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Vol. 28, No. 2, October 1985
Printed in Belgium

Population Dynamics in Two-Patch Environments: Some Anomalous Consequences of an Optimal Habitat Distribution

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Received January 17, 1984

The effect of dispersal on population size and stability is explored for a population that disperses passively between two discrete habitat patches. Two basic models are considered. In the first model, a single population experiences density-dependent growth in both patches. A graphical construction is presented which allows one to determine the spatial pattern of abundance at equilibrium for most reasonable growth models and rates of dispersal. It is shown under rather general conditions that this equilibrium is unique and globally stable. In the second model, the dispersing population is a food-limited predator that occurs in both a source habitat (which contains a prey population) and a sink habitat (which does not). Passive dispersal between source and sink habitats can stabilize an otherwise unstable predator–prey interaction. The conditions allowing this are explored in some detail. The theory of optimal habitat selection predicts the evolutionarily stable distribution of a population, given that individuals can freely move among habitats so as to maximize individual fitness. This theory is used to develop a heuristic argument for why passive dispersal should always be selectively disadvantageous (ignoring kin effects) in a spatially heterogeneous but temporally constant environment. For both the models considered here, passive dispersal may lead to a greater number of individuals in both habitats combined than if there were no dispersal. This implies that the evolution of an optimal habitat distribution may lead to a reduction in population size; in the case of the predator–prey model, it may have the additional effect of destabilizing the interaction. The paper concludes with a discussion of the disparate effects habitat selection might have on the geographical range occupied by a species.

1. INTRODUCTION

Each species of organism has a characteristic pattern of variation in abundance over space. Understanding the factors responsible for the manifest diversity in distributional patterns is a classic goal of ecology (cf. Elton, 1927; Andrewartha and Birch, 1954; Krebs, 1978; Brown

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and Gibson, 1983). A growing body of evidence suggests that dispersal is important in setting the mean abundances and patterns of fluctuations of many natural populations (e.g., Connor *et al.*, 1983; Gaines and McClenaghan, 1980). These empirical findings have stimulated much theoretical work on the effects of dispersal and spatial heterogeneity in population dynamics (for reviews and exemplary studies see Levin, 1976; Okubo, 1980; Hastings, 1982; Kareiva, 1982; Vance, 1984). Dispersal is also widely recognized to be important in the evolution of species, both directly as a component in life history strategies (Horn and Rubenstein, 1983), and indirectly as a determinant of geographical distributions, patterns of gene flow, and effective population sizes (e.g., Endler, 1977).

In this paper I examine in two models the influence of passive dispersal on the total size and stability of a population distributed over two distinct habitat patches. The first model considers a single species that experiences density-dependent growth in two patches coupled by dispersal. A graphical solution of this model is developed (see Fig. 1). This graphical approach allows one to determine the spatial pattern of abundance at equilibrium for any model in which there is negative density-dependence within patches and passive dispersal between patches. The two-patch model considered here is a special case of a n-patch, single-species model recently analyzed by Hastings (1983). Hastings shows that dispersal is selected against in a spatially varying but temporally constant environment, and he further

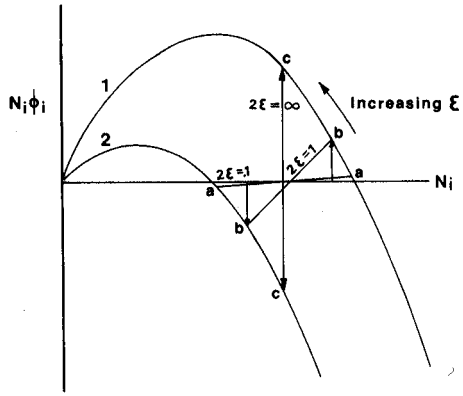

FIG. 1. A graphical solution to the two-patch growth model (♦). Three possible pairs of equilibrium points $(N_1^*, N_1^*\phi_1)$, $(N_2^*, N_2^*\phi_2^*)$ are shown (a, b, and c). In each pair, the points are displaced equal vertical distances from the N_i-axis and are connected by a straight line with slope 2ε. As the dispersal rate rises, the two patches converge in density, and in the limit of very high dispersal rates, density in the two patches is equalized. Refer to the text for details.

