CHAPTER SEVEN

Genetics and the Spatial Ecology of Species Interactions: The *Silene-Ustilago* System

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INTRODUCTION

To population geneticists, the idea that limited dispersal may influence subsequent evolutionary processes not only has been part of the fabric of the discipline but has also been a driving motivation for a large number of theoretical and empirical studies designed to clarify, elaborate, and illustrate (some may even say, deify) the early, seminal ideas of Sewall Wright (1931, 1940, 1943). He argued that evolution should be more rapid in subdivided populations than in a panmictic population because chance effects in small semi-isolated populations permit characters to move from one adaptive state to another through an intermediate state of lower fitness. This idea has received much attention from evolutionary biologists (Wade 1992; Barton 1992; Whitlock 1995), and there is now a particularly large literature on the influence of limited dispersal ("isolation by distance") on spatial genetic substructuring (Epperson 1993). However, the relationship of much of this work to ideas in ecology has remained tenuous. One of the main reasons is that studies on genetic substructuring have focused primarily on neutral genes. Yet neutral genes are of little direct interest to the ecologist concerned with the causal processes determining numerical abundance. Apart from studies on the interaction of gene flow and selection (Endler 1977), consideration of how spatial sub-

structuring of individuals influences selection response is rare in the population genetics literature. Some time ago, Levin and Kerster (1975) showed that if individuals occupy positions on a spatial array, then the dynamics of selection can be greatly affected by seed- and pollen-dispersal distributions. Yet there have been few subsequent attempts to comprehensively "test" the robustness of population genetic theory in spatially extended populations. That this theory may be drastically changed is now being suggested by ecologically motivated studies. For example, Molofsky et al. (1997, in preparation) and Durrett and Levin (1997) have shown that some forms of positive frequency-dependent selection can maintain polymorphism in stochastic spatially distributed systems. This is not possible in single, unstructured populations. Similarly, the degree of spatial aggregation of individuals can greatly affect the conditions for coexistence under competition (Pacala 1986a; Kreitman, Shorrocks, and Dytham 1992). Therefore the outcomes of even quite simple types of interactions among species (qua genotypes) may be qualitatively different when considered in a spatial context.

It is tempting for an ecologist to argue that "genetics is only in the details" and to subsume genetic variation as just another form of heterogeneity among the many inescapable and therefore perhaps ignorable complications of an already complex discipline. For example, at a recent symposium on disease in natural populations (Grenfell and Dobson 1995), there was a discussion section focused on the issue of "Is genetics just another heterogeneity?" In coevolutionary systems, genetic composition is likely to interact strongly with numerical abundance and vice versa. In host-pathogen systems, for example, the presence of genetic variation can greatly influence numerical dynamics and coexistence (May and Anderson 1983; Antonovics 1994). Conversely, purely genetic models that omit numerical dynamics fail to capture conditions for maintenance of resistance polymorphisms (Antonovics and Thrall 1994) and fail to predict how pathogen virulence might evolve (Lipschitz and Nowak 1994).

In the context of spatially structured populations and metapopulations, there are important reasons why it is productive

to include a genetic perspective. First and foremost, viewing connectedness simply as a property of colonization ignores the connectedness that comes from gene exchange. Gene-flow distances can be very different from colonization distances; this is particularly obvious in plants, where pollen is dispersed quite differently from the seeds. Thus genetic rescue (e.g., input of resistance genes into a diseased population) may be as important as ecological rescue (input of propagules).

Second, colonization events (and extinction events if they are gradual) are almost invariably accompanied by genetic drift ("founder effects"); genetic stochasticity accompanies demographic stochasticity. This may create large differences in the genetic composition of founding populations with these differences declining through time as a result of gene flow among populations (McCauley 1993). In the metapopulation that we have been studying, newly founded populations of the plant *S. alba* are more differentiated with regard to allozyme and chloroplast DNA markers than are long-established populations (McCauley, Raveill, and Antonovics 1995). In host-pathogen systems (and analogous coevolutionary interactions) such chance effects may result in a severe dislocation of any local correspondence between host resistance genes and pathogen virulence genes (Jarosz and Burdon 1991; Frank 1997). The fractionation processes will become even more severe as the numbers of genes and alleles involved in the interactions increase and may lead to locally unstable and unpredictable dynamics ("We are ready to see that host-parasite genetics is like the weather"; Frank 1997).

Third, because colonization and extinction result in increased variation among populations, it becomes important to explore the possibility that group selection may be an effective force. The potential consequences of group selection can be enormous (Gilpin 1975; Boerlijst, Lamers, and Hogeweg 1993; Kelly 1994). However, evaluation of group selection in nature requires knowledge of metapopulation dynamics at both a genetic and an ecological level.

