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8

Ecological Genetics of Metapopulations: The *Silene-Ustilago* Plant-Pathogen System

(with Peter Thrall, Andrew Jarosz, and Don Stratton)

Introduction

There has been a long-standing recognition that the numerical and gene-frequency dynamics of natural populations will be affected both by processes that occur locally within populations as well as by factors that affect the interconnectedness and persistence of populations on a more regional scale (Hutchinson 1953; Wright 1943). Historically, and perhaps also for heuristic and practical reasons, primacy has been given to a study of processes within populations. Recently, however, a number of issues have forced greater attention on population processes at a regional scale. These issues include practical problems such as predicting the consequences of habitat fragmentation (Burkey 1989; Wilcox 1990), as well as conceptual advances such as the demonstration that populations which cannot persist locally may still show equilibrium persistence on a regional scale (Levins 1969; Caswell 1978; Hanski and Gilpin 1991) and that genetic structure can be greatly affected not just by migration among extant populations but by colonization and extinction processes (McCauley 1991). Above all there has been an increasing recognition that in nature no population is an isolated entity, and that this reality needs to be quantified and its consequences need to be explored. Metapopulations (systems of interconnected populations) are more likely to be the rule, not the exception.

The major theoretical and empirical issue at the heart of discussions about metapopulations is how the global behavior of a system of interconnected populations can be understood in terms of the local dynamics of individual populations, their degree of synchrony, and the connectedness among them (Hanski and Gilpin 1991). This issue can be broken down into two interacting questions. The first question is the degree to which interconnectedness of populations affects local dynamics. The second question is the degree to which local dynamics in turn affects outcomes on a more regional scale. These questions are problematical because although "a population" may be easily defined in theory or by the confines of an experimental container, in a mosaic patchwork such as is found in nature there is no easily agreed-upon definition of a population. The ecological, genetic, and evolutionary criteria that have been used for delimiting a population may give quite different dimensions (Antonovics and Levin 1980; Uyenoyama and Feldman 1980). One extreme population structure would be exemplified by situations where "populations" are represented by cells within a grid superimposed upon a continuum of individuals and where there is a high degree of interpatch migration, while the other extreme would be exemplified by "populations" that are on quite isolated islands or in distinct habitat patches and where migration rate per generation is rare. The term "metapopulation" has often been restricted to the latter situation, and we use it in that sense here but with the clear expectation that actual population structures may be somewhere between these extremes, or represent some combination of them (e.g., the "core-satellite" hypothesis; Hanski 1982). Much of the challenge of the empirical study of metapopulations is simply defining the actual structure and connectedness of the component study units (Harrison 1991; Taylor, A. D. 1991).

In nature, the interaction between local population dynamics and population connectedness confounds their easy dissection. From a simple description of the numerical dynamics of a local population or patch, one does not know (unless the migrants can be explicitly identified) whether it is being influenced by connectedness. For example, local instability may be undetectable because population extinctions do not occur because of immigration from other nearby populations (the "rescue effect"; Brown and Kodric-Brown 1977). Experimental approaches are necessary which manipulate the frequency of migration/colonization events among component populations. Studies in the laboratory have shown that persistence of a set of populations can be increased by some connectedness among them (Huffaker 1958; Pimentel et al. 1963; Takafuji 1977), and although experimental studies under natural or seminatural conditions are much rarer, they have generally given the same result (Kareiva 1984, 1987; Sabelis and van der Meer 1986; Sabelis and Laane 1986).

The subject of metapopulations has also been approached from a theoretical point of view by subsuming the details of local dynamics in order to achieve generalizations about the more global behavior of the metapopulation as a whole. The pioneering approach of theoretical island biogeography, for example, assumed a source population (or "refuge"), and asked about the effect of migration on population extinction and colonization, while ignoring the details of within-island dynamics (MacArthur and Wilson 1967). The classical metapopulation model of Levins (1969) also assumed no within-population dynamics other than "presence" after

colonization and "absence" after extinction. Models which incorporate local population dynamics ("structured" metapopulation models; Hanski 1991) become much more difficult to analyze theoretically but can lead to qualitatively different results, such as the requirement that there be a minimum fraction of occupied populations for the metapopulation to increase (Gyllenberg and Hanski 1992) or results such as nonmonotonic changes in metapopulation size with increasing migration (May and Anderson 1990). It is therefore important to know under what conditions local dynamics matters; in some instances equilibrium may be approached rapidly and local dynamics can be subsumed with little loss of accuracy (Verboom and Lankester 1991).

Even where structured metapopulation models have been developed, they have usually assumed that migration is important in the colonization process, but that subsequent migrants have little effect on local dynamics (Diekmann et al. 1988; Hastings and Wolin 1989; Sabelis et al. 1991). However, the colonization process involves not only demographic stochasticities, but also genetic stochasticities. Thus, rare migrants subsequent to a colonization event may have little immediate effect on local numerical dynamics, but may have a large impact on the genetic dynamics, which in turn may affect population abundances. For example, migrants may reduce the level of inbreeding that might otherwise ensue if there were only one or a few colonists; or they may introduce genotypes (e.g., resistance or virulence types) that may have been absent in the founding population.

In this discussion we use the *Silene-Ustilago* system to illustrate several important aspects of the ecology and genetics of metapopulation systems. First, we present the results of a long-term study of populations of *Silene alba* and *Ustilago violacea* spanning a broad regional scale that includes several hundred populations; we examine the rates of population turnover, and the degree of interconnectedness among the component populations. Second, we present some initial results from empirical studies and computer simulations which address the question of how genetic structure of host-pathogen systems might affect metapopulation dynamics.

The Silene-Ustilago Host-Pathogen System

Silene alba or white campion (= *Silene latifolia*, *Melandrium album*; hereafter termed *Silene*) is a short-lived perennial herb, commonly found in ruderal habitats throughout the northern regions of the United States and in upland areas farther south (McNeill 1977). It is easily grown and flowers in about six weeks under long days in a growth chamber; it is dioecious and easily crossed. *Ustilago violacea* or anther-smut fungus

(= *Microbotryum violacea*; hereafter termed *Ustilago*) causes both male and female plants to produce anthers that carry fungal spores instead of pollen; diseased females produce a sterile, rudimentary ovary. The disease is systemic, and after the initial stages of infection, all flowers become diseased and the plant is sterilized, although plants becoming diseased toward the end of the growing season may recover. Diseased flowers are easily recognized by their dark, smutted centers. The spores are transmitted by pollinators and to a limited extent by passive scattering (Alexander 1990a). Spores germinate on the host and undergo meiosis to produce haploid sporidia which multiply by budding. Fusion of sporidia of opposite mating type produces a heterokaryotic infection hypha.

