2. THE EVOLUTION OF SPECIES' NICHES: A POPULATION DYNAMIC PERSPECTIVE

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2.1 INTRODUCTION

A full understanding of evolution requires one to consider the absence of evolutionary change as well as its presence. A surprising feature of the history of life is that populations exposed to novel environments often seem to fail to adapt to, or even persist in, those environments. For instance, Bradsaw (1991) has noted that although many plant species have evolved resistance to herbicides, many others have failed to evolve resistance despite repeated exposure. Such evolutionary "failures" span short and long time scales (Holt and Gaines 1992). There are many possible explanations for evolutionary conservatism. In this chapter we present one class of explanations, emphasizing how population dynamics can constrain species' evolutionary responses to novel environments.

We will examine the evolution of a species' "fundamental niche," which is intimately tied to population persistence and extinction. If N_t denotes population size at time t, a population goes extinct if $N_t \rightarrow 0$ with increasing t. The basic model for population growth in a closed, discrete-generation population is $N_{t+1} = \lambda N_t$, where λ is the finite rate of increase per generation. A population deterministically goes toward extinction if, at low densities, $\lambda < 1$. A crisp definition of a species' niche is thus: all sets of conditions, resources, etc. for which $\lambda > 1$ (Figure 2.1). A given habitat is within a species' niche if $\lambda > 1$. Conversely, if $\lambda < 1$, the habitat is outside the species' niche, and any population found there deterministically reaches densities where, in the real world, it would face inevitable extinction due to "demographic stochasticity" (i.e., chance demographic events in small populations; see, e.g., Renshaw (1991).) We refer to populations inside their species' fundamental niche (i.e., $\lambda > 1$) as "source populations" and those outside the niche ($\lambda < 1$) as "sink populations." In this terminology, niche evolution occurs when a population evolves such that a sink environment becomes a source environment. Our primary interest is in determining the circumstances in which populations will evolve sufficiently to permit persistence in an initially unfavorable environment.

A species' fundamental niche can evolve either as a correlated evolutionary response of populations that occupy source environments ("indirect

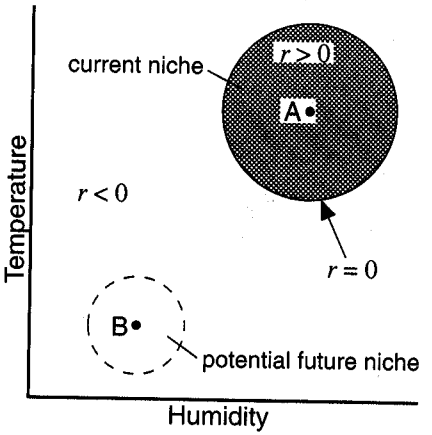

Figure 2.1. A hypothetical species' niche. The shaded region indicates combinations of temperature and humidity conditions within which population size can increase ($\lambda > 1$). Environmental conditions in habitat A permit a population to persist deterministically. By contrast, in habitat B, the same population becomes extinct — unless the niche itself evolves.

niche evolution") or through the evolution of populations directly exposed to sink environments ("direct niche evolution"). In this chapter we analyze four circumstances with the potential for direct niche evolution: (1) an isolated sink population, (2) a sink population maintained by recurrent immigration from a source population, (3) coupled sink and source populations, and (4) a network of source and sink populations ("metapopulation").

The material presented here focuses on our own past and present research. Important contributions to understanding niche evolution are also being made by other investigators (Pease et al. 1989; Brown and Pavlovic 1992; Lynch and Lande 1993; Burger and Lynch 1995; Kawecki 1995).

2.2 NATURAL SELECTION AND EXTINCTION IN A CLOSED ENVIRONMENT: DETERMINISTIC APPROACHES

We begin with a population that has suddenly encountered a novel environment outside its fundamental niche. This could characterize a colonizing group that encounters an inhospitable habitat, or a closed population experiencing abrupt environmental change. In either situation, the initial population occupies a sink habitat. When will such a population evolve into a source, thereby expanding its species' niche?

Consider an isolated sink population with discrete generations. We assume the population's finite rate of increase at time t is identical to the mean fitness of its members, $\bar{W}_t$. Population size thus changes according to

$$N_{t+1} = \bar{W}_t N_t. \tag{2.1}$$

Since the population is initially a sink, $\bar{W}_0 < 1$. If the finite rate of increase

does not change, then

$$N_t = \bar{W}_0^t N_0 \tag{2.2}$$

where N_0 is initial population size. Although the population deterministically tends toward extinction, $N_t \neq 0$ for any finite t. A more complete treatment that included demographic stochasticity (Renshaw 1991) would show that extinction becomes increasingly certain as the population declines to small sizes. For now, we will bypass the complexities of demographic stochasticity entirely by using a crude deterministic approximation for its effects. Specifically, we assume there is some "critical density" N_c below which a population is highly vulnerable to rapid extinction. This approximation can be justified on the grounds that the probability of rapid extinction often rises dramatically as population size declines (MacArthur and Wilson 1967). (The results in the next section offer a more direct justification.) One can thus approximate the time until extinction, t_E, by solving $N_c = N_0 \bar{W}_0^t$ for t: $t_E = (\ln N_c - \ln N_0)/\ln \bar{W}_0$ generations until extinction.

Without evolution, extinction is inevitable. However, a population may be able to avoid extinction if it can adapt sufficiently rapidly in the sink environment. This follows from Fisher's fundamental theorem of natural selection which suggests, roughly, that given genetic variation in fitness, mean fitness, $\bar{W}_t$, should increase through evolution by natural selection (Fisher 1958; Burt 1995). Consider the following very simple model of this adaptive process: assume mean fitness increases by a fixed amount δ each generation, i.e., $\bar{W}_{t+1} = \bar{W}_t + \delta$. Then t generations after a population first encounters the sink environment, $\bar{W}_t = \bar{W}_0 + t\delta$. Population density will increase whenever mean fitness exceeds one. Thus, the first time at which a population grows, t_R, can be found by solving $1 = \bar{W}_t = \bar{W}_0 + t\delta$ for t: $t_R = (1 - \bar{W}_0)/\delta$. Adaptation will "rescue" a population from extinction if $t_R < t_E$, whereas extinction is likely, even with adaptive evolution, if $t_R > t_E$.

This simple model gives us a sense of the important time scales involved in the race between evolution and extinction, but it is much too simplistic because mean fitness does not generally increase at a constant rate per generation. We now consider a more realistic evolutionary model that more accurately describes changes in a population's mean fitness under selection, viz., a quantitative genetics model.

This model makes the following basic assumptions. First, we assume individual fitness depends on a trait, z, with polygenic autosomal inheritance (e.g., body size). We assume fitness in the novel environment has the form $W(z) = W_{\max} \exp\left[-z^2/2\omega\right]$, where ω is a parameter inversely related to the strength of selection, so that (without loss of generality) the optimum phenotype lies at $z = 0$ with fitness $W_{\max}$. This "Gaussian" form can represent a variety of biological situations, including directional and stabilizing selection (it is also mathematically convenient). The distribution of phenotypes in generation t, $p_t(z)$, is assumed normal with mean d_t (the distance of the mean phenotype from the local optimum, $z = 0$) and variance P:

$$p_t(z) = (2\pi P)^{-1/2} \exp\left[-(z - d_t)^2/2P\right].$$

Quantitative traits are often normally distributed when measured on an appropriate scale (Falconer 1989). By completing the square in the exponent and

simplifying, it can be shown that the mean fitness in generation t is

$$\bar{W}_t = \int W(z)p_t(z)\,dz = \hat{W}\exp\left[\frac{-d_t^2}{2(P+\omega)}\right] \tag{2.3}$$

where $\hat{W} = W_{\max}\sqrt{\omega/(P+\omega)}$ is the population growth rate when the mean phenotype is at the local optimum ($d = 0$). Finally, we assume the effects of other evolutionary forces (e.g., drift) are negligible.