Fourth, conflicts between selection within populations and selection for colonization or population persistence (e.g., allocation to dispersal *vs.* competitive success) may limit character

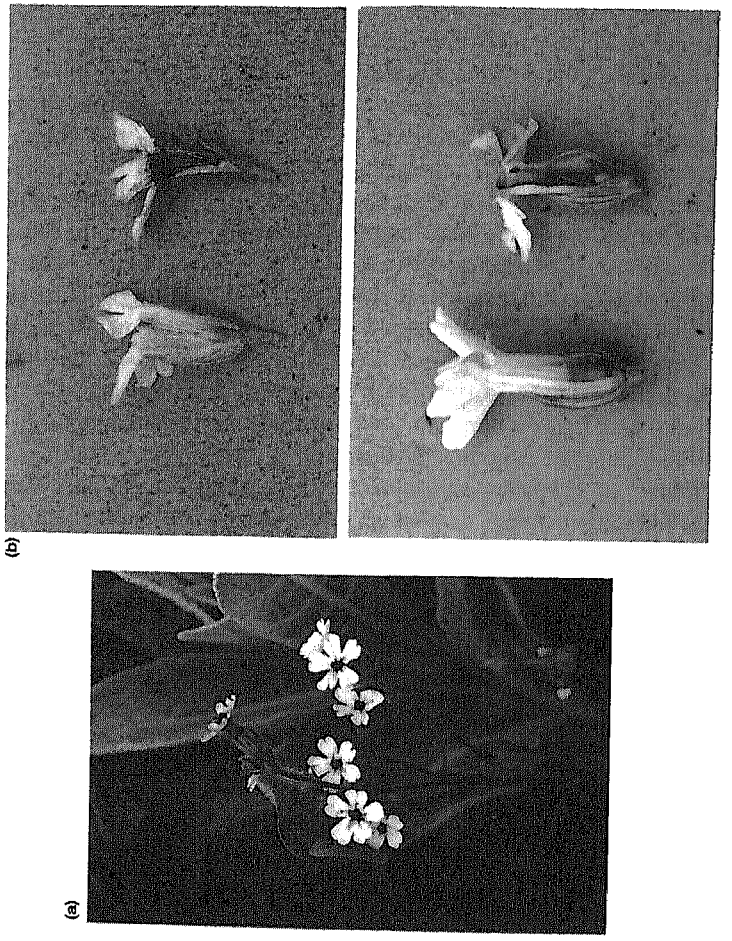
evolution (Roff 1994; Olivieri, Michalakis, and Gouyon 1995), and this in turn may limit evolutionary response to extinction (Meagher, Antonovics, and Primack 1978) or to range extension at species boundaries (Carter and Prince 1981). Metapopulation genetics may therefore play a crucial role in explaining limits to species distributions and predicting evolutionary responses to environmental change.

Therefore, while population geneticists can point with pride to the achievements of their discipline in taking spatial processes into account, there remain many unexplored areas, and there is room for new ideas and insights. There is also a need for many of the ideas to enter the mainstream of population genetics. For example, although all individuals live in spatially explicit situations, very few population genetics texts consider dispersal as a primary fitness component; at best, migration is introduced as a complication of the Hardy-Weinberg law and then promptly forgotten.

In this chapter our primary goal is to use our studies on the *Silene-Ustilago* host-pathogen system to illustrate how the interaction of genetic variation with population dynamics is critically important for host-pathogen dynamics on a broad regional scale. Our secondary goal is to illustrate some methodological principles regarding the study of spatially extended populations. In particular, we hope to convince the reader that the simultaneous study of multiple populations is not necessarily a dauntingly impossible task; indeed, it may actually be easier and more informative than the detailed study of a few target populations.

THE *SILENE-USTILAGO* METAPOPULATION

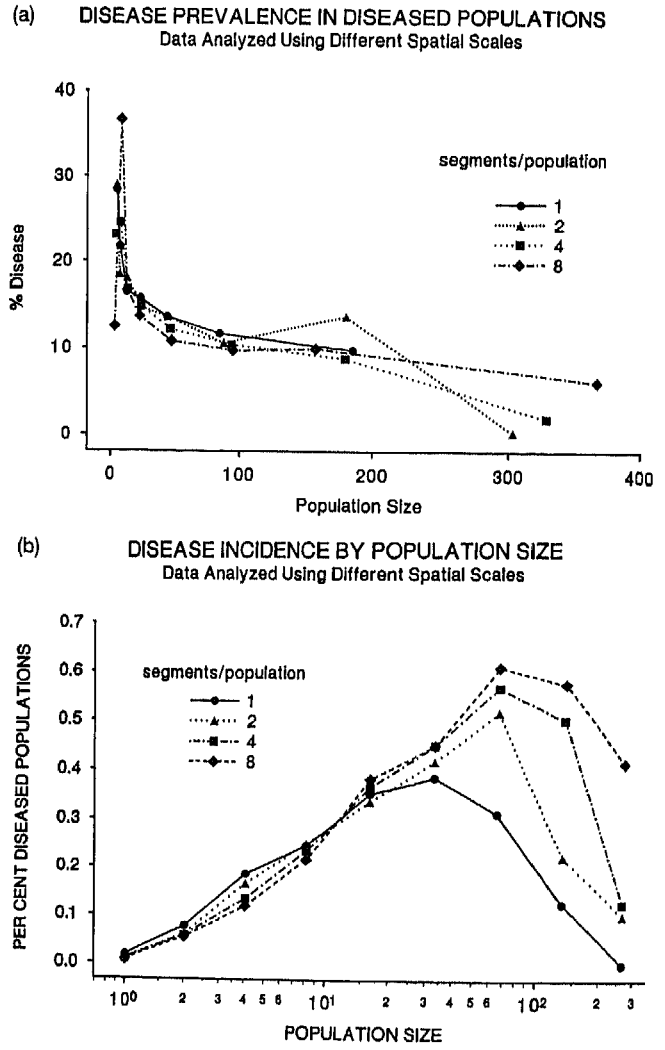
We have been studying populations of the short-lived perennial plant *Silene alba* (white campion) and its associated fungal pathogen *Ustilago violacea* (anther smut). The disease has an intriguing biology. Infection results in anthers that produce fungal spores rather than pollen (Figure 7.1). The disease is pollinator transmitted, and diseased plants are sterilized. This system has added interest because the transmission properties of the disease have much in common with other sexually