Silene shows genetic variation in resistance to the disease in field experiments (Alexander 1989; Thrall 1993). A single population may contain mixtures of highly susceptible and highly resistant individuals, but the precise inheritance of resistance is unknown. Resistance in the field is correlated with resistance in the greenhouse, except that differences in field susceptibility are additionally related to differences among host clones in flowering time (Alexander, Antonovics, and Kelly 1993).

The fungus appears to be genetically much less variable. Artificial inoculations revealed no differences in virulence among a limited number of lines isolated from one population (Alexander, Antonovics, and Kelly 1993). A search for other genetic markers revealed only very limited variation for allozymes and RFLP's (Stratton, unpublished). More recently the use of randomly amplified polymorphic DNA (or RAPDs: Williams et al. 1990; Martin et al. 1991) has been used to successfully differentiate twelve sporidial lines from one population; crosses among these isolates have confirmed the Mendelian inheritance of all markers tested to date (Oudemans, unpublished). However, we do not know to what extent such genetic variation at the molecular level is reflected by variation in virulence.

A major stimulus for our research has been the expectation that *Ustilago*, because it is pollinator transmitted, is likely to show frequency-dependent disease transmission, a transmission mode that is characteristic of venereal or vector-borne diseases (Getz and Pickering 1983). Frequency-dependent transmission occurs when the probability of a healthy individual becoming diseased is a function of the frequency or fraction of individuals in the population that are diseased, rather than a function of overall plant density (see chap. 7). Such transmission might be expected whenever disease vectors (mates or pollinators) move further to compensate for increased spacing among individuals at lower population densities. Antonovics and Alexander (1992) showed that spore deposition increased with frequency but not with density of diseased individuals in experimental populations of diseased and healthy *Silene* exposed to

natural pollinators. Simple models show that while normal density-dependent transmission processes require a threshold density for disease spread, and can readily regulate population size, frequency-dependent transmission modes can lead to either extinction of both host and pathogen or purging of the disease from the population (Getz and Pickering 1983; Antonovics 1992). Host-pathogen coexistence is possible if there is density-dependent host population regulation, for example, by resource limitation, and such coexistence is most likely if density acts more severely on the healthy class than on the diseased class (Thrall, Antonovics, and Hall 1993). Greater density-dependent limitation on the healthy class may be expected in cases (as in pollinator-transmitted and other venereal disease systems) where the disease is transmitted in the adult phase, and density-dependent population regulation occurs primarily in the juvenile (or seedling) stage.

Models that incorporate genetic variation in host resistance show that if there are costs to disease resistance, plant populations can be polymorphic for genotypes that in a monomorphic state would either lead to host-pathogen extinction or failure of the disease to establish (Antonovics 1992; see chap. 7). This leads to the prediction that extremes of resistance and susceptibility can be maintained in one population, a prediction that is supported by the results of Alexander (1989).

Metapopulation Dynamics: Natural Populations

To study a system of interconnected populations, it is necessary operationally to define a component "population" and the universe that constitutes the "metapopulation." We have chosen as our "metapopulation" a 25×25 km area in the vicinity of Mountain Lake Biological Station in southwestern Virginia (fig. 8.1). Our "populations" are defined as those individuals (healthy and diseased) in a 44-yard (c. 40 m) segment of roadside. Over the past three years, we have studied nearly 150 km of roadside, encompassing about seven thousand roadside segments. In any one season, over 300 of these have *Silene* growing in them, and about seventy have at least some diseased individuals. We have found the host and the disease in the immediate area outside our census region as well as up to 50 miles away. We therefore have no reason to believe that our study area is in any way exceptional.

Most metapopulation studies have focused on populations found in discrete habitat types, so that the potential sites available to them can be delimited geographically (Rey and Strong 1983; Harrison 1991; Peltonen and Hanski 1991). However, in our system potential sites are not easily defined, and we therefore describe metapopulation processes in terms of the numbers of individuals in each of the roadside segments. In the region