suggests that selection on dispersal rates does not in general maximize total population size. Here I amplify and extend these results in two ways. First, I use the ecological theory of habitat selection (Fretwell, 1972; Rosenzweig, 1974, 1979, 1981; Charnov, 1976; Whitham, 1980) to develop an intuitive, heuristic argument for why dispersal should be selected against in a temporally constant environment in which kin effects (Hamilton and May, 1977) can be ignored. This argument suggests that Hastings' result applies broadly to multispecies systems with stable equilibria. Second, I develop a graphical analysis of the two-patch case which allows one to predict whether evolution toward reduced dispersal will increase, decrease, or leave unchanged total population size.

The second model is of a general resource–consumer or predator–prey system in which consumers passively disperse between a "source" habitat, which contains the resource, and a "sink" habitat, which does not. It is shown that dispersal into the sink can stabilize an otherwise unstable interaction and can also allow a greater total number of consumers to be supported by the resource than if there were no dispersal. Selection against dispersal may therefore reduce the number of consumers sustained by the resource and, moreover, may even destabilize the system. This analysis helps delineate those situations in which the evolution of an optimal habitat distribution might be expected to reduce the total abundance of a population.

The effects analyzed in these models occur over short spatial scales, corresponding to a small multiple of the typical ranging distances of individuals. In the Discussion, I argue that optimal habitat selection may also have important consequences over broader spatial scales, and in particular may restrict the geographical range eventually occupied by a newly formed species.

2. Density-dependent Growth in Two Patches

2.1. *Passive Dispersal*

We will consider the following general model of density-dependent growth in two patches of equal size, linked symmetrically by passive dispersal:

$$\begin{aligned} dN_1/dt &= N_1\phi_1(N_1) - \varepsilon N_1 + \varepsilon N_2, \\ dN_2/dt &= N_2\phi_2(N_2) + \varepsilon N_1 - \varepsilon N_2. \end{aligned} \tag{1}$$

Here, N_i is the number of individuals in patch i, and the functions $\phi_i(N_i)$ encapsulate within-patch density-dependent processes influencing pop-

ulation growth. We assume that $d\phi_i/dN_i < 0$ for all N_i (i.e., no Allee effects). The carrying capacity K_i of patch i is defined to be that number N_i such that $\phi_i(K_i) = 0$. By convention, patch 2 will have the lower carrying capacity (i.e., $K_2 \leqslant K_1$).

With no dispersal ($\varepsilon = 0$), each patch equilibrates at its respective carrying capacity, and the total number of individuals present is just the sum of the carrying capacities, $K_1 + K_2$. The effect of passive dispersal should be to smooth out spatial variation in density. We show below that passive dispersal can in some circumstances lead to a total population size at equilibrium greater than $K_1 + K_2$. Before discussing the circumstances in which this is observed, we first explore the properties of the equilibrium, and then argue that natural selection should reduce the rate of dispersal.

Model (1) is equivalent to the following pair of equations for rates of change in the total population size, N_T, and in the between-patch difference in numbers, $N_1 - N_2$:

$$dN_T/dt = d/dt(N_1 + N_2) = N_1\phi_1 + N_2\phi_2, \tag{2}$$

$$d/dt(N_1 - N_2) = (N_1\phi_1 - N_2\phi_2) + 2\varepsilon(N_2 - N_1). \tag{3}$$

At equilibrium (denoted by an asterisk), assuming that $N_1^* \neq N_2^*$,

$$N_1^*\phi_1 = -N_2^*\phi_2, \tag{4}$$

and

$$\frac{N_1^*\phi_1 - N_2^*\phi_2}{N_1^* - N_2^*} = 2\varepsilon. \tag{5}$$

(To simplify our notation, the expression $N_i^*\phi_i$ is shorthand for $N_i^*\phi_i(N_i^*)$.)