transmitted diseases (Thrall, Antonovics, and Hall 1993; Lockhart, Thrall, and Antonovics 1996; Roy 1994; Kalz and Schmid 1995).

In our study area, in the Allegheny Mountains of western Virginia, the host plant is almost entirely restricted to roadsides, and, moreover, the pathogen is restricted to this one species (Antonovics et al. 1995b). Because the plant is distributed in patches of differing sizes and spacings, which may coalesce or separate due to colonization and extinction events, we do not define a population in terms of the patches themselves but count numbers of diseased and healthy individuals within contiguous forty-meter segments of roadsides (Antonovics et al. 1994). Local landmarks (unusual trees, driveways, telephone poles, etc.) are used to demarcate each segment. The scale of forty meters includes perhaps one or two, but not many, genetic neighborhoods (as estimated from spore-, pollen-, and seed-dispersal distances). Moreover, by pooling field data from adjacent segments, we have found that the patterns of disease incidence are remarkably robust over several scales (Figure 7.2). The census includes several thousand segments spanning 150 kilometers of roadsides, of which about four to five hundred are occupied by *S. alba* in any one year. For the past nine years, we have counted the number of diseased and healthy individuals within each segment, followed by a recensus later in the season to check extinctions of the host or pathogen. We generally make no attempt to map individuals within a segment to a precise location (except to help relocate, say, rare diseased individuals or new colonists). Our census is therefore simple and rapid, and fieldwork can be completed by three crews of two to three people in one week.

FIGURE 7.1. (a) A diseased plant of *Silene alba* from Virginia showing flowers with the conspicuous black centers that result from the production of spores by smutted anthers. (b) Sections of healthy and diseased flowers of *Silene alba*. Top panel: male flowers. Bottom panel: female flowers. Note that in females the smut fungus induces production of stamens with anthers that carry smut spores, and the female gynoecium is rudimentary and sterile.



Colonization, extinction, and population interconnectedness play an important role in the dynamics of this pathosystem (Antonovics et al. 1994; Thrall and Antonovics 1995). Between 1989 and 1993 the survey included from 412 to 494 occupied segments (which we call populations) per year. Of these, from 16%–19% were diseased, with an average disease frequency of 24%–42%. The populations have a high turnover rate: Extinction rates of healthy populations have been 14%–22%, and the disease has been lost from host populations at a rate of 19%–36%. The relative constancy of the system is maintained by correspondingly high colonization rates: Over this period, colonization rates were 15%–29% for healthy populations and 23%–45% for the disease. Extinction rates are higher for small populations, and colonizations are decreasing functions of distance from preexisting populations (Thrall and Antonovics 1995). Growth rate of healthy populations is density dependent, with the disease having a marked impact on population growth. In particular, high levels of disease shift population growth rates from positive to negative values (Figure 7.3). However, there is no significant difference in the extinction rate of diseased and healthy populations when corrected for population size. The impact of the disease on population extinction is therefore gradual; the disease results in a declining population growth rate, and small population size in turn presages an increased probability of extinction. The overall

FIGURE 7.2. (a) Relationship between the size of diseased populations and disease prevalence (percent infected individuals) for different sampling scales of the metapopulation. The different sampling scales are generated by pooling adjacent roadside segments into successively larger groups (as shown in the key). Data are from the metapopulation census (1988–93). To account for ascertainment bias, percent infection is calculated as $100 \times (D - 1)/(T - 1)$, where D and T are number diseased and total number, respectively. (b) Relationship between the probability that a population is diseased and the size of the population for different sampling scales of the metapopulation. The different sampling scales are generated by pooling adjacent roadside segments into successively larger groups (as shown in the key). Data are from the metapopulation census (1988–93). The points on the x-axis are means of logarithmically increasing size classes.