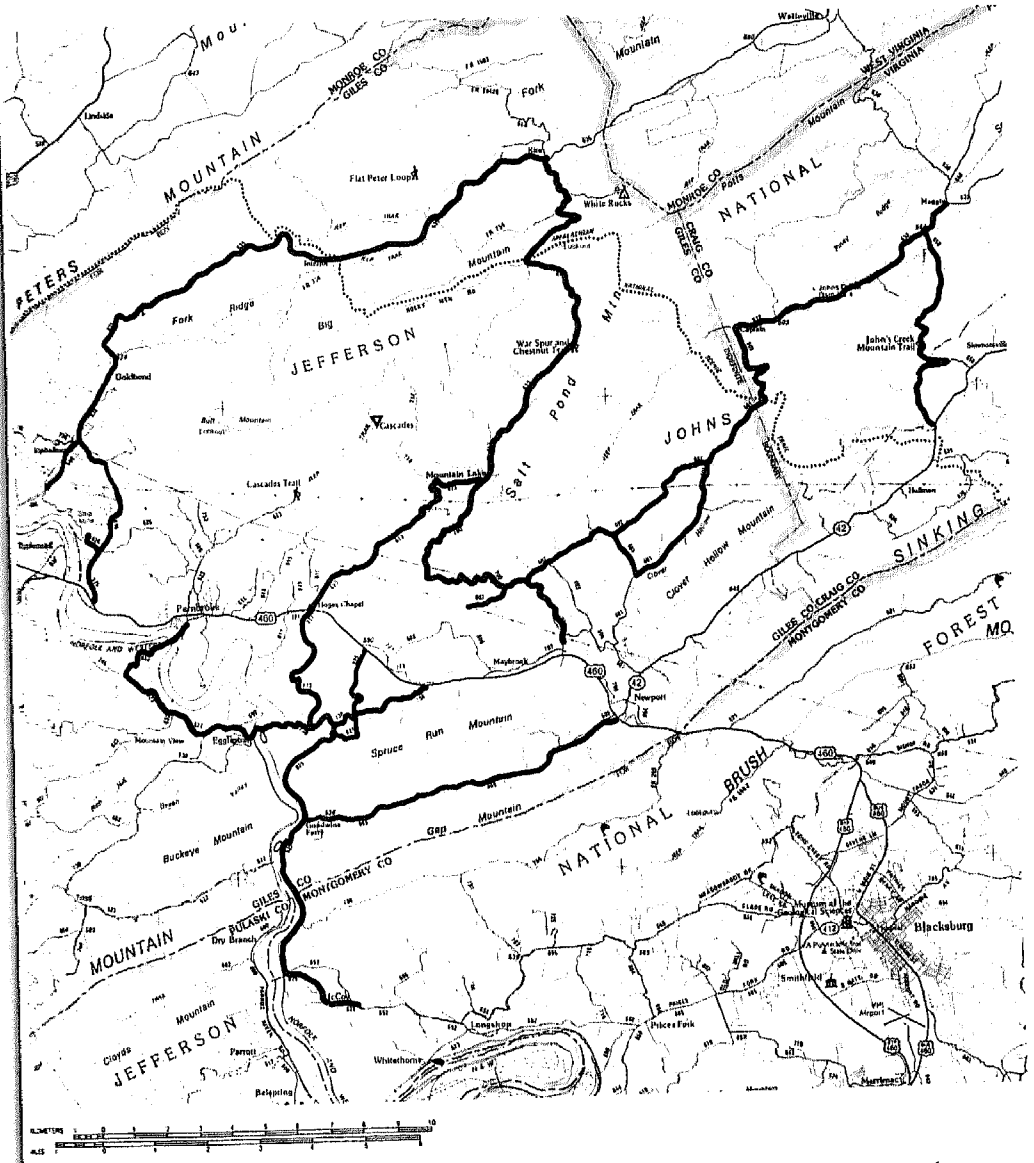

FIG. 8.1. Map showing the roads (thick black lines) included in the annual census of *Silene alba* populations. (From Virginia Atlas & Gazetteer, copyright DeLorme Mapping Co. Reproduced with permission)

of Virginia (Giles County) where we have been studying the *Silene-Ustilago* system, land use is mostly grazed pastures and woodlands and *Silene* is largely confined to roadsides. The scale of 44 yards allows us easily to locate a segment using a car odometer and to study a large number of roadside segments covering a wide area while having, at least potentially, a substantial number of individuals within each segment. Each segment also encompasses at least one, but not numerous, fungal and host genetic neighborhoods. Studies of local spore dispersal showed that at 10 m from a point source, only a third of the flowers had spores deposited on them relative to flowers close to the source; these flowers had only about a tenth of the number of spores as flowers at the source (Alexander 1990a). Because the spores are pollinator transmitted, pollen dispersal is likely to be of the same order of magnitude. On the other hand, seed dispersal is much more limited, with most seeds falling within 2 m of the seed source.

We census the populations twice a year. The main census is carried out in early June, with a secondary census at the end of July to recheck segments that showed critical state transitions (i.e., where plants or disease were not seen in the current year but were present the previous year). During the main census, each roadside segment is marked using local landmarks (unusual trees, telephone posts, buildings, etc.) so it can be relocated precisely from one year to the next. We record each side of the road separately because the sides often differ in aspect, vegetation, disturbance history, and disease incidence. We also take pains to avoid becoming disease dispersal agents ourselves: the census is carried out prior to midmorning closure of the flowers so diseased plants can be easily identified without touching the flowers. Data for two years from a representative one-mile section of road are shown in figure 8.2.

The results of the first three years of the census have shown that there is a high turnover rate of populations (table 8.1a), so much so that it is possible to calculate "vital statistics" for whole populations (table 8.1b) in a manner that is similar to what is normally done for individuals within populations. These results show that over the three-year period 1988–1990, the colonization rate of the disease was greater than the extinction rate. These results suggest that the disease is spreading in this region of Virginia, a trend that is confirmed by comparison with a smaller number of populations in the census area that were studied in some detail in 1984 (Alexander 1990b). For the host, the colonization rate exceeded the extinction rate in 1988–1989, but the reverse was true in 1989–1990. Population extinction rates were "size dependent," being higher in small than in large populations (table 8.2a). Although there has been much speculation about "minimum viable population" sizes in the conservation literature, empirical data which bears on this issue is almost nonexistent (Wilcox 1990). Extinction rates of small populations were less when they

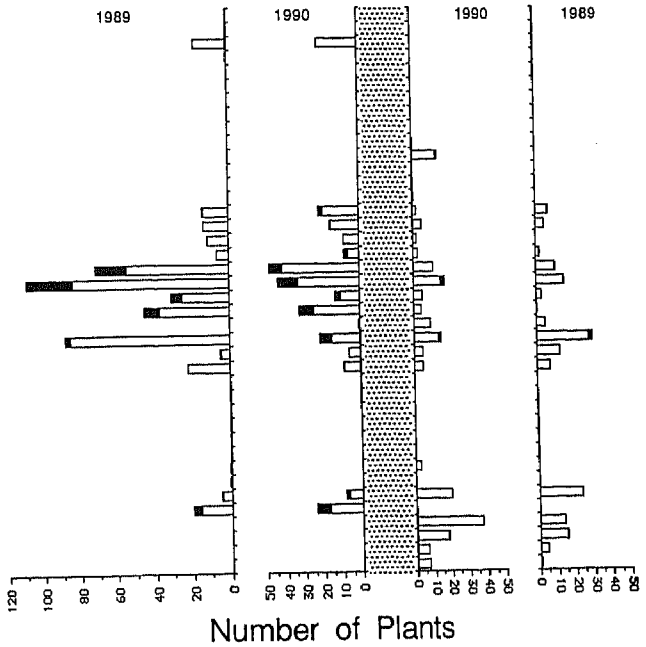

FIG. 8.2. Illustration of data from a representative section of roadway, showing the results of two successive censuses, 1989 and 1990. The road is symbolized by the shaded region, with data for the left- and right-hand side of the road shown on either side. Each tick on the vertical axes delimits a 44-yd roadside segment (total vertical scale = 1 mile); the horizontal bars show the numbers of healthy (open) and of diseased (dark) individuals in each roadside segment in each year.

were in proximity to other populations (table 8.2b), suggesting the possibility that they might have been "rescued" from extinction by migrants from nearby populations (although clearly one cannot exclude the possibility that populations near each other are in favorable habitats).

Interesting patterns have emerged also with regard to disease incidence. Populations are more likely to be diseased when large (fig. 8.3). This again suggests a number of possible scenarios: large populations may attract more long-distance pollinators/spore vectors; or if there is a demographic cost to resistance, large populations may be more disease susceptible; or larger populations may be simply older populations that have had a greater chance of becoming diseased. Patterns similar to these have been seen with *U. violacea* on *Viscaria vulgaris* in Europe (Jennersten et al. 1983). Given that a population is diseased, small populations generally have a higher percentage of disease than large populations (fig. 8.4). This is predicted from frequency-dependent but not density-dependent disease-transmission processes; a single diseased individual in

TABLE 8.1
State transitions and metapopulation vital statistics for the occurrence of *Silene* and *Ustilago* in roadside segments, 44 yd long, in Virginia.

a. State transitions for all segments, where $t - 1$ indicates status the previous year, and t the status in the subsequent year. The category "diseased" includes partially diseased populations. The top number in each cell represents the transition from 1988 to 1989, and the bottom number the transition from 1989 to 1990. (More segments were included in the later censuses.)