A standard result from quantitative genetics (Falconer 1989; Lande 1976) predicts that the mean phenotype, d_t, of a quantitative trait z changes between generations (i.e., evolves) according to the equation

$$\Delta d_t \stackrel{\text{def}}{=} d_{t+1} - d_t = h^2 s, \tag{2.4}$$

where h^2 is the "heritability" of z and s is the "selection differential." Roughly, h^2 measures the degree to which offspring phenotypes resemble their parents' phenotypes, in the absence of common environmental influences. The selection differential is the difference between the mean phenotype of individuals selected to be parents and the mean phenotype before selection. For our model,

$$s \stackrel{\text{def}}{=} \int z\left[W(z)/\bar{W}_t\right]p_t(z)\,dz - d_t = \frac{-d_t P}{P+\omega} \tag{2.5}$$

which implies that (2.4) has the form

$$\Delta d_t = -h^2\frac{d_t P}{P+\omega}. \tag{2.6}$$

Therefore,

$$d_{t+1} = d_t + \Delta d_t = \frac{\omega + (1-h^2)P}{P+\omega}\,d_t = kd_t \tag{2.7}$$

where $k = [\omega + (1-h^2)P]/(P+\omega)$ is the "evolutionary inertia" of the mean; $0 \le k \le 1$. Note that k will be near 1 if heritability is low ($h^2 \approx 0$) or selection is weak ($\omega \gg P$). If P and h^2 are constant, then

$$d_t = k^t d_0. \tag{2.8}$$

We now turn to the dynamics of population density, which are described once again by (2.1). Because $\bar{W}$ changes through time, $N_t = N_0\prod_{i=0}^{t-1}\bar{W}_i$. Recall that our goal is to determine the times t_R and t_E. To do this, we try to write $N_0\prod_{i=0}^{t-1}\bar{W}_i$ as an explicit function of t. Substituting (2.3) for $\bar{W}_t$ into (2.8) results in an expression with the geometric series $\sum_{i=0}^{t-1}k^{2i}$ in the exponential term. This series can be rewritten in the closed form $(1-k^{2t})/(1-k^2)$, giving

$$N_t = N_0\hat{W}^t\exp\left[\frac{-d_0^2(1-k^{2t})}{2(P+\omega)(1-k^2)}\right]. \tag{2.9}$$

We now use (2.9) to compute t_E, the time to reach N_c, and t_R, the first time at which $\bar{W} \geq 1$. By definition, t_E must satisfy

$$N_c = N_0 \hat{W}^{t_E} \exp\left[-\frac{1}{2}\left(\frac{d_0^2}{P+\omega}\right)\left(\frac{1-k^{2t_E}}{1-k}\right)\right]. \tag{2.10}$$

While it is not possible to solve for t_E explicitly, (2.10) provides an *implicit* definition for t_E as a function of the parameters. Two parameters can be eliminated by defining $\nu_0 = N_c/N_0$ and $\beta_0 = d_0^2/(P+\omega)$. These give "natural" scales for measuring, respectively, the relevant initial population density and distance from the optimum phenotype. This rescaling reduces (2.10) to

$$\nu_0 = \hat{W}^{t_E} \exp\left[-\frac{\beta_0}{2}\left(\frac{1-k^{2t_E}}{1-k}\right)\right]. \tag{2.11}$$

The time t_R must be a solution of the equation $\bar{W}_t = 1$. Using (2.3) and (2.8) leads to

$$t_R = \frac{\ln\left(\ln \hat{W}\right) - \ln(\beta_0/2)}{2\ln k}. \tag{2.12}$$

With expressions for t_E and t_R in hand, we can examine in detail the influence of adaptive evolution on the chances of population persistence.

We begin by using (2.11) and (2.12) to determine whether an adapting population is likely to persist (because $t_E > t_R$) or risk rapid extinction ($t_E < t_R$). We are especially interested in the respective influences of initial population size, N_0, and the magnitude of d_0, which indicates the initial degree to which the population is maladapted to the novel environment. We want to know the set of N_0 and d_0 at which $t_E = t_R$ separates the combinations of initial values consistent with likely persistence ($t_E > t_R$) versus extinction ($t_E < t_R$). We determined these critical values by substituting (2.12) into (2.11). This eliminates t, resulting in an equation that defines the relationship between ν_0 (the scaled form of N_0) and β_0 (the scaled version of d_0) at which $t_E = t_R$. Graphs of this relationship, for various values of heritability, h^2, are shown in Figure 2.2. Populations whose initial density and degree of maladaptation lie below the curve of critical values for a particular h^2 are likely to persist, whereas populations with combinations falling above the critical curve have a high risk of rapid extinction.

While evolution will not rescue all initially maladapted populations from extinction, evolution might slow the decline of populations destined to reach critically low densities. To assess this potential effect, we compared the times t_E to reach N_c with evolution (2.11) to the times to reach N_c without evolution:

$$t_E = \frac{\ln N_c - \ln N_0}{\ln \bar{W}_0} = \frac{\ln \nu_0}{\ln \bar{W}_0} \tag{2.13}$$

Some of the results are shown in Figure 2.3. Evolving populations with initial degree of maladaptation less than a critical level (β^* in Figure 2.3) never drop below N_c. This figure also shows that, in contrast to our expectations, evolution does little to slow the decline of populations destined to reach critically

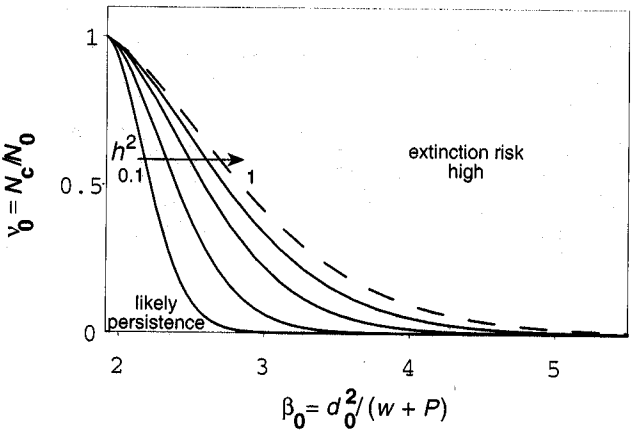

Figure 2.2. Combinations of scaled initial population densities (v_0) and degrees of initial maladaptation (β_0) leading to likely persistence or high extinction-risk heritability, $h^2 = 0.1$ (solid curve) and $h^2 = 1$ (dashed curve). For a given level of heritability, populations with v_0 and β_0 below the curve persist deterministically because they remain above N_c; those with v_0 and β_0 above the curve decline below N_c and become highly vulnerable to rapid extinction by stochasticity. (Adapted from Gomulkiewicz and Holt (1995).)

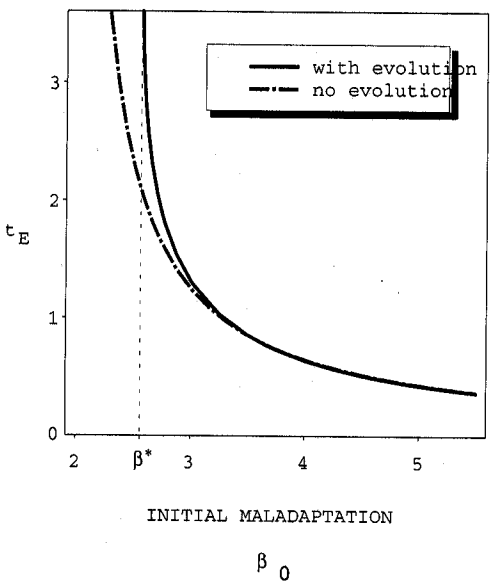

Figure 2.3. The time t_E a population will first reach N_c as a function of its initial degree of maladaptation (2.11). The dot-dashed curve indicates analogous times for the case of no evolution (2.13).

low densities. Apparently, the main effect of evolution is to prevent extinc-

tion altogether, rather than slow the approach of populations that are destined to reach critically low densities and face rapid extinction risks.