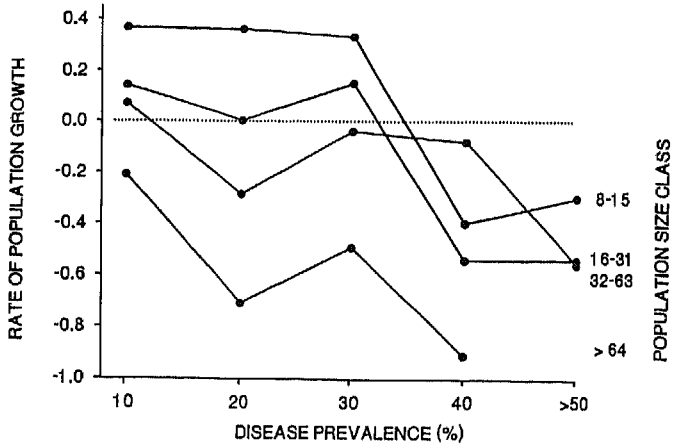


FIGURE 7.3. Growth rate of diseased populations of different sizes as a function of disease prevalence. Growth rate is measured as the log (numbers at time $t + 1$/numbers at time t); prevalence is the frequency of diseased individuals at time t. Data are for successive censuses of the metapopulation for the period 1988–93. Populations in the smallest size class (< 8) showed no significant relationship of growth rate with disease frequency and are not shown.

effects of the pathogen on host abundance are therefore difficult to infer directly, but using the simulation described below, we have shown that the presence of the pathogen can more than halve the number of occupied segments in the metapopulation as a whole. Such long-term regional consequences would be imperceptible from a simple, one-time descriptive study of disease incidence.

Superimposed on this metapopulation structure is the "complication" that there is substantial genetic variation for disease resistance in the host plant. Some genotypes are almost completely resistant, yet others are very susceptible; moreover these differences are highly heritable (Alexander 1989; Alexander, Antonovics, and Kelly 1993; Biere and Antonovics 1995). However, the precise genetics underlying the resistance is not known. Additionally, there are large fitness costs associated with resistance in the absence of the disease; more resistant plants flower later in the season and produce fewer flowers

(Alexander 1989; Biere and Antonovics 1995). Unexpectedly, the fungus appears to be relatively uniform with regard to its virulence, and therefore this host-pathogen system does not follow the classical gene-for-gene scenario (Burdon 1987; Jarosz and Burdon 1991).

THE *SILENE-USTILAGO* SIMULATION

In any large-scale study, experiments that manipulate the entire system are almost impossible. No doubt the metapopulation experimentalist dreams of the day when military-style spending (perhaps accompanied by military-style invasion and coercion) are part of ecology, but in the interim the only recourse is to develop simulation models of the system and to study it "experimentally" by manipulating parameters and conditions on a computer.

Because our goal has been understanding, not management, we have not tried to develop a totally "realistic" model, as might be the case if we wished to make precise predictions about the future fate of *Ustilago* and *Silene* in Giles County, Virginia. Instead, our strategy has been to begin with simple and general heuristic models of pollinator (or sexually) transmitted diseases and to add minimal complexity in a stepwise fashion so as to capture particular features of a real-world metapopulation. By having an understanding of the simpler single-population models, we can then assess the importance of the added features of spatial structure.

Within Population Dynamics

We assume that resistance is determined by a single locus with two alleles, but that the pathogen is genetically uniform. We have no knowledge of the number of genes involved in resistance and make the one-locus assumption for simplicity. We assume that plant reproduction occurs early in the season and is followed by infection, overwintering death, or both. The latent period between spore deposition and the appearance of infected flowers averages over six weeks (Alexander et al. 1993), and plants that become diseased in the first season still

show a substantial reproductive success in that year (Alexander 1990; Biere and Antonovics 1995). We also assume that there is no host recovery and that the death rates of healthy and diseased individuals are the same. Recovery can occur, but it is usually from late-season infections that are unlikely to impact greatly on the overall dynamics. Death rates of diseased plants can be greater than those of healthy plants in some years (Alexander and Antonovics 1995; Thrall and Jarosz 1994b), although the overall averages are usually not significantly different. If we let X_t, Y_t, and N_t represent, respectively, the numbers of healthy hosts, infected hosts, and total host population size at time t, then the within-population dynamics can be represented by the following equations (for brevity, we only present the equation for the i^{th} host genotype):

$$X_{i,t+1} = X_{i,t}[b_i + (1 - P_i)(1 - d)] \tag{7.1}$$

$$Y_{t+1} = \left(Y_t + \sum_i P_i X_{i,t}\right)(1 - d) \tag{7.2}$$

where d is the death rate and b_i is the recruitment rate (number of seeds reaching adulthood); a cost of resistance is included by assuming that b_i is greater for the less resistant genotype. P_i is the probability that a healthy plant becomes diseased. Under nonlinear frequency-dependent disease transmission, this is given by

$$P_i = 1 - \exp\left(-\beta_i \frac{Y_i}{N_t}\right). \tag{7.3}$$

The parameter β represents the effectiveness of disease transmission and can be expanded to take into account both number of contacts (i.e., pollinator visits) and per-contact infection rates (Thrall, Biere, and Uyenoyama 1995; Antonovics et al. 1995b). Seeds of each genotype are produced according to Mendelian expectations based on the frequency of genotypes in the pollen pool (including immigrant pollen) and the fecundity and genotype of each female parent.