	Year t		
Year t – 1	*No Plants*	*Healthy*	*Diseased*
No plants	5,254	139	12
	6,506	70	10
Healthy	19	161	23
	86	240	22
Diseased	3	5	40
	3	18	53

b. Metapopulation vital statistics showing rates of colonization and extinction of healthy populations, and rate of colonization and extinction of the disease (diseased populations). Rates are calculated as the number of new/extinct populations per existing healthy or diseased population per year. Population disease transmission rate is the probability of a healthy population becoming diseased per existing diseased population per year.

	1988 to 1989	*1989 to 1990*
A. Healthy populations:		
Colonization rate	0.60	0.19
Extinction rate	0.09	0.25
B. Diseased populations:		
Colonization rate	0.69	0.42
Extinction rate	0.16	0.30
C. Interaction:		
Rate at which healthy populations become diseased	0.10	0.07
Population disease transmission rate	0.002	0.001

Note: Here, and in subsequent tables, segments on opposite sides of the road are considered separately; occupied segments are termed populations.

TABLE 8.2
Extinction rate of healthy *Silene* populations as a function of their size and distance from other populations. Data are based on a comparison of the 1989 and 1990 censuses.

a. Relationship between population size and extinction rate (fraction of populations of a given size that went extinct between 1989 and 1990).

Population Size	*No. of Populations*	*Extinction Rate*
1	78	0.513
2–3	68	0.221
4–7	68	0.279
8–15	62	0.113
16–31	31	0.097
32–63	18	0
64–127	12	0
128–255	8	0
>255	3	0

b. Relationship between extinction rate of small populations (1–5 individuals) and their distance from the nearest population. Distance is measured in terms of number of roadside segments (each segment = 44 yd), and the nearest neighbor criterion considers populations on both sides of the road; populations directly opposite are considered to be one roadside segment away.

Distance (Roadside Segments) from Nearest Population	*No. of Populations Extinct*	*Extinction Rate*
1	80	0.600
2–3	24	0.667
4–7	24	0.682
8–15	26	0.654
>15	28	0.790

Note: Regression of distance versus weighted arcsine-transformed extinction rate: $Y = 0.845 + 0.0415X$, $p < 0.043$.

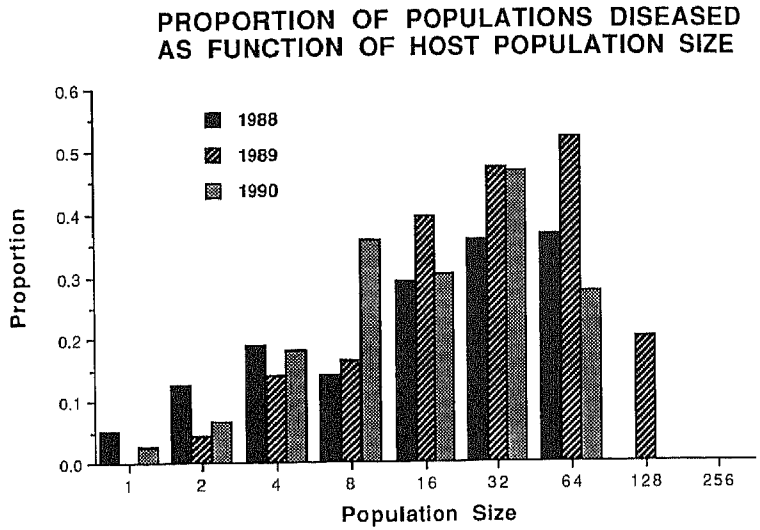

FIG. 8.3. Proportion of *S. alba* populations that are diseased in relation to total population size in 1988, 1989, and 1990. The value for population size on the horizontal axis represents the lower value of the range for each size class.

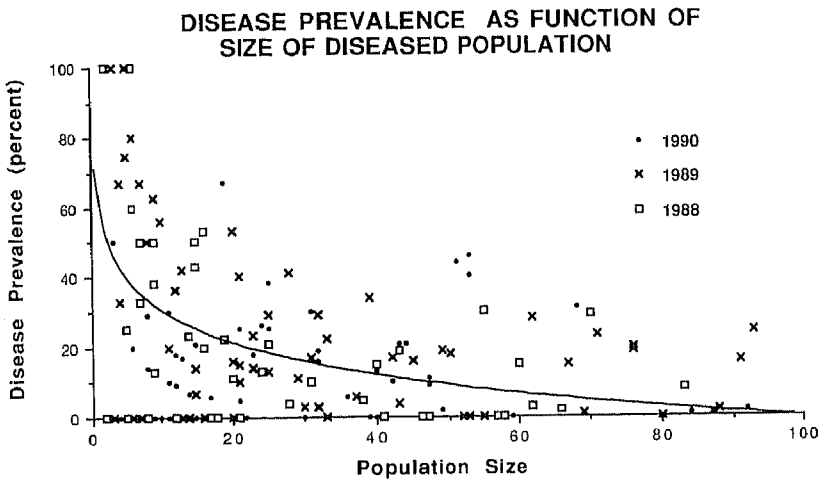

FIG. 8.4. Disease prevalence as a function of population size. To discount the bias that would be introduced by the fact that all diseased populations have to have at least one diseased individual, the disease prevalence (percentage of diseased individuals in the population) is calculated using the transformation $100*(D-1)/(T-1)$, where D and T are number diseased and total number, respectively. Formula for the fitted curve is: $Y = 61.5 - 30.9 * \log(X)$; $R^2 = 0.26$.

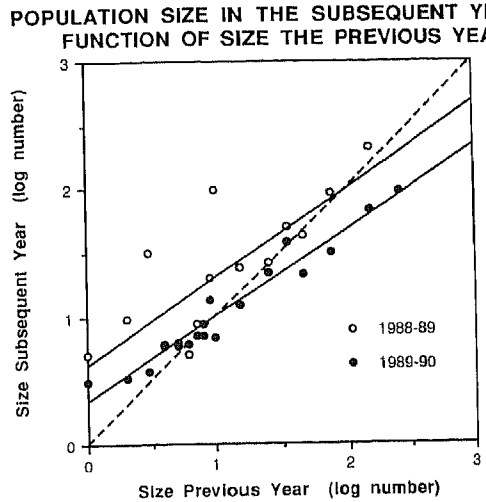

FIG. 8.5. Relationship between size of healthy populations and their size in the subsequent year. Each point represents the average of a population size class grouped such that numbers in each class are not less than five. The dotted line represents no change in population size over successive years; the solid lines are regressions for 1988–1989 (slope = 0.70, $R^2 = 0.64$) and 1989–1990 (slope = 0.67, $R^2 = 0.95$).

a small population will have a higher initial frequency than one in a large population, and the disease will therefore spread faster. With density-dependent disease transmission, smaller (and probably less dense) populations would have lower rates of disease increase.

