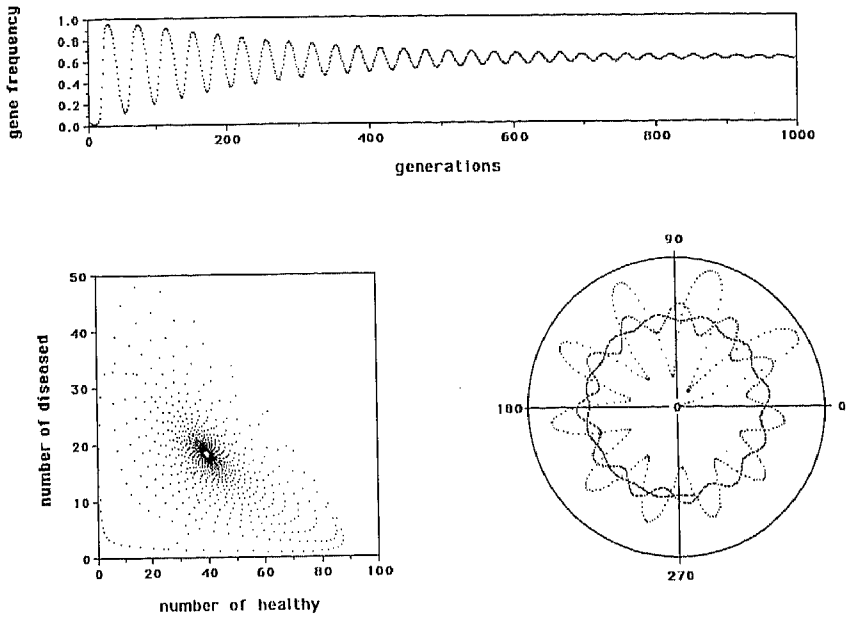

FIG. 7.4. Gene-frequency and numerical dynamics of a host-pathogen system assuming the resistance costs affect only male fecundity and the two resistance types have identical carrying capacities. Upper graph shows the change in frequency of the resistant allele over generations; the circle shows the same data plotted on polar coordinates (see legend to fig. 7.3 for explanation). Numerical dynamics is shown as a phase plane plot at the lower left. Model is based on eqs. (7.3) and (7.5), except that the resistance cost affects male fecundity. Parameter values: $b1$, $b2 = 0.8$; $d = 0.2$; $\beta 1$, $\beta 2 = 0.9$, 0.1; $K1$, $K2 = 100$; $m1$, $m2$ (male fecundities) = 1.0, 0.6.

ties and equal female fecundities (fig. 7.4). The reason is that when a highly fecund (and therefore susceptible) male is rare, it makes a relatively greater genetic contribution to the next generation than when it is common. This introduces an innate frequency dependence such that when susceptible individuals are reduced in frequency by the disease, their genetic contribution increases relative to that of resistant individuals. If there is in addition some cost in terms of overall carrying capacity, still different dynamics are evident, producing in this case stable limit cycles (fig. 7.5).

The interaction of venereal disease spread and male and female fitnesses is likely to be complex and have important repercussions for the evolution of mating systems and sexual selection. For example, low mating success (and greater resistance) of males may be genetically correlated with higher female fitness. Differential allocation to male and female

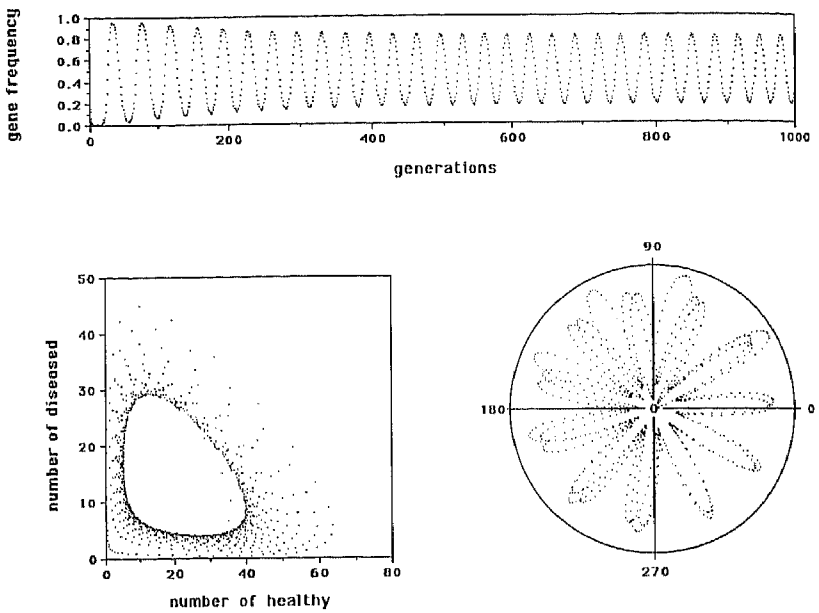

FIG. 7.5. Gene-frequency and numerical dynamics of a host-pathogen system assuming the resistance costs affect both male fecundity and carrying capacities of the two resistance types. For explanation and parameter values, see fig. 7.4, except here $K1$, $K2 = 100, 60$.

functions can itself maintain a genetic polymorphism: but the range of values over which polymorphisms in male and female mating success can be maintained may be much greater in the presence of venereal diseases.