While a population whose size drops below N_c faces a high risk of extinction, it may nevertheless avoid this fate. Such a population would continue to adapt and, at time t_R, begin to grow. Provided the population persists, at some later time, t_P, its size will climb above N_c. Thereafter, the population will be relatively invulnerable to extinction by demographic stochasticity. The time t_p needed for a population to grow above N_c is, like t_E, a solution of (2.11), because $N_{t_P} = N_c$. In fact, (2.11) has either zero or two roots for (essentially) every pair of initial conditions. When there are two roots, the lower one is t_E while the upper root is t_P. The difference, $t_P - t_E$, defines the maximum period that a population's density will be below N_c. It can be shown (Gomulkiewicz and Holt 1995) that this "period of extinction risk" is longer for populations that are initially more highly maladapted. Equations (2.11) and (2.12) can also be used to explore the dependence of t_E, t_R, and t_P on initial density, N_0. Initially small populations are similarly likely to face a period of extinction risk.

The above results lead to four main biological conclusions. First, only initially large and mildly maladapted populations are expected to evolve sufficiently to persist in novel environments, while small or severely maladapted populations are likely to face a high risk of rapid extinction. The range of initial population sizes consistent with likely persistence shrinks rapidly for intermediate degrees of initial maladaptation (Figure 2.2). Second, populations that are more severely maladapted (or initially small) will face a high extinction risk sooner and, if they manage to avoid chance extinction, remain at high risk for a longer period of time. Third, the main effect of local adaptation in a sink environment is to allow some populations to avoid facing high extinction risk altogether. Evolution by natural selection does little to slow the approach of populations destined to reach critically low densities (Figure 2.3). Finally, if niche evolution occurs primarily through isolated colonizations of novel environments outside a species' current fundamental niche (or through populations exposed to rapid environmental deterioration), then niche expansion will occur only rarely, unless colonizing groups tend to be very large.

The above models permit extensive analysis. An important task for future work will be to examine the robustness of our conclusions to relaxation of genetic and ecological assumptions. (We have elsewhere considered a standard one-locus diallelic model of a continuously breeding population and reached thematically similar conclusions (Gomulkiewicz and Holt 1995)). In the following sections, we first examine stochasticity, then permit recurrent immigration into the sink, and then emigration back to the source.

2.3 NATURAL SELECTION AND EXTINCTION IN A CLOSED ENVIRONMENT: STOCHASTIC APPROACHES

In the last section we considered the dynamics of a closed population that is suddenly exposed to an environment outside its species' niche. We found that the main issue is whether the population can evolve sufficiently rapidly to avoid reaching low densities where it is highly vulnerable to chance

extinction. Our deterministic treatment of evolution and extinction in a closed environment rested on a crude (but mathematically convenient) device, the notion of a critical density N_c, to avoid dealing with the complicated probabilistic details of the actual extinction process.

Besides validating our deterministic approach, a fully stochastic analysis can address issues that a deterministic analysis cannot. For example, deterministic analyses do not provide a probability distribution of actual times to extinction which may be important in applications. However, stochastic models are typically difficult to analyze, except for certain simple cases, and usually require approximations or computer simulations. In this section we present a stochastic model of evolution and extinction that is sufficiently simple to allow mathematical analysis.

The simplest version of our basic ecological scenario involves a population of clonal organisms facing a novel environment. As it turns out, a substantial body of empirical work involves such systems. The experiments performed by Lenski, Bennett and their colleagues (e.g., Lenski and Bennett (1993)) on the evolutionary responses of *E. coli* to novel thermal and nutritional environments is one prominent example. Our mathematical model assumes that reproduction is asexual and generations do not overlap. Suppose that the population contains two genotypes, A_1 and A_2. In the novel environment, individuals with genotype A_1 have an expected absolute fitness $W_1 > 1$ while those with genotype A_2 have expected absolute fitness $W_2 < 1$. The *actual* number of offspring left by an individual is a random variable. Finally, resources and space are sufficient to allow individuals to survive and reproduce independently of one another.

Our analysis has two goals. First, we will determine how the probability of extinction depends on the size and composition of the initial population, particularly when the "adapted" clone A_1 is initially rare. Second, we want to examine how initial population size and composition affect the probability distribution of times to extinction. Besides providing useful quantitative information, these analyses will allow us to compare the results of our previous deterministic analyses with a full-blown (albeit simple) stochastic model and thereby begin to assess the adequacy of our deterministic approximations.

Our asexual model is an example of a "two-type branching process" (Karlin and Taylor 1975). The standard way to analyze branching processes is through the use of a *probability generating function*. Probability generating functions are useful for studying stochastic processes because they conveniently "package" critical information about the process.[1] In particular, it is straightforward to compute extinction probabilities and times once an appropriate probability generating function is available. We thus begin by defining and computing a generating function for our asexual model.

It is easiest to construct the probability generating function for our two-type branching process by combining two separate generating functions, one for each of the two genotypes. The generating function for genotype A_1,

[1] To quote Lin and Segel (1988), pg. 76: "It is not difficult to follow manipulations using the generating function, but it is amazing that anyone would have thought of this device. Amazement is lessened upon learning that the first person to make use of the generating function was the genius Euler .... In discussing the motivation for using a generating function, Polya ... states that 'a generating function is a device somewhat similar to a bag. Instead of carrying many little objects detachedly (the individual coefficients), which could be embarrassing, we put them all in a bag (a generating function), and then we have only one object to carry, the bag.' "

$f(\cdot)$, is defined as

$$f(u) = \sum_{i=0}^{\infty} p_i u^i \tag{2.14}$$

where p_i is the probability that an individual with genotype A_1 produces i offspring ($i = 0, 1, \ldots$). Similarly, the generating function for genotype A_2 is defined as

$$g(v) = \sum_{i=0}^{\infty} q_i v^i \tag{2.15}$$

where q_i is the probability that an individual with genotype A_2 produces i offspring ($i = 0, \ldots$). Variables u and v are "dummy variables"; they serve as nothing more than placeholders during computations.

To make further progress, we must make specific assumptions about the offspring-number distributions, $\{p_i\}$ and $\{q_i\}$. We assume that the probability distribution of an individual's offspring number follows a Poisson distribution whose expected value depends on genotype. Assume that the offspring number of an A_1 individual is a Poisson random variable with expectation $W_1 > 1$, and the offspring number of an A_2 individual is a Poisson random variable with expectation $W_2 < 1$: $p_i = e^{-W_1} W_1^i / i!$ and $q_i = e^{-W_2} W_2^i / i!$. Not only are these assumptions mathematically convenient, they conform to the offspring distribution assumed in a frequently used model of genetic drift (Crow and Kimura 1970). With these assumptions, (2.14) and (2.15) simplify (using the result $e^{ax} = \sum_{i=0}^{\infty} (ax)^i / i!$) to

$$f(u) = \sum_{i=0}^{\infty} \frac{e^{-W_1} W_1^i u^i}{i!} = e^{W_1(u-1)} \tag{2.16}$$

and

$$g(v) = \sum_{i=0}^{\infty} \frac{e^{-W_2} W_2^i v^i}{i!} = e^{W_2(v-1)}. \tag{2.17}$$

We now use $f(u)$ and $g(v)$ to define $h_t(u, v)$, the probability generating function in generation t for an asexual population that initially consists of x_0 clones with genotype A_1 and y_0 clones with genotype A_2:

$$h_0(u, v) = u^{x_0} v^{y_0} \tag{2.18}$$

and

$$h_t(u, v) = h_{t-1}(f(u), g(v)) \qquad \text{for } t = 1, 2, \ldots . \tag{2.19}$$

For example, $h_1(u, v) = [f(u)]^{x_0}[g(v)]^{y_0}$, $h_2(u, v) = [f(f(u))]^{x_0}[g(g(v))]^{y_0}$, etc. In most cases (including the present one), it is difficult to obtain a closed-form expression for $h_t(u, v)$ for any given t. We overcame this difficulty by using a symbolic manipulation program to compute the generating functions for different generations.

The generating function $h_t(u, v)$ can now be used to determine critical information about the stochastic process associated with our model. In fact, the joint probability that there are k copies of genotype A_1 and l of genotype A_2 in generation t is exactly the coefficient $c_{kl,t}$ of the product $u^k v^l$ in the bivariate power-series expansion of $h_t(u, v)$:

$$h_t(u, v) = \sum_{i=0}^{\infty} \sum_{j=0}^{\infty} c_{ij,t} u^i v^j \tag{2.20}$$

where

$$c_{kj,t} = \left. \frac{\partial^k \partial^l h_t(u, v)}{\partial u^k \partial v^l} \right|_{u=v=0} \tag{2.21}$$

(Karlin and Taylor 1975). In particular, the probability of extinction (i.e., of $l = k = 0$) at or before generation t, denoted F_t, is

$$F_t \equiv c_{00,t} = h_t(0, 0). \tag{2.22}$$

Note that F_t is the *cumulative* probability distribution function for the time to extinction.