In Fig. 1 we plot the total growth rate of each patch, $N_i\phi_i$, against the number of individuals in that patch. There are an infinite number of pairs of population sizes N_1 and N_2 for which the total growth rate equals zero [Eq. (4)] (e.g., pairs a, b, and c in Fig. 1). For the system to be in equilibrium, however, the relative proportions of the two populations must also be unchanging [Eq. (5)]. For a given pair of possible equilibrial points, $(N_1^*, N_1^*\phi_1)$ and $(N_2^*, N_2^*\phi_2)$, the slope of the straight line connecting them in the figure is $(N_1^*\phi_1 - N_2^*\phi_2)/(N_1^* - N_2^*)$, which from Eq. (5) must equal 2ε. A simple geometrical construction for finding the equilibrium is thus to find a pair of points on the growth curves, chosen such that $N_1^*\phi_1 = -N_2^*\phi_2$, that can be connected by a line of slope 2ε. This graphical technique can be used to find the pattern of equilibrial densities for any pair of growth functions $(N_i\phi_i)$ and rate of dispersal (ε).

In Appendix I, I show under very broad assumptions that this

equilibrium is unique and that no limit cycles exist, ensuring global stability. Several qualitative conclusions may be drawn from the figure (see also Appendix I). As seems intuitively reasonable, if $K_1 > K_2$, $K_2 < N_2^* < N_1^* < K_1$: dispersal causes the two patches to converge in abundance, but the patch with higher K retains the higher number of individuals. The population as a whole is in balance because its positive growth in patch 1 is matched by its negative growth in patch 2. At equilibrium, if $\varepsilon > 0$ there is a positive correlation across space between population density and local growth rates (the ϕ_i). The total population, N_T^*, is bounded by $2K_2 < N_T^* < 2K_1$. As $\varepsilon \to \infty$, the line in Fig. 1 connecting the equilibrium points becomes increasingly vertical, and the two patches converge to a common abundance. Below, we use the latter fact to construct a graphical model for contrasting the total population size sustained at high rates of dispersal (i.e., for $\varepsilon \to \infty$) with the total population size at low dispersal rates.

2.2. *Optimal Habitat Selection Implies* $\varepsilon = 0$

We might expect natural selection to act on the rate of dispersal, thereby indirectly influencing the total abundance of the population in the two patches. The theory of habitat selection (Fretwell, 1972; Rosenzweig, 1974, 1979, 1981; Charnov, 1976; Whitman, 1980) predicts the evolutionarily stable distribution of a population in which individual strategies of habitat utilization have been honed by natural selection. This theory provides a heuristic argument for why $\varepsilon = 0$ is the only evolutionarily stable rate of dispersal in temporally constant but spatially varying environments. Hastings (1983) has recently provided a formal proof that $\varepsilon = 0$ is the ESS for a single species distributed over n patches.

Let F_i be the fitness of an animal that spends all its time in patch i ($i = 1, 2$). If it spends a fraction p of its time in patch 1, its average fitness is $pF_1 + (1-p)F_2$. We assume that per capita growth rates are adequate fitness measures; this assumption is strictly accurate only for models of haploid or asexual selection (Ginzburg, 1983). The expected direction of natural selection is simple to predict: if $F_1 > F_2$, an animal should obviously attempt to spend all its time in patch 1. With some additional assumptions, this intuitive prediction leads to the "ideal free distribution" proposed by Fretwell (Fretwell and Lucas 1970; Fretwell, 1972). All individuals in a given patch are assumed to have equal expected fitnesses (Whitham, 1980, discusses the consequences of relaxing this assumption), to unerringly assess patch quality, and to move between patches without cost or interference from other individuals. Fitness within a patch is assumed either to be constant or to decrease as the number of conspecifics in that patch rises. With these assumptions, at an evolutionary equilibrium either