Comparison of population size changes over successive years shows that the proportionate rate of increase of populations is less if the population size in the previous year was larger (fig. 8.5; table 8.3), or if there was a higher proportion of diseased individuals in the population (table 8.3).

TABLE 8.3
Effect of disease frequency and total population size on population growth rate of diseased populations between 1988 and 1989, and between 1989 and 1990. The table shows regression coefficients from a multiple regression of the natural log of change in size between years as a function of total size and disease frequency of the population in the previous year.

	Disease Frequency at Time t	*Population Size at Time t*	*Model R^2*
1988 to 1989	–0.35 n.s.	–0.42**	0.21
1989 to 1990	–0.52*	–0.36**	0.14

Significance levels: * = < 0.05, ** = < 0.01.

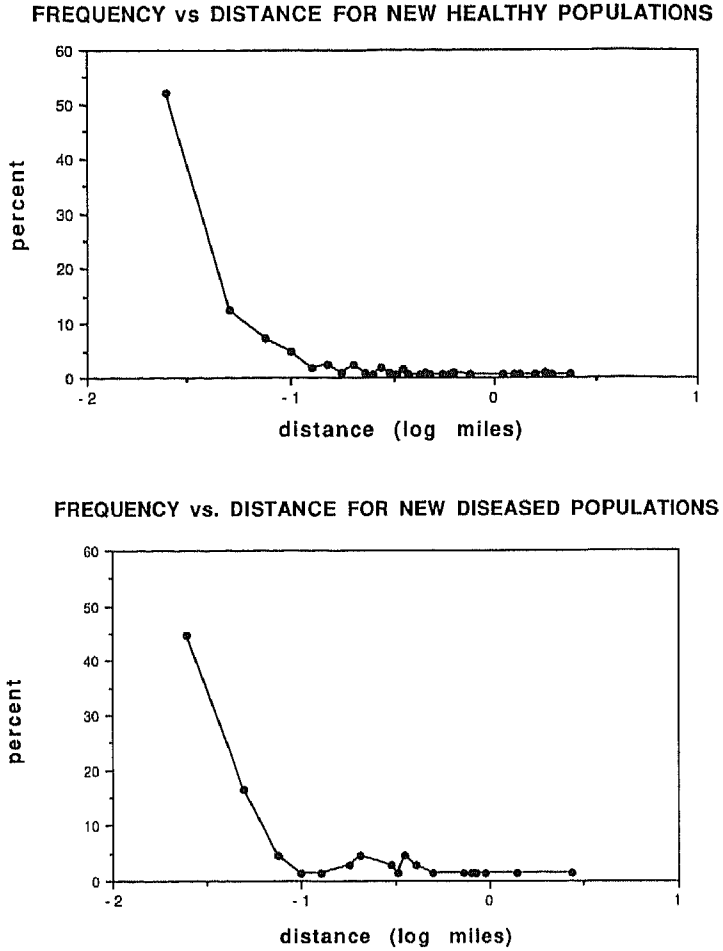

FIG. 8.6. Percentage of newly established populations that establish at a given distance from the population nearest to it in the previous year. *Top*: Host populations. *Bottom*: Pathogen populations. Combined data for 1989 and 1990. *Note*: distance measures are in log miles such that −1 = 176 yd, and 0 = 1 mile.

These results suggest density-dependent population regulation and a negative impact of the pathogen on the host population growth rate. The establishment of both the host and the disease depended on the distance from the nearest source population (fig. 8.6). While most new populations were founded close to existing ones, new populations often arose at considerable distances from preexisting ones. These data argue strongly for population interconnectedness even over substantial distances. Although we can never be absolutely sure that *S. alba* is not present in some

intermediate nonroadside population (it is impossible to look in everyone's backyard!), such long-distance establishment strongly suggests occasional long-distance dispersal of seeds and, in the case of the pathogen, spores. The mechanism of long-distance transport is unknown; because roadsides are frequently mowed and disturbed, we suspect that humans and their machines may be important long-distance vectors. In the host, it is likely that the seeds can stay viable in the soil, and "long-distance" colonization may also reflect the earlier presence of populations which still persists in the seed bank; studies are under way to examine this possibility. Long-distance spore dispersal may also be affected by particularly vagile pollinators such as hawk-moths which have been observed to visit *Silene*.

Metapopulation Dynamics: Simulation Studies

In this section we present the results of computer simulations designed to examine whether the metapopulation processes of extinction and colonization can maintain host-pathogen coexistence even in situations where in any one local population the host is driven to extinction by the pathogen (and also resulting in the extinction of the pathogen itself). The simulations follow the general protocols used by Caswell (1978). We consider an array of segments, where each segment is characterized by the presence or absence of healthy or diseased individuals. The probability of a host or pathogen arriving in any segment is independent of distance, and therefore is simply proportional to the number of hosts or pathogens summed over all segments, multiplied by a probability of per propagule establishment within a segment. Establishment within an already occupied site is ignored, and pathogens can establish in segments only where the host is already present. There is also an externally imposed likelihood of disturbance resulting in disease-independent extinction. We assume that internal dynamics are simple (disease incidence is a linear function of time since disease arrival, and host abundance is inversely proportional to disease incidence); postcolonization migrants are assumed to have no effect on local dynamics. We also assume there may be a minimum time ("finding time") before the disease can establish itself in a newly founded healthy population.

The results of this model (fig. 8.7) show that, given estimates of extinction and establishment rates based on our first two seasons of census data, host and pathogen coexistence can occur even when within-population dynamics leads to local extinction. As with other similar studies, a critical component was finding time (Murdoch 1977; May 1978; Nisbet and Gurney 1982). With no delay in pathogen arrival, host and pathogen