With our simple model, it is possible that a population will never go extinct. The exact probability that this occurs is $F_\infty = \pi_1^{x_0} \pi_2^{y_0}$, where π_1 and π_2 are the respective unique non-zero solutions less than or equal to one of $\pi = f(\pi) = e^{W_1(\pi-1)}$ and $\pi = g(\pi) = e^{W_2(\pi-1)}$. It can be shown that $W_2 < 1$ implies that $\pi_2 = 1$ (clone A_2 goes extinct with probability 1), so

$$F_\infty = \pi_1^{x_0}. \tag{2.23}$$

(See Karlin and Taylor (1975), Chapter 8, for details.) We can use the above definitions and results to address our main questions about how adaptive evolution affects the probability and timing of extinction of a population facing a novel environment, given both demographic stochasticity and genetic drift. First, consider how initial population size and the initial degree of population maladaptation affect the likelihood of extinction. If there are initially x_0 individuals with genotype A_1 and y_0 with genotype A_2, then the initial population size is $N_0 = x_0 + y_0$. We let the initial frequency of the maladaptive A_2 genotype, $q_0 \equiv y_0/N_0$, indicate the initial degree of population maladaptation. It is not hard to show that, for a given q_0, the probability of extinction at or before generation t decreases with N_0, whereas, given N_0, this probability increases with q_0. The minimum N_0 that is consistent with at least a 95% chance of extinction by generation 100 varies with q_0 in a qualitatively similar manner to the deterministic results shown in Figure 2.2. This suggests that an appropriate interpretation of "likely to face a high risk of rapid extinction" in our deterministic treatment is that there is at least a P% chance of extinction by generation T, for specified P and T.

We are also interested in how initial population size and composition affect the time a population persists. We can use our generating function methods to determine E_k, which is the smallest time t such that $F_t \geq k$. The graph of $E_{0.05}$ versus q_0 for a fixed initial population size closely resembles Figure 2.3,

which indicates how the "time until high extinction risk" in our deterministic treatment depends on the initial degree of maladaptation for fixed N_0. (Similar comparisons hold when the initial degree of maladaptation is fixed, rather than N_0.)

While these results support the qualitative results of our deterministic analyses, this stochastic model can provide quantitative and further qualitative information about the extinction process that our deterministic approach cannot. For example, using the cumulative distribution function F_t, it can be shown that the most rapid increase in extinction probability occurs in the first few generations. This suggests that if a population is destined to become extinct, it will likely do so quickly. Median times to extinction can also be easily computed using F_t. In fact, $M = E_{0.5}$. It can be shown that M increases rapidly with N_0, as one might expect.

Our analysis of this simple stochastic model yields two main conclusions. First, the qualitative features of the race between extinction and adaptation suggested by our deterministic analyses are supported. Second, our analysis found that populations tend to go extinct quickly, if they go extinct at all.

Our simple, asexual model could be extended in many ways, but in most cases, analysis will be sufficiently difficult to require Monte Carlo simulation methods. We have analyzed a one-locus, two-allele version of the above scenario in this way and found that the above results hold for this more complex model. However, much more work needs to be done before we will be fully convinced that the deterministic results are robust to stochasticity.

2.4 THE INFLUENCE OF IMMIGRATION ON LOCAL ADAPTATION: FRESH PERSPECTIVES ON AN OLD PROBLEM

In Sections 2.2 and 2.3 we considered a completely isolated population that finds itself outside its species' niche. How is niche evolution affected given recurrent immigration from a source? In this section, we consider a simple case we call a "black-hole sink," a sink population that recurrently receives locally maladapted immigrants that arrive from a separate source population but returns no emigrants to the source (Holt and Gomulkiewicz 1996).

A black-hole sink closely resembles the "island-continent model" used in population genetics theory to understand how one-way recurrent gene flow from a "continent" can impact local adaptation on an "island." Before presenting our model, it is instructive to consider the intuition provided by population genetics theory. Generally speaking, analysis of an island-continent model results in a "rule of thumb": for a given selective advantage of a locally favored allele, there is some rate of gene flow below which that allele will spread when rare (e.g., Nagylaki (1977), p. 125). This implies that the greatest scope for local adaptation should occur at low to zero rates of gene flow. Now suppose that the island population is a "sink" population. Without immigration, such a population goes extinct deterministically. This presents a paradox: At zero immigration — which provides the greatest scope for local adaptation — a sink population goes extinct and, thus, local adaptation is impossible! How

can this be explained? To answer this, one must examine more explicitly the demographic consequences of immigration.

Consider, as in the last section, a discrete breeding asexual population with two genotypes, A_1 and A_2, where the absolute fitness on the island of genotype A_i is W_i. Assume that A_1 individuals have the higher local fitness, or $W_1 > W_2$. The mean fitness (and finite growth rate) of the island population is $\bar{W} = pW_1 + (1-p)W_2$, where p is the frequency of A_1. Immigrants arrive on the island just after reproduction at a rate m per generation, where m is the percentage of the post immigration island population that consists of immigrants (i.e., the rate of "gene flow"). We assume all immigrants have the locally less fit genotype A_2 (Nagylaki 1992). The frequency of A_1 in the next generation is

$$p' = (1-m)\left(\frac{W_1}{\bar{W}}\right)p. \tag{2.24}$$

The first term in (2.24) indicates how the frequency of A_1 is reduced by A_2 immigrants who arrive at gene flow rate m; the second term describes how selection increases the frequency of the locally fitter genotype A_1.

The conditions under which the locally favored allele A_1 will increase in frequency when initially rare are found when $p \approx 0$ and $\bar{W} \approx W_2$ in (2.24), which shows that $p' > p$ if $(1-m)W_1/\bar{W} > 1$, that is

$$\frac{W_1}{W_2} > \frac{1}{1-m} > 1. \tag{2.25}$$

Equation (2.25) is an example of the rule of thumb mentioned above: for given fitnesses (W_1, W_2), there is a rate of migration (m) below which the locally more fit genotype will spread when initially rare. Note that the maximal scope for local adaptation (spread of A_1) occurs as the gene flow rate m approaches zero.

We now reanalyze the same scenario, but follow genotype numbers rather than frequency. For this purpose, let N_i denote the *number* of genotype A_i and let N be total population density, $N = N_1 + N_2$. Consider first the dynamics of a population fixed for the less fit genotype A_2 so that $N = N_2$. Because the island population is a sink, $W_2 < 1$. We assume that a constant number I of A_2 individuals immigrate to the island each generation. The island population size changes according to

$$N' = NW_2 + I. \tag{2.26}$$

The size of this population will equilibrate when $N' = W_2N + I = N$. Solving for N gives the equilibrium density of A_2 genotypes:

$$\hat{N} = \frac{I}{1-W_2} \equiv \hat{N}_2. \tag{2.27}$$

Now assume that a few A_1 individuals are introduced to such an equilibrium population. The frequency of A_1 is $p = N_1/(N_1 + N_2)$. Because all immigrants have genotype A_2, the dynamics of A_1 are simply

$$N_1' = N_1W_1. \tag{2.28}$$

The density of A_2 will return to its equilibrium $\hat{N}_2$. The dynamics of A_1 are determined completely by the absolute fitness W_1. On the one hand, if $W_1 < 1$, then $N_1 \to 0$ and $p \to 0$; the locally favored A_1 is lost. On the other hand, if $W_1 > 1$, then $N_1 \to \infty$ and $p \to 1$; the locally favored A_1 spreads through the population. This analysis provides a criterion for determining whether a locally favored genotype will spread: its *absolute* fitness must be greater than one — regardless of the immigration rate I or the fitness of the less fit genotype. This criterion seems strikingly different from the rule of thumb provided by (2.25), which involves both the immigration rate and fitness of A_2.