We assume the recruitment rate b_i declines hyperbolically as population density (N_t) increases such that per-capita reproduction is given by

$$b_i = \frac{\lambda_i}{\gamma N_t + 1} \tag{7.4}$$

where λ_i is the maximum reproductive rate of the i^{th} host and γ is the strength of density dependence. Hyperbolic functions are good representations of density-dependent growth in plant populations (Harper 1977; Thrall, Pacala, and Silander 1989). In the simulation, we assign carrying capacities to the component populations by varying γ. We assume that the carrying capacity of the populations is variable, and we infer the distribution of carrying capacities from the average size of healthy populations that have persisted for the whole census period.

Among Population Dynamics

We assume that the dynamics within each segment are deterministic, whereas the dispersal and extinction/colonization phases are stochastic. This allows us to distinguish chance processes associated with spatial structure from other chance effects occurring within small populations. It also allows a clearer comparison of the simulation outcomes with results from single-population models.

Seed, pollen, and spores are dispersed using dispersal curves that approximate the empirical data for new colonizations of the plant and the pathogen (Antonovics et al. 1994). Following dispersal, new numbers of hosts and pathogens are calculated for each patch, and the dynamics are repeated. We consider initial colonists and subsequent migrants as part of the within-population dynamics. At the end of each time interval, for both disease and host, a probability of extinction is calculated for each occupied segment using the relationship between extinction probability and populations size as determined from the census data (Thrall and Antonovics 1995).

Unless otherwise indicated, the following estimates of the disease transmission parameter (β_i), the birth rate (λ_i), and the death rate (d) were used in the simulation. For susceptible and resistant hosts, respectively, values of β_i were 5.8 and 0.4, and values of λ_i were 2.0 and 1.5. The death rate for healthy and diseased individuals was assumed to be 0.5. These values were obtained by pooling data from several field experiments (Thrall and Jarosz 1994a,b; Alexander and Antonovics 1995). An example of the graphical output of a typical simulation run is shown in Figure 7.4a. It can be seen that as the disease spreads in an area it reduces the number of occupied sites, and often the disease goes locally extinct as a result of both the increased spacing among occupied sites and the local increase in the resistance gene (not shown).

IN VITRO METAPOPULATION EXPERIMENTS

In this section we use the simulation of the *Silene-Ustilago* system to carry out "experiments" that explore issues pertaining to the interaction of numerical and gene frequency

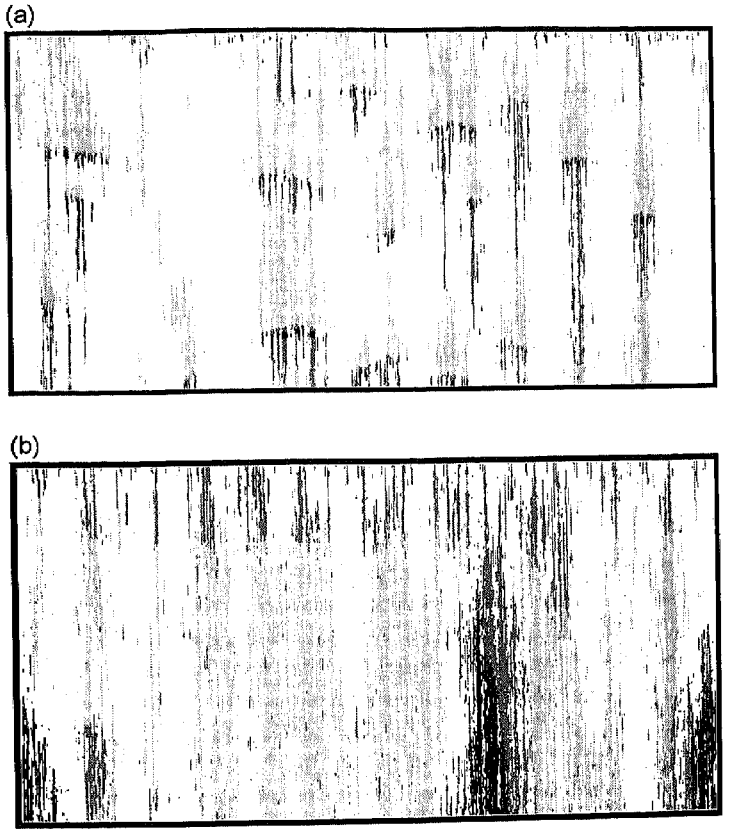


FIGURE 7.4. (a) Example of graphical output of the metapopulation simulation. The horizontal axis represents a linear array of six hundred roadside segments, and the vertical axis represents a time period of three hundred years starting at the top. Each screen pixel is therefore one roadside segment at one time interval. White (background) represents unoccupied segments. Gray represents healthy populations, dark gray represents diseased populations with less than 50% diseased individuals, and black represents heavily diseased populations (more than 50% diseased). (b) Graphical output of the metapopulation simulation illustrating the operation of Wright's shifting balance theory (Wright 1931). The form is as in (a) except that the shading represents the genotypic frequencies of AA, Aa, and aa in each roadside segment. Gray represents populations fixed (frequency > 99%) for a, dark gray represents polymorphic populations, and black represents populations fixed for A. There is heterozygote disadvantage (relative fitnesses are $AA = 1$, $Aa = 0.5$, and $aa = 0.67$), but A is initially at a lower frequency (10% in all populations). Single-population deterministic models predict that A should be eliminated; instead, it spreads because it attains a high frequency in local sites due to stochastic founding events, as predicted by the shifting balance theory.

dynamics in spatially extended populations. We first investigate how the expectations from single-population dynamics are changed by metapopulation structure, in populations where the host is either genetically uniform or where it shows genetic variation for resistance.