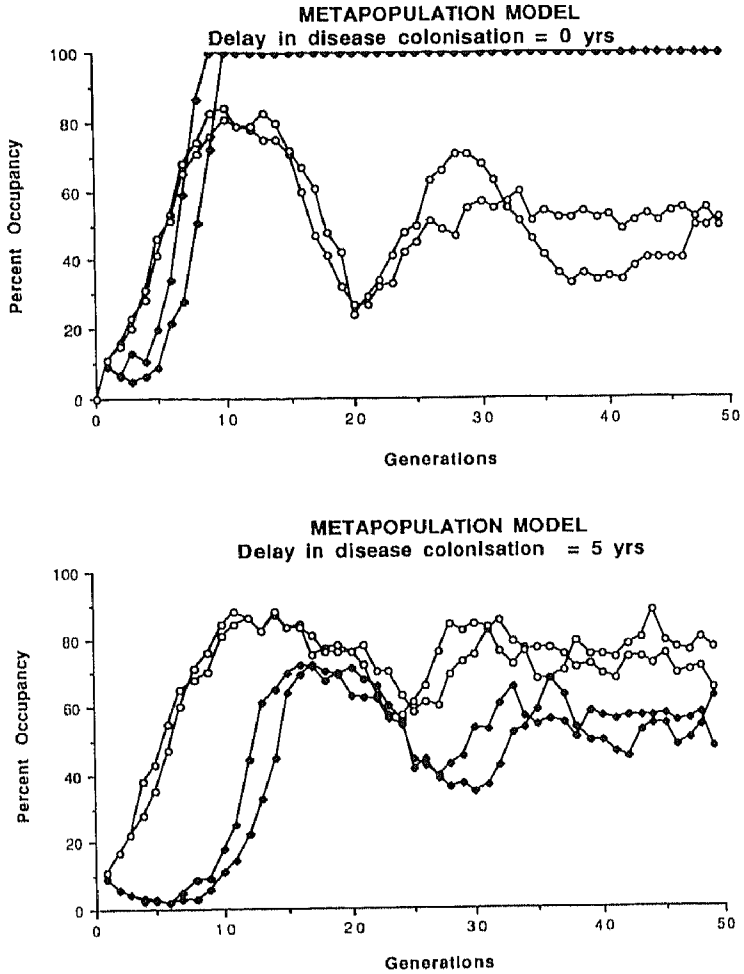

FIG. 8.7. Results from a computer simulation of metapopulation dynamics of one hundred interconnected segments. The two simulations differ in the time taken for the disease to colonize a previously established healthy population: (*top*) simultaneous colonization is possible; (*bottom*) colonization is not possible before five years. Two replicate runs are shown for each simulation. Vertical axis shows the percentage of segments occupied by populations regardless of whether they are diseased or healthy (open circles) and the percentage of these occupied segments that are diseased (solid circles). The models assume that the extinction time due to disease = 10 years; disease independent extinction rate = 0.08 per year; plant population productivity = 0.6 potential colonizations per year; pathogen population productivity = 10 potential colonizations per year.

continued to coexist within the metapopulation, but all populations became diseased (fig. 8.7, top). If there was a five-year delay in disease arrival, then only about half the populations became diseased (fig. 8.7, bottom).

The Genetic Component in Among-Population Processes

Although metapopulation models incorporating genetic processes have shown large effects of extinctions and colonizations on overall heterozygosity and genetic substructuring (Gilpin 1991; McCauley 1991; and Slatkin, chap. 1, this volume), very few have examined how genetic processes may influence numerical dynamics (for an exception, see Frank 1991). Clearly, the fate of a newly colonized host or pathogen population will depend in part on the genotypes of the initial colonists, and on how subsequent migrations augment the genetic variation present among these colonists.

Genetic variability is likely to affect metapopulation dynamics whenever new populations are founded by relatively few individuals that represent only a subsample of the global genetic diversity. This will have two major consequences: it will greatly affect the dynamics within each of the component populations, and it will generate the conditions for interdemic group selection. Within populations, the presence of only a few founders is likely to result in inbreeding because of mating among relatives. The reduction in fecundity and survival that results from inbreeding depression may contribute to the extinction of newly colonized populations through chance events, or it may have an impact on the subsequent growth rate of the population. In addition, if only a subsample of the global genetic diversity is represented in the founding population, then the evolutionary and numerical responses of the component populations may come to vary dramatically. This will be particularly important in host-pathogen systems where the genetics of the host and the pathogen can play an important role in numerical abundance (see chapter 7).

At the interpopulation level, if population turnover is high, whether by stochastic effects or because of a pathogen or predator, colonizers are likely to be from populations that show the greatest persistence and numerical abundance. This sets the stage for evolution of traits that increase group productivity or reduce variation in group size (Gilpin 1975; Wilson 1983). Therefore, assessing numerical changes caused by the interaction of ecological and evolutionary processes is critical to an evaluation of the likelihood of group selection in real-world systems.

We have many reasons to believe that the dynamics of the *Silene-Ustilago* metapopulation system may be affected by genetic processes, even

though at present we have direct evidence only of genetic variation in resistance in the host. First, it is likely that the pool of potential colonists will be affected by the history of disease in the source populations. Thus colonists from populations with a history of disease may well be resistant, while those from populations without a history of disease will be susceptible. The potential impact of this is illustrated by our results from some experimental populations that we have set up to study the long-term dynamics of the *Silene-Ustilago* system. In 1990 we established sixteen "population cages" in the vicinity of Mountain Lake Biological Station. These cages consist of fenced (to prevent deer grazing) areas in which there are pots with healthy individuals, pots with diseased individuals, and pots that have soil but no plants and into which there can be seedling recruitment. The cages were established using a range of initial frequencies of diseased and healthy plants, but more critically, they were established from the progeny of only three pairs of parents. These parents were either susceptible or resistant individuals as determined in the experiments of Alexander (1989). The first year's results from these experiments dramatically illustrate how disease spread can be influenced by initial population composition. In those populations started from the progeny of susceptible individuals, 15.2% of the flowering plants (n = 224) became diseased in the first summer; however, in those populations started from the progeny of resistant individuals, only 0.6% of the flowering plants (n = 181) became diseased.

We can further illustrate the potential impact of genetics on the numerical dynamics of local populations using computer simulations (table 8.4; see also chap. 7, this volume). For illustration, we again restrict ourselves to cases where there is genetic variation in the host but not in the pathogen. The results are rather obvious but nonetheless dramatic. First, populations with different underlying genetic structures but similar numerical equilibria may approach these equilibria in quite different ways (fig. 8.8a,b; see also chap. 7, this volume). Given the stochastic nature of colonization events, colonizers are likely to differ in their genetic composition, and this can result in totally different numerical dynamics (fig. 8.8c,d). If there are now subsequent migration events, they may alter the genetic composition of the original founding populations, and this will have a corresponding profound effect on subsequent dynamics (fig. 8.8e,f). In a genetically uniform population, rare migration events subsequent to an initial colonization would have almost no impact.