The key to resolving the apparent discrepancy between these two criteria is to recognize that the migration rate m is a *variable*, not a fixed parameter as is implicitly assumed in the standard approach. Consider the recursion for total population size:

$$N' = N\bar{W} + I. \tag{2.29}$$

By definition, $m = I/N'$, which implies that $1 - m = N\bar{W}/N'$. By substituting this expression in (2.24), the recursion for p can be rewritten as

$$p' = \left(\frac{1-m}{\bar{W}}\right) W_1 p = \left(\frac{N}{N'}\right) W_1 p \tag{2.30}$$

Now if A_1 is rare and the immigrant genotype A_2 is near its equilibrium density, then $N' \approx N$ which implies from (2.30) that $p' \approx W_1 p$.. Thus we have recovered from (2.24) that the necessary and sufficient condition for A_1 to increase when rare is $W_1 > 1$.

Our analysis of this simple model leads to two unexpected conclusions. First, absolute — not relative — fitness governs the spread of a locally favored allele in a sink population. Second, provided it is not zero, the immigration rate, I, has no influence over the spread or loss of the favored allele in a sink population. To what extent do these conclusions depend on the simplicity of the model we analyzed? We next analyze the same black-hole-sink scenario for a diploid sexual population.

Consider a model in which fitness is determined by variation at a diploid locus with alleles A_1 and A_2. Assume that adults immigrate after selection but before reproduction, and that the population is censused immediately after (sexual) reproduction. (Similar conclusions hold if immigration occurs before selection.) As above, let N be total population size and p be the frequency of the locally favored A_1 allele. Assume that I immigrants, all with genotype A_2A_2, arrive each generation. Denote the birth-to-immigration viability of A_iA_j by v_{ij}. For simplicity, we assume that the expected fecundity is f, independent of genotype. The fitness of genotype A_iA_j is thus $W_{ij} = fv_{ij}$. Finally, assume that $W_{22} < 1$, so that in the absence of A_1, the population is a black-hole sink maintained only by immigration.

Recursions for the dynamics of this population can be derived as follows. Following random mating, genotype frequencies are in Hardy-Weinberg proportions: the densities of A_1A_1, A_1A_2, and A_2A_2 among newborns are Np^2, $N2p(1-p)$, and $N(1-p)^2$. After viability selection and immigration, the density of breeding adults is

$$N^* = v_{11}Np^2 + v_{12}N2p(1-p) + v_{22}N(1-p)^2 + I \tag{2.31}$$

Following reproduction, the density of newborns, N', is

$$N' = fN^* = N\bar{W} + fI \tag{2.32}$$

where $\bar{W} = p^2W_{11} + 2p(1-p)W_{12} + (1-p)^2W_{22}$ is mean fitness. Random mating returns genotype frequencies to Hardy-Weinberg proportions without altering allele frequencies. The frequency of A_1 of the newborns, p', is equal to the frequency of A_1 of the parents, i.e., $p' =$ (number of parental A_1 alleles)$/2N^* = (2Np^2v_{11}+2Np(1-p)v_{12})/2N^*$. Multiplying the numerator and denominator by f and using (2.32) shows

$$p' = \left(\frac{N}{N'}\right)\bar{W}_1 p \tag{2.33}$$

where $\bar{W}_1 = pW_{11} + (1-p)W_{12}$ is the average fitness of individuals with an A_1 allele. This recursion closely resembles the asexual equation (2.30).

Now examine the conditions under which the fitter A_1 allele will spread when rare. As above, consider first a population in which A_2 is the only allele present. Then $p = 0$ and $\bar{W} = W_{22}$. Setting the left-hand side of (2.32) equal to N and solving for N shows that the population will equilibrate at density

$$\hat{N} = \frac{fI}{1 - W_{22}} \equiv \hat{N}_{22}. \tag{2.34}$$

Now suppose a few copies of A_1 are introduced into this population so that $p \approx 0$ and $N' \approx N$. The gene-frequency recursion (2.33) is given approximately by $p' \approx pW_{12}$. Clearly, $p' < p$ if $W_{12} < 1$ and $p' > p$ if $W_{12} > 1$. The locally favored allele will increase when rare-provided the absolute fitness of heterozygous individuals exceeds one. As in the simpler asexual model, the spread of A_1 depends on absolute — not relative — fitness, and is independent of both the rate of immigration, I, and the fitness of the immigrant genotype, W_{22}.

Together, our asexual and diploid model results suggest that an absolute fitness criterion for spread of an initially rare allele is a generic feature of local adaptation and, hence, niche evolution in a black-hole-sink population. Before taking such a generalization too seriously, it is important to consider some potentially important ecological and genetic limitations of our two models.

The most obvious ecological deficiency in our models is that population size may increase without bound. How does density regulation affect the spread of a rare locally favored allele? We have analyzed a version of the above haploid model in which fitness is density dependent (Holt and Gomulkiewicz 1996) as well as the diploid model (Gomulkiewicz, Holt, and Barfield, in preparation). Our analysis shows that, once again, the locally fitter allele will spread in a black-hole sink population if the absolute fitness of heterozygotes is greater than one. Because fitness is density dependent, this criterion does depend on population size, but only indirectly through its influence on absolute fitness. (Density-dependence plays a more pronounced and complicated role in determining the eventual size and genetic composition of a population, once an initially rare locally favored allele increases.)

There are also a number of genetic limitations in our models (e.g., no mutation or genetic drift). Given our analysis in the first section, it is reasonable to ask how populations might adapt in the face of recurrent immigration if fitness depends on characters with polygenic inheritance. One could easily (and naively) extend the quantitative genetics model we considered in the first section to include recurrent immigration as follows. The population-size dynamics is described by (2.32) except that mean fitness $\bar{W}$ is defined by (2.3). It is not hard to show that the evolution of d, the distance of the current mean phenotype from the local optimum, satisfies the recursion equation $d' = (1 - M)kd + md_I$, where k is the evolutionary inertia (see Section 2.2), $M = fI/(N\bar{W} + fI)$ is a gene flow variable, I and f are as defined above, and d_I is the (fixed) difference between the mean phenotype of immigrants and the local optimum.

The dynamic features of this model are relatively simple to explore, and they are, in fact similar to those of the one-locus models. Unfortunately, there are several reasons why it seems premature to declare with any confidence that these evolutionary features are "robust" to genetic assumptions. First, this quantitative genetic analysis completely ignores departures from normality that are caused by immigration. Such departures are known to alter dynamical behavior under some circumstances. Second, this formulation also ignores the linkage disequilibrium (nonrandom associations between alleles at different loci) that is constantly generated by immigration, which in turn can affect variances and heritability. In the future, we plan to explore whether these neglected assumptions will have a noticeable impact on our main biological conclusions by analyzing more realistic (and complex) multilocus models.

2.5 NICHE EVOLUTION IN COUPLED SOURCE-SINK ENVIRONMENTS

The previous section considered evolution in a sink population maintained by immigration without dispersal back to the source. A natural generalization is to two habitats, one a source, the other a sink, with reciprocal dispersal (Holt 1996). For simplicity we consider a species with discrete generations and haploid genetics.

Census the population following dispersal. Let $N_i(t)$ be population size in habitat i at the start of generation t. In generation $t+1$ there are $N_i(t+1)$ individuals, who either immigrated from habitat j (denoted $N_{ij}(t+1)$) or did not (denoted $N_{ii}(t+1)$). Necessarily, $N_i(t+1) = N_{ii}(t+1) + N_{ij}(t+1)$. Individuals in habitat i at time t contribute to the population there at $t+1$ via production of offspring which do not emigrate. Let $N_{ii}(t+1) = a_{ii}(t)N_i(t)$. The quantity a_{ii} is the per capita contribution of habitat i to itself. Likewise, $N_{ij}(t+1) = a_{ij}(t)N_j(t)$ defines the per capita contribution of habitat j to habitat i. With this notation, a 2×2 matrix model describes the dynamics of the two coupled habitats over a single time step: $N(t+1) = A(t)N(t)$, where the vector $N(t) = (N_1(t), N_2(t))$, and the ijth element of matrix A is $a_{ij}(t)$. In general, $a_{ij}(t)$ may vary as the external environment changes, or because dispersal or local growth rates are density dependent and population size changes. Consider first the case of constant transition rates, $A(t) \equiv A$.