Discussion

My goal in this chapter has been to emphasize the importance of including both genetic variation and numerical dynamics in models of host-pathogen interactions. Not only are expected equilibria and transient dynamics quite different if both components are included, but it becomes clear that purely genetic models based on frequencies of interacting components unwittingly subsume a frequency-dependent transmission mode (Jayakar 1970; Lewis 1981; Seger 1988). These models are therefore only directly applicable to venereal and vector-borne diseases. Several such models were originally developed with crop diseases in mind (Leonard 1977; Barrett 1988), and no doubt the investigators may be quite surprised to realize that they were in fact modeling venereal diseases! A number of authors, including some of the above, have pointed out that host-parasite interactions are likely to be both frequency and density dependent, but as far as I know only one paper has investigated this explicitly (May and Anderson 1983). In the other studies the focus has been on gene frequencies, but the models have been formulated with the additional assumption that disease spread is most likely when the susceptible genotype is most common, thereby generating frequency-dependent selection (Gillespie 1975; Clarke 1976).

The models presented here, while reflecting the essential features of a real host-pathogen system, are essentially heuristic; no attempt has been made to explicitly simulate the *Silene-Ustilago* system. Nor have the dynamical consequences of the models been exhaustively analyzed; the dynamical patterns are shown for illustrative purposes and are specific to the chosen parameter values. Nevertheless, there is no doubt that the overall conclusions regarding the importance of combining genetic and numerical dynamics are quite general. Indeed, the analyses presented here may understate the case because they may be uncharacteristic of other kinds of host-pathogen systems. Thus they assume no genetic variation in the pathogen, and therefore exclude the dynamical behaviors that can result from genetic specificity in host-pathogen interactions: adding pathogen variation can result in a highly complex gene-frequency dynamics (Seger 1988) and can lead to the maintenance of genetic variation in both host and pathogen in the absence of any externally imposed population regulation. The models that have been presented here also exclude the possibility of "within season" population dynamics, resulting because the generation time of the pathogen is much shorter than that of the host: such within-season epidemics can have severe effects on relative fitnesses and on total abundance (Gillespie 1975; May and Anderson 1983). In the *Silene-Ustilago* system, the anther-smut has a long latent period (from three to six weeks or even longer; Alexander, Antonovics and Kelly 1992) and therefore the number of pathogen generations per season is probably quite limited. May and Anderson (1983) modeled a pathogen with a fast life cycle relative to host (such that epidemics occurred on the host each season due to rapid density-dependent disease transmission). They also assumed there was host-pathogen specificity, such that each host-genotype could only be attacked by the corresponding pathogen genotype. This system exhibited very rich dynamics and could lead to frequency-dependent selection sufficiently severe to result in chaotic cycles of numbers and gene frequencies. If the actual abundances and not just the relative frequencies of the different genotypes were affected, gene frequency dynamics was even more likely to be chaotic. Although they did not explicitly investigate the consequences of disease-independent population regulation, they suggested that this may actually dampen any large or

chaotic oscillations and result in more predictable dynamics. Many other host-pathogen systems are therefore likely to demonstrate a complex interplay between numerical dynamics and gene frequency.

Because of the multiplicity of factors that impinge on their dynamics, host-pathogen systems pose a serious challenge to the ecological geneticist. From a purely theoretical perspective, any model almost inevitably has to be an uncomfortable compromise between generality (simplicity and tractability) and realism (specificity). From an empirical perspective, understanding the forces that impinge on the dynamics of host-pathogen systems requires detailed information of the kind that is not easily gleaned by, say, a simple population survey. Were nature so simple and convenient as to be describable by these basic models, estimation of the model parameters would still present formidable problems. For example, in the *Silene-Ustilago* system considered here, parameterization of the models would require knowledge of genetic variation in the recruitment rates, the death rates, and the degree and type of density-dependent population regulation. We would also need to know about genetic variation in the disease transmission coefficient. Even calculating a disease transmission coefficient in the absence of genetic variation is fraught with difficulties: it may appear easy to calculate this from the rate at which healthy individuals become diseased and the frequency of the disease in the previous time interval (year), but there are further hidden assumptions. Most importantly we have to assume that the disease transmission coefficient is constant and does not vary with population composition, or with the frequency and density of diseased individuals. None of these assumptions can be validated by a descriptive study of one population at one time interval. If the population is itself genetically variable for resistance, then any predicted trajectory based on purely ecological data may be very misleading precisely because the genetic composition of the population will change.