In natural systems, additional forces may come into play. For example, the level of resistance may be altered by the colonization process itself. Thus Alexander (1989) found that resistant plants produce fewer flowers than susceptible plants; susceptible individuals may therefore be over-

TABLE 8.4
Difference equation models describing local dynamics of numbers of hosts and pathogens for different levels of genetic variation in host resistance and pathogen virulence. Equations assume frequency-dependent disease transmission and density-dependent growth of healthy but not diseased class.

a. Both host and pathogen invariant

$$X_{t+1} = X_t[1 + r - (\beta Y_t)/N_t]$$
$$Y_{t+1} = Y_t[1 + (\beta X_t)/N_t - d]$$

b. Host variation in resistance; pathogen invariant

$$X_{1,t+1} = X_{1,t}[1 + r_1 - (\beta_1 Y_t)/N_t]$$
$$X_{2,t+1} = X_{2,t}[1 + r_2 - (\beta_2 Y_t)/N_t]$$
$$Y_{t+1} = Y_t[1 + (1/N_t)(\beta_1 X_{1,t} + \beta_2 X_{2,t}) - d]$$

c. Host invariant; pathogen variation for virulence

$$X_{t+1} = X_t[1 + r - (1/N_t)(\beta_1 Y_{1,t} + \beta_2 Y_{2,t})]$$
$$Y_{1,t+1} = Y_{1,t}[1 + (\beta_1 X_t)/N_t - d_1]$$
$$Y_{2,t+1} = Y_{2,t}[1 + (\beta_2 X_t)/N_t - d_2]$$

d. Host variation for resistance; pathogen variation for virulence

$$X_{1,t+1} = X_{1,t}[1 + r_1 - (1/N_t)(\beta_{11} Y_{1,t} + \beta_{12} Y_{2,t})]$$
$$X_{2,t+1} = X_{2,t}[1 + r_2 - (1/N_t)(\beta_{21} Y_{1,t} + \beta_{22} Y_{2,t})]$$
$$Y_{1,t+1} = Y_{1,t}[1 + (1/N_t)(\beta_{11} X_{1,t} + \beta_{21} X_{2,t}) - d_1]$$
$$Y_{2,t+1} = Y_{2,t}[1 + (1/N_t)(\beta_{12} X_{1,t} + \beta_{22} X_{2,t}) - d_2]$$

Explanation of symbols: $X_{i,t}$ and $Y_{i,t}$ are respectively the numbers of the ith host and pathogen type at time t; N_t is the total population ($X_t + Y_t$) at time t. The host growth rate r_i is given by the hyperbolic decay function: $b_{0,i}/(b_{1,i} N_t + 1) - d_i$, where $b_{0,i}$ is the maximum per capita reproduction of the ith host type (at $N_t = 0$), $b_{1,i}$ is a constant that determines the strength of the density dependence, and d_i is a constant death rate for the ith host (or pathogen) type. Disease is transmitted at a rate $\beta_{ij} X_{i,t} Y_{i,t}/Nt$ (i.e., proportional to the absolute density of healthy and the frequency of infective individuals), where β_{ij} is the transmission parameter for the ith host and jth pathogen.

represented in the migrant pool. It is well known from the agricultural literature that plants may show genetic variation for extremes of resistance and susceptibility to pathogens, which in turn may show variation in virulence and avirulence. Such resistance and virulence types may often show gene-for-gene interactions, where susceptibility of a particular host genotype to the disease is contingent on the presence of a particular virulence gene in the pathogen (Flor 1956; Barrett 1985; Burdon 1987). Although relatively little work has been done on natural populations, there is already strong evidence that many natural populations are highly het-

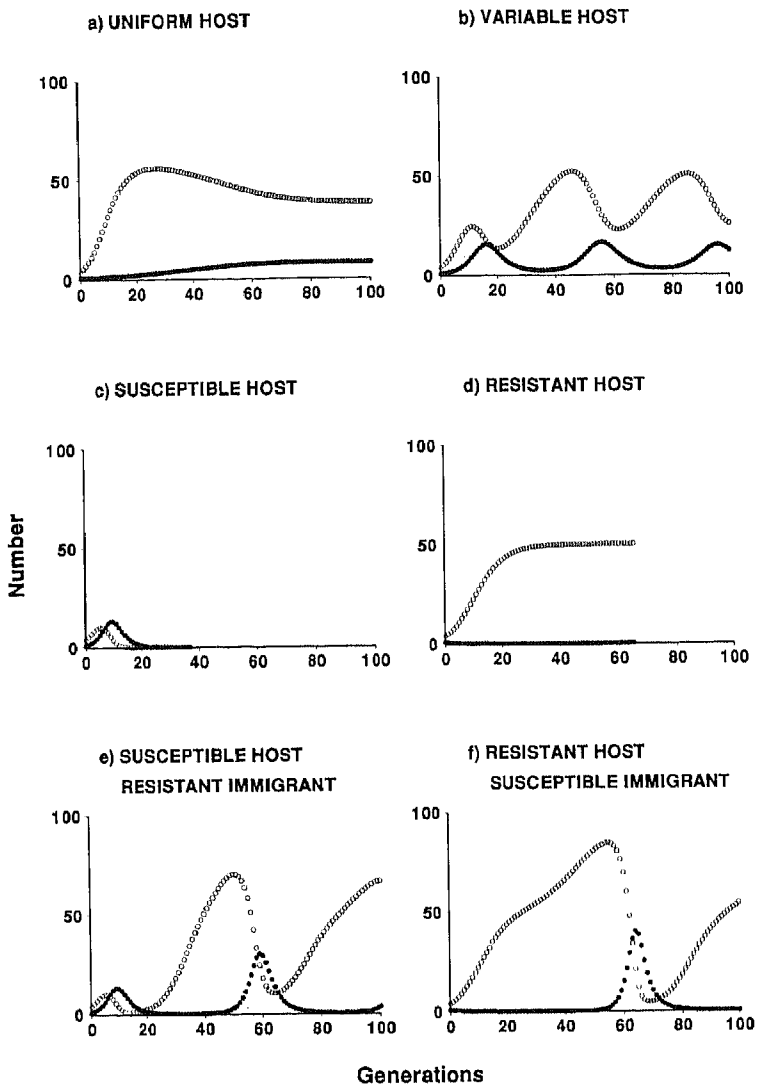

FIG. 8.8. Results from computer simulations showing how the numerical dynamics of small, newly established populations are influenced by genetic composition of the source population and of subsequent immigrants. Recursions on which the models are based are given in table 8.4b. Throughout, $d = 0.3$, $b1 = 0.02$. All populations are started with four healthy (open circles) and one diseased (solid circles) individual. (a) Uniform population of average resistance (with parameter values that result in the same numbers of healthy and diseased individuals at equilibrium as the variable population). (b) Variable population started with two susceptible ($\beta_1 = 1.0$, $b0_1 = 0.9$), and two resistant ($\beta_2 = 0.1$, $b0_2 = 0.6$) individuals. (c) Uniform population started with all susceptible individuals. (d) Uniform population started with all resistant individuals. (e) An initially uniform susceptible population into which there is, at generation 15, a single resistant migrant. (f) An initially resistant population into which there is, at generation 15, a single susceptible migrant.

erogeneous in their resistance/virulence structure. In the *Amphicarpaea bracteata–Synchytrium decipiens* system (Parker 1985), pathogen isolates were virulent on plants from the site where the pathogen was collected but largely avirulent on plants from other sites. In the *Linum marginale–Melampsora lini* system (Jarosz and Burdon 1991), local semi-isolated populations were extremely heterogeneous in their resistance and virulence structure, with no clear relationship between the virulence of particular fungal isolates and the resistance of the host plants from those same populations. However, on a broader geographical scale, all host-resistance types were matched by a corresponding pathogen-virulence type. Whether or not there is local host specialization may depend on the interconnectedness among the component populations.