As with any matrix model (Caswell 1989), as t increases the popula-

tion settles into a *stable patch distribution* , defined by the right eigenvector of A, and changes in size at a constant rate λ (the dominant eigenvalue of A),

$$\lambda = \frac{1}{2}(a_{11} + a_{22} + \sqrt{(a_{11} - a_{22})^2 + 4a_{12}a_{21}}). \tag{2.35}$$

(Because of the low dimensionality of the above model, one can explicitly solve for eigenvalues and eigenvectors as a function of arbitrary matrix elements a_{ij}; this is not generally possible). The stable patch distribution has a defined fraction of the population in each patch. A right eigenvector for A (with elements summing to unity) is

$$(w_1, w_2) = \left(\frac{\lambda - a_{22}}{\lambda - a_{22} + a_{21}}, \frac{a_{21}}{\lambda - a_{22} + a_{21}}\right). \tag{2.36}$$

A familiar interpretation of the left eigenvector in an age-structured matrix model is that it gives "reproductive value" (Caswell 1989) — the relative contribution of an individual to future generations — as a function of age. A left eigenvector of this stage-structured patch model is

$$(v_1, v_2) = \left(\frac{\lambda - a_{22}}{\lambda - a_{22} + a_{12}}, \frac{a_{12}}{\lambda - a_{22} + a_{12}}\right) \tag{2.37}$$

which likewise describes *spatial reproductive value*, the contribution of an individual in habitat i to future generations (in both habitats).

Adaptive evolution may occur if genetic variants arise with different values for the a_{ij}. A novel mutant spawns a subpopulation, whose dynamics can also be described by a 2×2 matrix model; this subpopulation settles into its own stable patch distribution and grows at its own asymptotic growth rate. If this growth rate exceeds that of the resident clone, then initially the new clone is favored, and it will (deterministically) increase in frequency. Because favorable mutants can be lost to stochastic birth-death effects when sufficiently rare, the larger the positive effect of the mutational change upon clonal fitness, the more likely it is to increase when rare.

A concept which underlies much of the modern theory of life history evolution (e.g., the evolutionary theory of senescence) is the notion of the *force of selection.* If we have a measure of fitness F which is a function of parameters q_i, the force of selection on parameter i is $\partial F/\partial q_i$. If $\partial F/\partial q_i > 0$, a clonal variant with slightly higher q_i increases when rare; the larger this quantity is, the more rapidly the mutant spreads, and the less likely it is to be lost due to demographic stochasticity.

In the above matrix model, an appropriate fitness measure is the dominant eigenvalue of A. This measure is a function of all the a_{ij}. Caswell (1989) formalized the notion of force of selection for transition matrices using eigenvalue sensitivity analysis. He showed that if one tweaks only element a_{ij} in a transition matrix, the effect on the dominant eigenvalue is

$$\frac{\partial \lambda}{\partial a_{ij}} = \frac{v_i w_j}{<v, w>} \tag{2.38}$$

where $<v, w>$ is the inner product of v and w. If mutations arise which slightly alter single matrix elements, the above expression can be used to evaluate the

relative strength of selection favoring, or disfavoring, them. Selection should be strongest for transitions from classes that numerically dominate the population (large w_j) into classes with a high reproductive value (large v_i). The quantity $v_i w_j$ in essence describes a demographic "weight" accorded by selection to favor (or disfavor) some transitions, over others.

More generally, mutations may affect multiple transition elements. Assume all the matrix elements are functions of a single parameter, q. The strength of selection favoring mutations increasing q is

$$\frac{\partial \lambda}{\partial q} = \sum_{i,j} \frac{v_i w_j}{\langle v, w \rangle} \frac{\partial a_{ij}}{\partial q}. \tag{2.39}$$

We now make the model a bit more concrete. We will first describe an ecological realization of the above matrix model, then return to the evolutionary question. Imagine that in generation t, the growth rate in habitat i before dispersal is $R_i(t)$. A fraction e of individuals in habitat 1 disperse to habitat 2; a fraction e' of individuals disperse from habitat 2 to 1. The following matrix model describes population growth in the two coupled habitats:

$$\begin{pmatrix} N_1(t+1) \\ N_2(t+1) \end{pmatrix} = \begin{pmatrix} (1-e)R_1 & e'R_2 \\ eR_1 & (1-e')R_2 \end{pmatrix} \begin{pmatrix} N_1(t) \\ N_2(t) \end{pmatrix}. \tag{2.40}$$

Now assume that the external environment is constant and that habitat 2 is a sink with R_2 less than 1. In habitat 1, growth rates are locally density dependent, such that R_1 declines monotonically as a function of density. Denote the density at which $R_1(N_1) = 1$ as K_1 (the carrying capacity of habitat 1) and let the maximal growth rate as N_1 approaches 0 be R_1'.

If the population persists at a stable equilibrium, $\lambda = 1$. After substitution and some algebraic manipulation, we find

$$R_1(N_1^*) = \frac{1-(1-e')R_2}{(1-e)-(1-e-e')R_2} > 1 > R_2 \tag{2.41}$$

where N_1^* is equilibrium density in habitat 1. Thus, $N_1^* < K_1$; coupling to a sink depresses source density. This increases source growth rates, compensates for decline in the sink, and permits landscape-level equilibrium. The ability of a species to compensate is set by its maximal growth rate in the source, R_1'; if this is less than the left quantity in the above inequality, the species is driven to extinction because dispersal drains away source growth. A sufficient condition for persistence is that $R_1'(1-e) > 1$. We assume source growth rates permit persistence.

At demographic equilibrium, the realized source growth rate is independent of source parameters (e.g., R_1') and depends solely on the rate of decline in the sink, and dispersal. At this equilibrium, the matrix elements have fixed values, and we can apply the machinery of the force of selection to study adaptation. The relative reproductive values and patch abundances of source and sink habitats are as follows: (1) $v_{\text{source}} > v_{\text{sink}}$ if $1 > R_2$ (which is always true for a sink); (2) $w_{\text{source}} > w_{\text{sink}}$ if $1 > (1-e')R_2 + eR_1(N_1^*)$ (which often — but not always — holds).

Now, consider mutations which improve fitness slightly in the sink habitat. The cumulative effect of such mutations as they become fixed may be to transform a sink habitat into a potential source habitat. If so, a species' niche will have evolved: the population can persist in the original sink habitat without immigration. We now have the ingredients needed for predicting the likelihood of such evolution. Holt (1996) describes a number of limiting cases (e.g., involving tradeoffs in fitness in the two habitats). Here we consider two simple examples (the student should work out details as an exercise). Consider mutations whose only effect is a slight increase in sink fitness (with no back-effect on source-fitness parameters).

First, assume $e \approx 0$ (little dispersal, source-to-sink). This implies $R_1 \approx 1$, and $a_{21} \approx 0$. Hence, the stable patch distribution is approximately $(w_1, w_2) = (1, 0)$. After substitution, one finds $\partial\lambda/\partial R_2 \approx 0$. The force of selection for increasing fitness in the sink is negligible, basically because no individuals encounter the sink habitat.

Second, assume $e' \approx 0$ (little back-dispersal, sink-to-source). In this case, $R_1 \approx 1/(1-e)$, and $a_{12} \approx 0$, so the vector of spatial reproductive values is $(v_1, v_2) \approx (1, 0)$. Again one finds that $\partial\lambda/\partial R_2 \approx 0$. Because individuals in the sink make no long-term contribution to the overall population, small improvements in their fitness are of negligible evolutionary importance. Moreover, if mutations arise which have deleterious effects in the sink (but not in the source), selection is weak for removing such mutants from the population. With recurrent mutation, the load of deleterious mutations is likely to be heavier in the sink, than in the source (Kawecki 1995; Holt 1996).

This result meshes with the explicit genetic models for a "black-hole" sink in the last section. The two-patch model should converge on a black-hole-sink model when $e' \to 0$. We showed above that mutations of very small effect on fitness are unlikely to be selected in black-hole sink at demographic equilibrium. Because the expression for the force of selection aims at characterizing the fate of mutations of small effect, the two results are equivalent.