In our second experiment, we "pretend" (using our simulation) that we are ecologists who have made field observations of the disease, but that we have done so without knowledge of the underlying genetics. To do this we run our simulation assuming the transmission parameters and reproductive outputs are weighted averages of the component genotypes (as would happen if we made measurements without regard to the underlying genetic heterogeneity). We then ask whether assuming no genetic variation makes a difference to the predictions about subsequent coexistence and abundance of the host and pathogen. Given that extra effort is needed to characterize genetic parameters in a field study, it is important to know whether genetics is simply "another form of heterogeneity" that has little effect on the average trajectory of the system, or whether it can have substantial effect on the dynamics.

In the final experiment we illustrate how the outcome of selection in single populations can be quite different from the outcome of selection in a spatially explicit and more realistic ecological context. When we first simulated selection on alleles at a single locus in an earlier *Silene-Ustilago* model, we found that the rate of loss of a deleterious allele was far slower in the metapopulation than would have been predicted in a single large population (Thrall and Antonovics 1995). Here we use the example of selection at a single locus when there is heterozygote disadvantage to illustrate the potential operation of Wright's shifting balance theory in the *Silene* metapopulation that we have been studying.

Experiment 1: The effect of metapopulation structure on coexistence in genetically uniform versus genetically variable host-pathogen systems

We begin by considering how host-pathogen coexistence is affected by different levels of host resistance in a single genetically uniform population. If the hosts are uniformly as resistant

as the most resistant genotypes that we find in the field, then the pathogen cannot persist in a single population (Table 7.1a; lowest value of β). On the other hand, if the hosts are uniformly less resistant (i.e., as susceptible or even more so than the most susceptible genotypes found in the field), then hosts and pathogens coexist (Table 7.1a; high values of β). Because we assume reproduction in any given season always occurs prior to disease transmission (Eqs. 7.1–7.4), it is not possible for the pathogen to cause the extinction of the host. However, where disease transmission occurs prior to reproduction, the pathogen can cause the extinction of the host (Antonovics 1992; Thrall et al. 1995).

We next compare the predicted single-population dynamics with the outcomes in the metapopulation simulation. The results (Table 7.1a) show that in genetically uniform populations it is harder for the disease to persist in the metapopulation as a whole than in a single population. This is because in small populations the disease has a high extinction probability, and all newly founded populations are initially healthy because the disease is not seed transmitted. Only when the host is quite susceptible (β is 2 or greater) does the disease persist consistently (90% of the time or more) in the metapopulation (Table 7.1a). Even when the disease persists, a substantial fraction of the populations are healthy because they are newly established and have not yet acquired the disease.

We now introduce genetic variation for resistance and susceptibility, and an associated cost of resistance (as we have found in our real-world populations), and again examine single versus metapopulation dynamics. For brevity, we term the less resistant genotype "susceptible" and the more resistant genotype "resistant." We use these terms to evoke contrasting properties, acknowledging that susceptibility is really the inverse of resistance. In a single population, genetic polymorphism is more likely when the genotypes differ greatly in their resistance and susceptibility (Figure 7.5); this happens over quite a broad range of resistance costs (as in other models of this type; Antonovics and Thrall 1994). When the genotypes differ less in their resistances, either the resistant or the susceptible allele goes to fixation (depending on the cost of

TABLE 7.1. Effect of varying plant resistance to the anther smut disease on disease frequency, in either a single population or in the metapopulation. The single-population values are from deterministic models, and the metapopulation values are means of ten runs of the *Silene-Ustilago* metapopulation (standard errors are not shown but were generally 5–10% of the means). Values for % persistence refer to persistence for three hundred generations out of one hundred runs; values for % disease are averages of diseased populations only. Models are described in the text.

(a) Disease frequency in genetically uniform hosts	Resistance (β) 0.41	1.25	1.50	2.00	5.86
Single Population					
% individuals diseased	0	13.2	21.8	32.2	48.5
Metapopulation					
% disease persistence	0	9.0	38.0	90.0	91.0
% sites diseased	—	6.6	22.2	55.5	43.0
% disease	—	42.1	36.0	19.5	23.2

(b) Disease frequency and resistance in genetically variable hosts	Resistance (β) of *RR* 0	0.41	1.50	2.00
Single Population				
% diseased	2.9	3.25	21.9	48.5
Metapopulation				
% disease persistence	14.0	51.0	79.0	80.0
% sites diseased	16.2	22.5	42.0	50.8
% disease	34.5	33.5	41.1	44.3
Frequency of Resistant Allele (%)				
Single Population	81.5	87.8	100.0	0.0
Metapopulation	33.8	31.7	2.3	0.0