We might expect that in a metapopulation context, increasing genetic complexity (measured in terms of the number of "matching" resistance/virulence alleles) could lead to less overall disease incidence. Disease spread would require that a pathogen virulence genotype finds a "matching" susceptible host, something that may be particularly difficult if there is a high rate of population extinction/colonization. If this expectation is borne out, the genetic complexity of host-pathogen systems may provide an explanation for the low disease prevalence seen in many natural host-pathogen systems. Somewhat paradoxically therefore, host-pathogen coevolution may lead to lower disease levels at the metapopulation level.

From a genetic perspective, it has also been shown that complex genetic determination can lead to severe host-pathogen cycles within populations (Seger 1988; see chapter 7, this volume) such that chance extinction of genetic variants is a likely possibility. However, such cycles may be stabilized by migration among populations (Seger 1988; Frank 1991). A metapopulation structure may therefore be important in maintaining genetic variation. Using a computer simulation, Frank (1991) concluded that spatial substructuring of coevolutionary systems can maintain high levels of polymorphism among environmentally identical patches even with high migration rates.

Our eventual goal is to explicitly incorporate genetic variation in host resistance and pathogen virulence into metapopulation models and to ask how it affects the numerical dynamics. We will then be able to ask whether, for example, there are explanations for some of the unusual aspects of our long-term census data. For example, our census data have produced consistent "incidence patterns": small populations are less likely to have disease, but when they are diseased, they have a higher proportion of disease than large populations. We can ask what combination of within- and among-population processes produce such patterns. Specific incidence functions have been predicted for single-species meta-

populations (Taylor, B. 1991), but we do not know what to expect in coevolutionary systems. Patterns emerging from our models that are reflected in the empirical data may therefore suggest processes that are important in nature. The simulations will also enable us to ask if some critical initial disease frequency (or genotype frequency) is necessary before a disease is likely to spread. The extreme patchiness in pathogen distribution may result from single colonists being ineffective at establishing the disease. Instead there could be a wave of advance from adjacent populations that might initially have achieved high disease levels purely by chance. The real-world importance of metapopulation processes in disease dynamics has been emphasized recently in a model of the spread of AIDS in situations where populations are organized into villages (May and Anderson 1990). An increase in the interconnectedness of village populations resulted in an initial short-term decrease in overall disease incidence, followed by a subsequent shift of the disease from initially low equilibrium endemic levels to epidemic status.

Conclusions

Much of metapopulation dynamics theory has assumed that within-population dynamics is rapid relative to rates of population extinction and colonization, and that the dynamics of these systems can be represented by presence or absence in patches characterized by singular colonization and extinction probabilities. Our results show that population extinction and colonization rates in the *Silene-Ustilago* system are very rapid, whereas theoretical models indicate that approach to equilibrium within populations may be quite slow (Alexander and Antonovics 1988; Thrall, Biere, and Uyenoyama 1993; and chapter 7, this volume). In such circumstances, which may be quite general for host-pathogen and other coevolutionary systems, it is necessary to understand how within-population processes impact on metapopulation behavior. The issue becomes even more critical when, as in the example presented here, the trajectories of the within population dynamics may be greatly affected by the genetic composition of the initial colonizers and the subsequent migrants. We clearly have to entertain the idea that population interconnectedness may have a large effect on local and metapopulation dynamics and that local populations may be rarely in equilibrium, either genetically or ecologically. However, we hope this study indicates that the expectation of local "non-equilibrium" is not a cause for pessimism, but instead presents a fascinating challenge for an ecological geneticist willing to drive around the countryside.

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9

Ecological Genetics of Life-History Traits: Variation and Its Evolutionary Significance

Introduction

A major goal of evolutionary biology has been the development of a general theory that will allow the accurate prediction of those life-history traits most likely to evolve in different ecological settings. This goal is motivated by two sets of observations. First, within any well-defined phylogenetic group, life-history traits vary nonrandomly with respect to ecological setting (Cody 1966; Pianka 1970; Baker 1972; Brewer and Swander 1977; May and Rubenstein 1985; Primack 1985; Dunham et al. 1988; Wilbur and Morin 1988). For example, actively foraging lizard species have smaller clutch sizes than sit-and-wait foragers (Dunham et al. 1988), and species of buttercup (*Ranunculus*) that occur in drier habitats have larger seeds (Baker 1972). Second, specific patterns of covariation among life-history traits, like the inverse relation between offspring size and number, recur in many groups (Primack 1987; Rohwer 1988; Elgar and Heaphy 1989; Mazer 1989; Mitton and Lewis 1989; Read and Harvey 1989; Reznick and Miles 1989; Elgar 1990), which suggests that there must be general rules for the organization of life-history variation.

Mathematical models dedicated to understanding the genesis and maintenance of these patterns examine how different rates of age- or stage-specific mortality select for different optimal schedules of reproductive timing, investment, and packaging. The problem is made tractable by adding a set of a priori constraints on the permissible schedules. This approach to the problem is motivated by another common pattern that emerges from broad taxonomic surveys: some measure of average stage-specific mortality rate is always found to be correlated with the average value of a life-history trait like clutch size or the age at first reproduction (Millar and Zammuto 1983; Saether 1988; Charnov 1991). Any particular model can be connected to a particular association of life-history variation and ecological variation (e.g., active vs. sit-and-wait foraging in lizards or moisture regime for buttercups) by examining how the ecologi-

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Contents

List of Contributors vii

Introduction: Current Directions in Ecological Genetics
by Leslie A. Real ix

Montgomery Slatkin

1. Gene Flow and Population Structure 3
2. Cladistic Analysis of DNA Sequence Data from Subdivided Populations 18

Sara Via

3. The Evolution of Phenotypic Plasticity: What Do We Really Know? 35
4. Population Structure and Local Adaptation in a Clonal Herbivore 58

Michael Lynch

5. Neutral Models of Phenotypic Evolution 86
6. Evolutionary Genetics of *Daphnia* (*with Ken Spitze*) 109

Janis Antonovics

7. The Interplay of Numerical and Gene-Frequency Dynamics in Host-Pathogen Systems 129
8. Ecological Genetics of Metapopulations: The *Silene-Ustilago* Plant-Pathogen System (*with Peter Thrall, Andrew Jarosz, and Don Stratton*) 146

Joseph Travis

9. Ecological Genetics of Life-History Traits: Variation and Its Evolutionary Significance 171
10. Evolution in the Sailfin Molly: The Interplay of Life-History Variation and Sexual Selection 205

Index 233