(i) all individuals should be in patch 1, if $F_1 > F_2$, or in patch 2, if $F_2 > F_1$; or,

(ii) the numbers of individuals in the two patches should be adjusted such that fitnesses are equalized, i.e.,

$$F_1 = F_2.$$

In general, case (ii) requires that fitnesses be a direct or indirect function of population density. The expected equalization of fitness across patches can be thought of as a special case of selection acting upon assortment into alternative phenotypic classes when phenotypic fitnesses are frequency-dependent (Slatkin, 1978).

We now assume that the population is stable in size and distribution, occupies both patches, and exhibits an optimal habitat distribution (i.e., matches conditions (i) or (ii) above). If there is spatial variation in density these assumptions are inconsistent with passive dispersal for any $\varepsilon > 0$. The growth rates of the population in each of the two patches are

$$dN_1/dt = N_1 F_1 + \varepsilon(N_2 - N_1) \text{ and } dN_2/dt = N_2 F_2 + \varepsilon(N_1 - N_2). \quad (6)$$

Adding and subtracting, at equilibrium $N_1^* F_1 = -N_2^* F_2$ and $(N_1^* F_1 - N_2^* F_2) + 2\varepsilon(N_2^* - N_1^*) = 0$. Given optimal habitat selection, we know that $F_1 = F_2$, hence $F_1 = F_2 = 0$ and $2\varepsilon(N_1^* - N_2^*) = 0$. If $N_1^* \neq N_2^*$, then ε must be 0. These growth equations include as a special case direct density-dependence in the two patches (i.e., $F_i = \phi_i(N_i)$ in model (1)). The form of the argument suggests that the conclusion that $\varepsilon = 0$ is the only ESS in a temporally constant environment holds for a much broader class of models than considered in Hastings (1983). For instance, in the predator–prey model to be considered below, the per capita growth rate of the predator is a function of prey density, with no direct density-dependence, yet the only ESS for the predator is a zero rate of dispersal.

To see why this conclusion holds, note that when $F_1 = F_2$, an individual does not gain any fitness benefit from moving between patches. When $F_1 > F_2$, individuals in patch 1 would suffer a fitness loss were they to move to patch 2. Conversely, individuals in patch 2 benefit (and by an amount equal in magnitude) from dispersal into patch 1. Since the behavior in question, passive dispersal at rate ε, is invariant across space, it is appropriate to ask whether an individual chosen at random from the population would benefit from changing patches. We earlier saw that at equilibrium the patch with higher fitness (patch 1 in Fig. 1) also had the higher number of individuals. Therefore, on average, passive dispersal is disadvantageous and should be selected against, simply because more individuals are found in high-fitness than in low-fitness patches. In Appen-

dix II we complement this heuristic argument with a formal demonstration that selection drives dispersal rates toward $\varepsilon = 0$ for both the models considered in this paper.

In seeming contradiction to this result, Hamilton and May (1977) showed that a nonzero rate of dispersal may be advantageous even in a saturated, constant, and spatially homogeneous environment. In their discrete generation model, the population occupies sites, with one adult per site. Offspring present at a site compete equably among themselves to become the next adult at that site. Dispersal is positively selected because, all else being equal, it is better to compete with nonsiblings than with siblings. By contrast, the models considered here and in Hastings (1983) represent population growth with differential equation models. This formalism implicitly assumes that populations within a habitat are large enough for population size to be treated as a continuous vaiable rather than as an integer. In a large population with random interactions among individuals, individuals should interact mainly with nonkin, and the dispersal strategy that increases individual fitness should be favored by selection. Comins *et al.* (1980) generalized the original Hamilton–May model to allow for $K > 1$ adults at each site. They demonstrated that without extrinsic disturbances leading to site vacancies, as $K \to \infty$ the ESS approaches a zero rate of dispersal. Our result thus emerges as a limiting case of the Hamilton–May model.