There are two general lessons to be learned from consideration of the interplay of numerical and genetic processes in host-pathogen systems. The first is that any pathogen control strategy based on modeling population behavior needs to be based on both genetic and ecological information. The second is that if we are to understand disease in natural populations, then two divergent but complementary strategies are available to us. One strategy is to study a particular system, but this perforce carries the expectation that we will need a wealth of genetic and demographic detail to gain a thorough understanding. The other strategy is to develop generalized expectations about the role of pathogens in maintaining species diversity in communities and genetic diversity within species, and then use comparative and experimental approaches to test these expectations. I have suggested that there is a real place for such a holistic approach to understanding biotic interactions in natural populations, and that this should perhaps be encompassed by a new discipline termed "community genetics" (Antonovics 1992). As we have seen from the examples in this chapter, biotic interactions in communities are likely to be, if not dominated, then at least greatly influenced by genetic factors. Conversely, there is now increasing evidence that pathogens are likely to be important forces in the evolution of mating systems (Levin 1975; Hamilton 1980; Schmitt and Antonovics 1986; Lively 1987; Lively et al. 1990). Because of this close interplay of ecological and genetical processes, it is very likely that features such as the mating system or the recombination frequency, which in the past have been viewed as only affecting long-term evolutionary potential and were of little direct interest to the ecologist, may themselves influence species abundances and distributions, via their effect on disease and pest incidence (Burdon and Marshall 1981). The trend in ecology toward a consideration of biotic rather than abiotic forces in determining species abundances and distributions will increasingly engage the expertise and interest of the ecological geneticist.

Acknowledgments

I wish to thank Peter Thrall for overseeing my algebra, and Helen Alexander for her insights and collaboration which stimulated this study. Support was provided by NSF grant BSR-8717664, a Guggenheim Fellowship, and a Royal Society Visiting Research Fellowship at the Biology Department, Imperial College at Silwood Park, University of London.

References

Alexander, H. M. 1989. An experimental field study of anther-smut disease of *Silene alba* caused by *Ustilago violacea*: Genotypic variation and disease incidence. *Evolution* 43: 835–847.

Alexander, H. M. 1990. Epidemiology of anther-smut infection of *Silene alba* caused by *Ustilago violacea*: Patterns of spore deposition and disease incidence. *J. Ecol.* 78: 166–179.

Alexander, H. M., and J. Antonovics. 1988. Disease spread and population dynamics of anther-smut infection of *Silene alba* caused by the fungus *Ustilago violacea*. *J. Ecol.* 76: 91–104.

Alexander, H. M., J. Antonovics, and A. W. Kelly. 1993. Genotypic variation in plant disease resistance: Physiological resistance in relation to field disease transmission. *J. Ecol.* 81: 325–333.

Anderson, R. M., and R. M. May. 1978. Regulation and stability of host-parasite population interactions. I. Regulatory processes. *J. Animal Ecol.* 47: 219–247.

Antonovics, J. 1992. Towards community genetics. In *Plant Resistance to Herbivores and Pathogens: Ecology, Evolution and Genetics*, ed. R. S. Fritz and E. L. Simms, pp. 426–449. University of Chicago Press, Chicago.

Antonovics, J., and H. M. Alexander. 1989. The concept of fitness in plant-fungal pathogen systems. In *Plant Disease Epidemiology*, ed. K. J. Leonard and W. E. Fry, vol. 2, pp. 185–214. McGraw-Hill, New York.

Antonovics, J., and H. M. Alexander. 1992. Epidemiology of anther-smut infection of *Silene alba* caused by *Ustilago violacea*: Patterns of spore deposition in experimental populations. *Proc. Roy. Soc. London B* 250: 157–163.

Baker, H. G. 1947. Infection of species of *Melandrium* by *Ustilago violacea* (Pers.) Fuckel and the transmission of the resultant disease. *Ann. Botany* 11: 333–348.

Barrett, J. A. 1988. Frequency dependent selection in plant-fungal interactions. *Phil. Trans. Roy. Soc. London B* 319: 473–483.

Burdon, J. J. 1987. *Disease and Plant Population Biology*. Cambridge University Press, Cambridge, U.K.

Burdon, J. J., and G. A. Chilvers. 1977. The effect of barley mildew on barley and wheat competition in mixtures. *Australian J. Botany* 25: 59–65.

Burdon, J. J., and D. R. Marshall. 1981. Biological control and the reproductive mode of weeds. *J. Appl. Ecol.* 18: 649–658.