Drawing these examples together, they suggest that niche evolution is less likely when dispersal rates are low, or are asymmetrical, with little dispersal from sink to source. Conversely, niche evolution may be more likely if dispersal rates are high and symmetrical (Kawecki 1995; Holt 1996).

2.6 EVOLUTION OF DISPERSAL AND TEMPORAL HETEROGENEITY

2.6.1 Implications for niche conservatism

This model, explored in the last section, highlighted the importance of dispersal in defining how selection averages over different environments in determining the evolutionary trajectory of a species. There is an enormous literature on the ecology and evolution of dispersal. This is an entire topic on its own, beyond the scope of this chapter. However, the above simple matrix model can be used to illustrate a few basic points about the evolution of dispersal relevant to niche evolution in heterogeneous landscapes.

Consider genetic variants which have the same fitness within patches,

but differ in their rates of movement. Evolution in dispersal occurs because different dispersal syndromes define how a given variant experiences environmental heterogeneity in determining its overall fitness. Let habitat 1 have higher fitness than habitat 2. In the two-habitat matrix model, for the moment assume that local fitnesses R_i are fixed, and that dispersal is symmetrical among patches (i.e., $e = e'$). Overall fitness λ is a function of dispersal rate e, $\lambda(e)$; fitness decreases monotonically with e, declining from $\lambda(0) = R_1$ to $\lambda(.5) = (R_1 + R_2)/2$ to $\lambda(1) = \sqrt{R_1 R_2}$.

Now assume the population is initially fixed for a particular dispersal rate, e'', that fitness in the source habitat is density dependent, and that the resident population is at demographic equilibrium (viz., $\lambda(e'') = 1$). An invading clone with a different dispersal rate, when rare, experiences density dependence in the source from the resident. When the invader is rare, the resident's abundance can be assumed fixed during the initial stages of invasion; the invader thus experiences constant habitat-specific growth rates and settles into its stable patch distribution and asymptotic growth rate. Because of the monotonic relationship between overall growth rate and dispersal rates, a rare clone with lower e than the resident always increases when rare; a clone with higher e is excluded. A fuller analysis shows that a polymorphic equilibrium is not feasible, provided one habitat has a fixed fitness less than one, and the system moves toward demographic equilibrium.

This suggests that dispersal rates should evolve toward lower values in spatially heterogeneous (but temporally constant) environments. Given unlimited flexibility in dispersal, the evolutionarily stable state of the system described by our two-patch model is zero dispersal, with all individuals occupying habitat 1, and none in habitat 2. (This conclusion depends on the assumption that abundances are sufficiently large to be treated as continuous variables, rather than discrete integers (Holt 1985; Holt and McPeek 1996).) As dispersal becomes lower, so does the exposure of individuals to the sink habitat. We earlier saw that the force of selection favoring improved adaptation to the sink becomes negligible at low dispersal rates. Thus, evolution of dispersal in a spatially heterogeneous landscape indirectly strengthens the tendency toward niche conservatism.

McPeek and Holt (1992) and Holt and McPeek (1996) have explored the evolution of dispersal in two-habitat models, in which local fitness in each habitat is a monotonically declining function of density, $R_i(N_i)$, and there is some $N_i = K_i > 0$, where $R_i(K_i) = 1$, but $K_1 > K_2$. In this case, no habitat is inevitably a sink. However, given dispersal, if more individuals leave a patch than enter it, densities there decline, leading to an increase in local fitness. Conversely, more individuals must enter than leave the low-K patch, pushing numbers up and depressing local fitness. The net effect of dispersal is to create gradients in local fitness, down which individuals on average tend to move. This is clearly disadvantageous.

In one special circumstance, however, dispersal can occur without this deleterious fitness effect. If $eK = e'K_2$, as many individuals leave as enter each habitat. Thus, each habitat equilibrates at its respective carrying capacity, such that fitnesses are equalized across space (the "ideal free distribution" of habitat selection theory (Fretwell 1972)). McPeek and Holt (1992) showed that this fitness equilibration could be generated in two distinct ways: (1) phe-

notypic plasticity, in which each individual disperses at different rates in different habitats, or (2) a mixture of fixed dispersal types, one low and one high.

An extreme but illuminating case is for all individuals to leave their natal habitat each generation, but then to resettle into the ideal free distribution. The probability that an individual will end up in habitat i is thus $K_i/(K_1+K_2)$. Individuals carrying novel mutations with habitat-specific effects on fitness in habitat i are likely to experience this change in fitness a fraction $K_i/(K_1+K_2)$ of generations. This implies that adaptive evolution is "skewed" toward the habitat with the greater K. For a given allelic change in local fitness, positive selection should be greater in the habitat with higher K, and negative selection weeding out deleterious mutants should likewise be stronger. This may imply that low-K habitats, initially within the niche of the species, might be lost over evolutionary time.

Drawing together the various strands of theory presented above, we see that spatial heterogeneity alone tends to foster niche conservatism. Given limited dispersal, selection tends to be weighted against adaptive improvement in sink habitats, outside a species' niche. This tendency is weakened if dispersal forces individuals to experience the sink habitat. Yet selection acts against dispersal if there is spatial heterogeneity in fitness, which is ensured if some habitats are permanent sinks. Considering the coevolution of dispersal and local adaptation suggests an overall tendency toward increased habitat specialization or niche conservatism.

2.6.2 Temporal variation and niche evolution

All the above models assumed environments in which fitness parameters varied in space, but not in time. Introducing temporal variation raises challenging, unsolved research problems in evolutionary ecology. In this section we will touch on several distinct issues, indicating the range of effects of temporal variation to be expected.

Consider again the haploid black-hole-sink model. Let local absolute fitnesses and immigration rates vary with time, as follows

$$N_1(t+1) = N_1(t)W_1(t) + I(t) \quad (2.42)$$

$$N_2(t+1) = N_2(t)W_2(t) \quad (2.43)$$

where allele 1 is the ancestral immigrant type, and allele 2 is a new mutation. Further, imagine that temporal variation is cyclic, with period T. In the absence of density dependence, the fate of allele 2 is clearly independent of the fitness or rate of immigration of allele 1. Allele 2 increases, provided its geometric mean rate of increase over T generations exceeds 1; if the geometric mean fitness is less than 1, allele 2 will decline toward extinction. Because the geometric mean is always less than the arithmetic mean, temporal variation in fitness makes it harder for an allele to increase when rare.

This simple model suggests that temporal environmental variation tends to hamper niche evolution in sink environments. Extending this to the coupled source-sink environment quickly leads to models which are analytically intractable. There are two sources of difficulty.

First, even in the absence of density-dependence, temporal variation in fitness parameters can confound expectations. Tuljapurkar (1991) [p. 82]

provides an interesting example for a 2×2 matrix model. There is cyclic variation between matrix A in one generation, with dominant eigenvalue $\lambda_A < 1$, and matrix B in the next, with dominant eigenvalue $\lambda_B < 1$. Yet the overall eigenvalue for the compound matrix AB, which describes growth over successive generations, exceeds 1. Unraveling the effects of different patterns of temporal variation in fitness parameters on the overall course of selection in a spatially heterogeneous environment is an important, challenging problem.

Second, given density dependence, temporal variation in fitness parameters implies variation in local densities. Even in the simplest models (e.g., logistic growth in two habitats in a cyclic environment), this leads to nonlinear expressions for local density that cannot be solved explicitly; thus the temporal pattern of variation in fitness cannot be expressed analytically.

One indirect consequence of temporal variation which may have profound implications for niche evolution is its effect on dispersal. If local fitness varies through time, dispersal can become advantageous. Theoretical studies have highlighted the importance of asynchronous temporal variation in fitness in promoting the evolution of dispersal in heterogeneous landscapes. Such temporal variation can arise from extrinsic environmental factors or endogenously. For instance, Holt and McPeek (1996) examined a two-habitat model in which local fitnesses were defined by $R_i(N_i) = \exp[r_i\,(1 - N_i/K_i)]$. As is well known, at low r_i, in the absence of dispersal populations settle into stable equilibria, whereas at high r_i, chaotic dynamics emerges. At low r, dispersal is strongly selected against. At high r, the population exhibits chaotic dynamics; a dispersing clone can increase when rare, and persists in a stable equilibrium with a low-dispersal clone. If individuals have habitat-specific dispersal rates, a chaotic population evolves toward a state with persistent dispersal. Similarly, extrinsic temporal variation in fitness parameters that is uncorrelated among habitats favors persistent dispersal (McPeek and Holt 1992). Temporal variation in local fitness parameters thus favors dispersal. High rates of dispersal in turn tend to weaken forces favoring niche conservatism. Thus, temporal environmental variation may indirectly lead to niche evolution via its influence on the evolution of dispersal.