Note: Resistance of the susceptible genotype is set to $\beta = 5.86$.

resistance). The effects of decreasing the resistance of the more resistant genotype are somewhat surprising (the row of X's in Figure 7.5; Table 7.1b): as its resistance is decreased (keeping costs the same), the resistant allele actually reaches a higher frequency, and there is a region over which it becomes fixed. The reason is that as the resistance of the most resistant genotype decreases, disease frequency increases, and this fur-

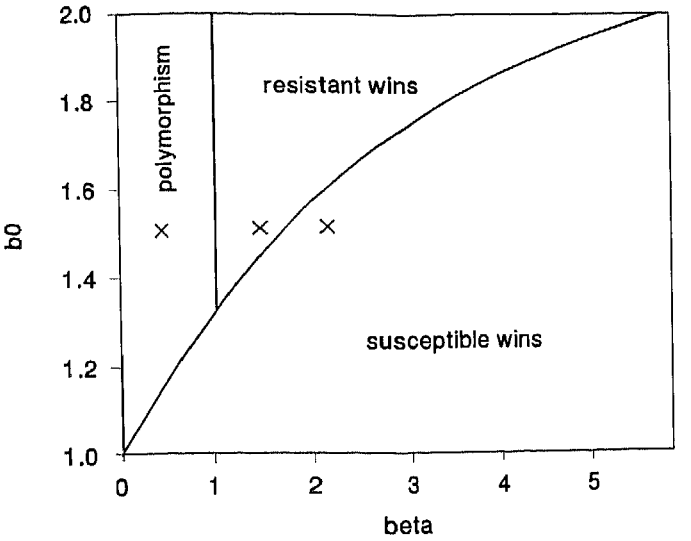


FIGURE 7.5. Phase diagram illustrating regions of polymorphism and monomorphism when resistance is determined by one locus with two alleles, R (resistant) and r (susceptible), in a single-population deterministic model. See the text for the model description. It is assumed that rr has a transmission coefficient of 2 and a reproductive output of 2. The diagram shows the equilibrium regions for increasing values of the transmission coefficient and increasing values of the reproductive output of the more resistant RR genotype (we assume heterozygotes are intermediate). Resistance of this genotype decreases from left to right, and the cost of the resistance decreases from top to bottom.

ther increases the selection on this allele. This continues until the selective advantage due to resistance becomes too small relative to the cost, at which point the susceptible allele now goes to fixation. If we consider resistances and costs typical of the *Silene-Ustilago* system, the resistant allele is predicted to increase to a high frequency (87.8%) and disease levels should be very low (3.2%) (Table 7.1b). Given such low equilibrium values, one might expect that the disease would almost never persist in a metapopulation context.

However, in the metapopulation simulation the disease persists 56.7% ($n = 20$) of the time. Moreover, when it persists,

the percentage of populations that are diseased is substantial (22.5%) as is the prevalence of the disease in these populations (33.5%). There are several reasons for the high level of persistence of the disease in the metapopulation. First and foremost, many newly founded populations contain only susceptible individuals: In these populations the disease can establish rapidly and reach a high frequency. Second, the initial disease frequency in most populations where the disease has just arrived will be greater than the low equilibrium frequency expected in a single isolated population. Third, these two effects will interact—the high incidence of disease in sites with susceptible hosts will result in higher disease colonization rates of all populations, that is, there is an increase in the effective pathogen growth rate over the metapopulation as a whole.

Whereas in a single population, decreasing the resistance of the most resistant genotype results in an increase in its frequency (see above), in the metapopulation the reverse effect is seen. Indeed, at values where the resistance allele would go to fixation in a single population, it persists only at a low frequency in the metapopulation ($\beta = 1.5$; Table 7.1b). The reason is that when the resistance allele is less extreme, it reaches a higher final equilibrium frequency, but it takes much longer to attain that frequency. For example, if the resistant allele is started at a frequency of 10%, after twenty-five generations it reaches 42.6% in the case of $\beta = 0.41$, but only 12.5% when $\beta = 1.50$. Continual extinctions and recolonizations therefore maintain the resistance allele at a low frequency because within most populations equilibrium is not reached before there is extinction of either the host or the pathogen.

Experiment 2: The effect of ignoring genetics on the future dynamics of a metapopulation

In this experiment, we ran the simulation for a substantial period of time (three hundred generations) and then assumed we were ecologists estimating population parameters without regard to the underlying genetics. To do this, the disease transmission coefficient and host reproductive output were

calculated from the weighted averages of the component genotypes (as would happen if we made measurements on individuals randomly sampled from the metapopulation). Because we assumed an exponential model of transmission, the disease transmission coefficient was calculated as follows: We first calculated the weighted average of the probability that the genotypes would become diseased at the extant disease prevalence and then calculated the transmission coefficient that would give this same probability in a uniform population with the same disease prevalence. Disease prevalence was calculated as the overall frequency of disease in diseased populations. Only runs where the disease had persisted for three hundred generations were included.