Clarke, B. 1976. The ecological genetics of host-parasite relationships. In *Genetic Aspects of Host-Parasite Relationships*, ed. A.E.R. Taylor and R. Muller, pp. 87–103. Blackwell, Oxford.

Clay, K. 1991. Parasitic castration of plants by fungi. *Trends in Ecol. and Evol.* 6: 162–166.

Day, P. R. 1974. *Genetics of Host-Parasite Interaction*. Freeman, San Francisco.

De Wit, C. T. 1960. On competition. *Verslagen van landbouwkundige onderzoegen*, no. 66–8. Wageningen.

Fritz, R. S., and E. L. Simms. 1992. *Plant Resistance to Herbivores and Pathogens: Ecology, Evolution and Genetics*. University of Chicago Press, Chicago.

Getz, W. M., and J. Pickering. 1983. Epidemic models: Thresholds and population regulation. *Amer. Nat.* 121: 892–898.

Gillespie, J. H. 1975. Natural selection for resistance to epidemics. *Ecology* 56: 493–495.

Hamilton, W. D. 1980. Sex versus non-sex versus parasite. *Oikos* 35: 282–290.

Harper, J. L. 1977. *Population Biology of Plants*. Academic Press, London.

Hassell, M. P. 1975. Density dependence in single species populations. *J. Animal Ecol.* 44: 283–295.

Hassel, M. P. 1978. *The Dynamics of Arthropod Predator-Prey Systems*. Princeton University Press, Princeton, N.J.

Jayakar, S. D. 1970. A mathematical model for interaction of gene frequencies in a parasite and its host. *Theor. Pop. Biol.* 1: 140–164.

Leonard, K. J. 1977. Selection pressures and plant pathogens. In *The Genetic Basis of Epidemics in Agriculture*, ed. P. R. Day, pp. 207–222. Annals of the New York Academy of Sciences 287.

Levin, D. A. 1975. Pest pressure and recombination systems in plants. *Amer. Nat.* 109: 437–451.

Lewis, J. W. 1981. On the coevolution of pathogen and host. I. General theory of discrete time coevolution. *J. Theoret. Biol.* 93: 927–951.

Lively, C. M. 1987. Evidence from a New Zealand snail for the maintenance of sex by parasitism. *Nature* 328: 519–521.

Lively, C. M., C. Craddock, and R. C. Vrijenhoek. 1990. Red Queen hypothesis supported by parasitism in sexual and clonal fish. *Nature* 344: 864–866.

May, R. M., and R. M. Anderson. 1983. Epidemiology and genetics in the coevolution of parasites and hosts. *Proc. Roy. Soc. London B* 219: 281–313.

Schmitt, J., and J. Antonovics. 1986. Experimental studies of the evolutionary significance of sexual reproduction. IV. Effect of neighbor relatedness and aphid infestation on seedling performance. *Evolution* 40: 830–836.

Seger, J. 1988. Dynamics of some simple host-parasite models with more than two genotypes in each species. *Phil. Trans. Roy. Soc. London B* 319: 541–555.

Thrall, P. H., A. Biere, and M. Uyenoyama. 1992. The population dynamics of host-pathogen systems with frequency dependent disease transmission. *Amer. Nat.* (to be submitted).

Thrall, P. H., J. Antonovics, and D. W. Hall. 1992. Host and pathogen coexistence in vector-borne and venereal diseases characterised by frequency dependent disease transmission. *Amer. Nat.* (in press).

Vanderplank, J. E. 1984. *Disease Resistance in Plants*. Academic Press, London.

Wolfe, M. S., and C. E. Caten. 1987. *Populations of Plant Pathogens: Their Dynamics and Genetics*. Blackwell, Oxford.

ECOLOGICAL GENETICS

Edited by Leslie A. Real

PRINCETON UNIVERSITY PRESS PRINCETON, NEW JERSEY

Published by Princeton University Press, 41 William Street,
Princeton, New Jersey 08540
In the United Kingdom: Princeton University Press,
Chichester, West Sussex

Library of Congress Cataloging-in-Publication Data
Ecological genetics / edited by Leslie A. Real.
p. cm.
Includes bibliographical references (p.) and index.
ISBN 0-691-03241-6 (cl) — ISBN 0-691-00066-2 (pb)
1. Ecological genetics. 2. Evolution (Biology)
3. Population genetics. I. Real, Leslie.
QH456.E26 1993
575.1—dc20 93-4762

This book has been composed in Adobe Sabon

Princeton University Press books are printed
on acid-free paper and meet the guidelines
for permanence and durability of the Committee
on Production Guidelines for Book Longevity
of the Council on Library Resources

Printed in the United States of America

10 9 8 7 6 5 4 3 2 1

(Pbk.)
10 9 8 7 6 5 4 3 2 1

Contents