2.7 NICHE EVOLUTION IN METAPOPULATIONS

So far, we have concentrated on the details of adaptive evolution in very simple landscapes: single habitat patches coupled to an external source, and pairs of habitat patches with reciprocal dispersal. Most species exist in much more complex landscapes, with mosaics of discrete habitat types and smooth gradients. A useful way station between simple one- and two-habitat landscapes and realistic landscapes is provided by metapopulation models which ignore the details of localized dispersal but do capture some important aspects of patchiness. In this final section of the chapter, we explore the interplay of niche evolution and metapopulation dynamics.

There are two canonical metapopulation structures: (1) island-mainland, and (2) multiple identical patches (Hanski 1991). We consider each of these in turn:

2.7.1 Island biogeography

The black-hole-sink model considered a species in a single habitat patch, coupled to a source which did not experience reciprocal effects. Now imagine there are many such habitats, which like islands in a sea may vary in size and distance from the source.

Rather than consider the detailed population and evolutionary dynamics of the species in each patch, we can attempt to abstract the essence of the microevolutionary and population dynamic processes as follows (where, for simplicity, we refer to "islands" rather than patches):

1. All immigration is from the mainland, which is assumed to contain a species at its evolutionary equilibrium. Colonization is defined as immigration onto unoccupied islands; the rate parameter c_m defines the rate of colonization per empty island.

2. All colonizing propagules are initially maladapted to the local environment and therefore will inevitably go extinct, in the absence of evolution (see Section 2.2). We assume that such extinctions are described by an exponential distribution with rate parameter e_m. All the rate parameters may vary with island area or distance (see below).

3. Given appropriate genetic variation, a local population may evolve so as to increase its mean fitness or carrying capacity, which enhances population persistence. Catastrophes can still occur, however, leading to local extinctions even for well-adapted populations. We assume that such extinctions occur at rate $e_a < e_m$.

4. The final ingredient we need is the rate of evolution. We assume that local populations exist in just two states: "maladapted" (their original state, just after colonization) and "adapted" (their final state, conditional on local persistence, after selection has pushed the population to its new, local optimum). For simplicity, we ignore intermediate states and assume that an exponential distribution with rate parameter E describes evolutionary transitions from maladapted to adapted states.

Any given island can occur in three states: empty; occupied but maladapted; occupied and adapted. Let P_m be the fraction of islands in which populations are maladapted, and P_a be the fraction in which local adaptation has occurred. The fraction of empty islands available for colonization is $1 - P_m - P_a$. The dynamics of the system are described by a coupled pair of differential equations:

$$\frac{dP_m}{dt} = c_m(1 - P_a - P_m) - EP_m - e_m P_m \tag{2.44}$$

$$\frac{dP_a}{dt} = EP_m - e_a P_a \tag{2.45}$$

At equilibrium,

$$P_a^* = \frac{E}{e_a} P_m^*, \qquad \frac{P_a^*}{P_a^* + P_m^*} = \frac{E}{E + e_a} \equiv \epsilon \tag{2.46}$$

The quantity ϵ describes the fraction of island populations which have become adapted to their environments. Because we assume no interisland migration, each such population is a distinct taxon, so ϵ also measures the fraction of island populations which might be viewed as endemic by a taxonomist. By writing down the above set of equations, we implicitly assume that the number of islands is sufficiently great that ensemble dynamics can be treated deterministically.

Island area, and distance to the source, can influence both the ecological rate parameters of colonization and extinction, and the rate of evolution from a maladapted to adapted state. Somewhat surprisingly, the average equilibrial evolutionary state does not depend upon the rate of colonization from the source, or the rate of extinction of maladapted populations, but only upon the rate of local evolution and extinctions of adapted populations.

Consider first purely ecological effects upon extinction. Owing to demographic stochasticity, adapted populations on large islands are likely to persist longer, per population, than adapted populations on small islands. If dispersal from the source is rare, it is not clear that distance should have any systematic effect upon extinction. However, there may be indirect effects of distance on extinction rates, such as fewer competing or predatory species on more distant islands, leading to reduced extinctions at greater island distance from the source. Considering just these ecological effects, one expects e_a to increase with island area, and possibly to increase as well with island distance from the source (see Holt (1997) for examples and further discussion).

Island area and distance could also indirectly determine the rate of microevolution, E. For instance, if evolution is limited by the pool of variation, then all else being equal, larger islands will harbor larger populations, which can generate more variation via mutation and sustain such variation in the face of genetic drift. The rate of evolution could thus increase with island area, augmenting the ecological effect of island area on endemism.

2.7.2 "Proper" metapopulation

In the long run, even source populations can go extinct, or evolve. Consider a species which occupies an ensemble of habitat patches, where each occupied patch is a potential source of colonists for empty patches. Let P be the fraction of patches occupied. The canonical metapopulation model (Hanski 1991) is

$$\frac{dP}{dt} = cP(1-P) - eP \qquad \Rightarrow \qquad P^* = 1 - \frac{e}{c} \tag{2.47}$$

where e is the rate of local extinction, per patch, c describes the rate of colonization of empty patches, per occupied patch, and P^* is patch occupancy at equilibrium.

Now, as before, assume that local populations can be either adapted or maladapted, and that their evolutionary state is made manifest in local extinction rates. One can imagine various scenarios regarding colonization (Holt and Gomulkiewicz, in prep.). The simplest is to assume that only empty patches are colonized, and that only adapted populations are sufficiently vigorous to send out colonists. Assuming again that the rate of local evolution is characterized

by a constant transition-rate parameter, E, the metapopulation model is

$$\frac{dP_m}{dt} = -e_m P_m - E P_m \tag{2.48}$$

$$\frac{dP_a}{dt} = c_a(1 - P_a - P_m)P_a + E P_m - e_a P_a. \tag{2.49}$$

There are two possible equilibria: (1) global extinction, if $e_a > c_a$; (2) $P_m^* = 0$, and $P_a^* = 1 - e_a/c_a$ if $e_a < c_a$. The rate of evolution, E, is thus irrelevant to the long-term state of the system (in sharp contrast to the above island model). However, evolution can matter crucially in determining transient dynamics in the metapopulation. Consider a system in which there has been an abrupt change in climate, such that all initial populations are maladapted. Numerical integration of the above model reveals that a species may display an excursion to very low values of occupancy $P_a + P_m$ before the population increases to its eventual equilibrium. As noted above, a key assumption in patch occupancy models is that the number of patches in question is very large, so that deterministic approximations of stochastic processes are sensible. In real metapopulations, when occupancy gets very low, there is likely to be a small number of actual populations in question, and a metapopulation equivalent of demographic stochasticity can arise (Hanski et al. 1996). Analogous to our initial model of adaptation in a changed environment in a single patch, a metapopulation may suffer extinction due to chance events as it passes through a phase of transient maladaptation in a novel environment.

The above metapopulation models, of course, provide a caricature of population and evolutionary dynamics. Future work will address the adequacy of some of their key assumptions (e.g., constant evolutionary rates). Moreover, an obvious next step will be to assume localized dispersal, and to examine evolutionary dynamics in environments with different patterns of heterogeneity. Environments are heterogeneous in many ways, ranging from gentle spatial gradients, fixed in time, to ephemeral habitat patches winking on and off during succession, to landscapes with complex fractal spatial structure. The "texture" of the selective environment is likely to be a key determinant of whether or not a species exhibits niche conservatism, or evolution.

The basic message of this chapter is that population dynamics acts as a kind of constraint within which evolutionary dynamics occur, and that an understanding of niche conservatism and evolution, in particular, mandates analyzing evolutionary processes within an appropriate landscape and population dynamical context.

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