When the simulation was continued assuming the populations were genetically variable, the disease only persisted for a further three hundred generations in 40% of the runs ($n = 40$). However, when we assumed the populations were genetically uniform, the disease nearly always persisted (85%, $n = 40$). In the genetically variable populations, the periodic spread of the highly resistant genotypes resulted in large fluctuations in the frequency of disease and a high probability of global extinction. In the uniform population, the moderate but relatively constant level of host susceptibility resulted in much more stable dynamics and longer-term persistence.

Experiment 3: Selection against heterozygotes

In 1931 Wright suggested that evolution might be more rapid in a group of smaller populations connected by occasional migration than in a continuous panmictic population because chance effects in small populations might permit the evolution of traits whose intermediate condition might be disadvantageous. The simplest single-locus scenario of this is when the alleles underlying the trait in question show heterozygote disadvantage. In a single population with heterozygote disadvantage, the intermediate allele frequency equilibrium is unstable, and a rare allele cannot invade even if it is superior in fitness in its homozygous condition to the alternative, more common, allele.

In our simulation, we excluded the disease completely and reset the fitness values (as given by the birth and death rates) of the host genotypes such that heterozygotes were at a disadvantage. We illustrate the case where the relative fitnesses of *AA*, *Aa*, and *aa* were set to 1, 0.5, and 0.67, respectively. In a single infinite population, there is an unstable equilibrium at $A = 0.25$, and below this frequency, *a* always wins (even though *aa* has a lower fitness than *AA*). In the metapopulation simulations we introduced *A* at an initial frequency of 0.1 in all populations.

Contrary to single-population expectations, in 20% ($n = 25$) of the runs, the *A* allele spread to fixation (Figure 7.4b), although often it took several thousand generations to do so. There were long periods when the population was characterized by spatially separated patches that were close to fixation for either *A* or *a*. As expected, either decreasing the degree of heterozygote disadvantage or increasing the initial allele frequency of *A* increased its likelihood of spreading in the metapopulation. These results show that Wright's conjecture that metapopulations may be theaters for evolutionary processes that are impossible in large single populations has cogency in this particular simulated real-world metapopulation. Moreover, it is likely that our results underestimate the likelihood of such effects because in our simulated metapopulation the numerical and allele frequency dynamics within each population were deterministic.

SUMMARY

In this chapter we have used a combination of field, theoretical, and simulation studies to illustrate several principles relating to the impact of genetics on metapopulation dynamics. It is clear that genetics is not "just another" heterogeneity; averages of the genetic values fail to accurately capture the dynamics of the system. Spatially explicit processes create situations that amplify the impact of genetic variation. In population genetics, it has long been recognized that limited dispersal will result in the spatial redistribution of genetic variation such that variation increases among subpopulations but decreases within

patches. As emphasized by Frank (1997), chance effects associated with colonization and extinction processes in metapopulations can result in the dynamics of coevolutionary systems becoming highly unpredictable from single-population theory. Our study shows that the redistribution of genetic variance during the colonization phase can have profound effects even in the simplest of cases (we assumed genetic variation only in the host).

For a biologist, the final arbiter of the importance of a theoretical construct is whether it generates explanatory power that can lead to a greater understanding of the natural world. Theory can provide powerful insights into what is plausible, but it needs to be integrated with empirical studies to assess whether particular processes are actually likely in nature. Nowhere is this currently more true than in the area of population substructuring and its relationship to group selection (Wilson 1983). Therefore we need more empirical data on spatially distributed populations in the real world, with an emphasis on studies that gather information about genetic and ecological connectedness, and extinction and colonization dynamics. Ironically, our own study was initiated by an interest in finding populations as targets for detailed individual study, until we realized the feasibility of extending the sampling to a regional scale.

At first sight, it would seem that there are many deterrents to the study of populations on a regional scale. More effort may be necessary to study many versus a few populations, and colonization and extinction may be rare events and hard to document. If populations form a patchy continuum, it is hard to know at what scale a study should be done or how populations that are components of the larger metapopulation should be defined. In addition, experiments are likely to require large spatial and time scales. Our study was greatly facilitated by both the accessibility and linear arrangement of the roadside populations. Of particular importance was our decision to study the system as a "connected lattice," using a grid superimposed on a patchwork of populations. This enabled us to obtain crude census data on each segment of the grid quickly (obviating the mapping and detailed study of the fate of individuals) and to

focus more on events at a regional scale. After nine years of experience with this study we have become convinced not only that the study of metapopulations in nature is very feasible (especially in plants) but that major misunderstandings can emerge from studying only one or a few populations as a guide to long-term dynamical outcomes. Thus, in the study of single populations, it is unclear what criteria should be used to choose a "representative" population, how population boundaries should be defined, how to test causal processes when replication is limited, and when to discount the "unusual disaster"; there is also the mundane problem of how to avoid damaging plots in the process of intensive measurement and sampling. Whereas experimental field studies on metapopulations are likely to be labor intensive, computer simulations developed interactively with both descriptive field data and smaller manipulative studies can become a valuable surrogate for such experiments.

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