2.3. *The Effect of Rapid Dispersal upon Total Population Size*

We now compare the population size expected under rapid, passive dispersal ($\varepsilon \to \infty$) with the number of individuals sustained when individuals choose habitats optimally (or $\varepsilon = 0$), so that at equilibrium no individual can increase its fitness by moving between habitats. For this comparison we use another geometrical representation of the model. Following Fretwell (1972), in Fig. 2 we plot the per capita growth rate in each patch (ϕ_i) as a function of N_i. If passive dispersal is sufficiently rapid to equalize the numbers of individuals found in the two patches, the average individual should experience a per capita rate of growth that is an arithmetic average of its growth rates in the two patches. This average growth rate is represented by dashed lines in the figure. The total population is in equilibrium when its positive rate of growth in one patch just matches its negative rate of growth in the other (the x's in the figure denote the number of individuals per patch at equilibrium).

Under optimal habitat selection, at equilibrium individual fitness is the same in the two habitats, so $\phi_1(N_1^*) = \phi_2(N_2^*) = 0$, or $N_1^* = K_1$ and $N_2^* = K_2$. The average population size per patch is thus $(K_1 + K_2)/2$ (marked by dots in the figure). Comparing this average population size to that expected with rapid dispersal, we see that optimal habitat selection

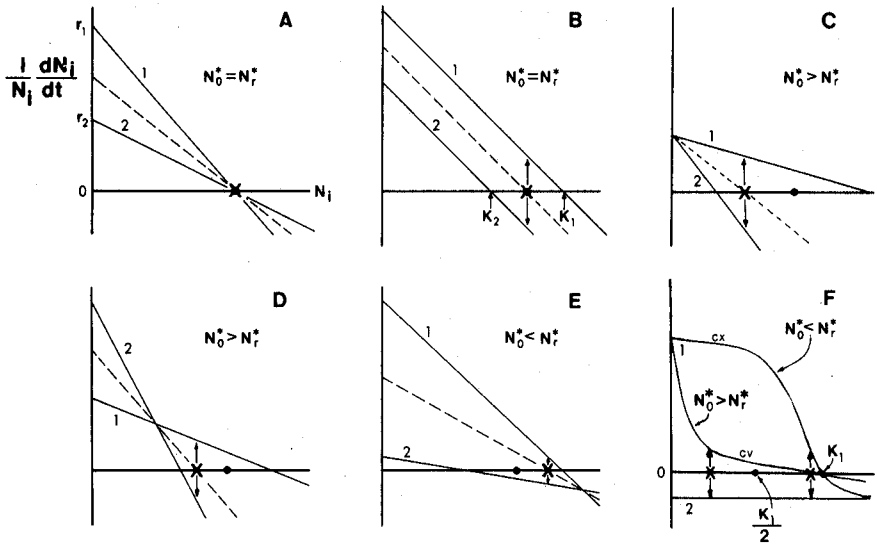

FIG. 2. A comparison of population sizes generated by optimal habitat selection with rapid, passive dispersal. Axes are labeled in A. The solid lines are per capita growth rates in each patch, graphed as a function of the number of individuals in that patch. Given optimal habitat selection, per capita growth rate is zero in both patches when $N_1^* = K_1$ and $N_2^* = K_2$. The average number of individuals per patch, $(K_1 + K_2)/2$, is marked by a dot. If rapid movement equalizes densities, $N_1 = N_2 \equiv N$; the dotted line is the average per capita growth rate, given that densities are equal. Where this line crosses the N_i-axis (marked by x), positive growth in one patch equals negative growth in the other, and the population is in equilibrium. N_0^* is the total population sustained under optimal selection; N_T^* is the total sustained when dispersal is passive and rapid. In figures (A)–(E), growth in each patch is logistic; in (F), growth is nonlogistic. (A) Equal K's, unequal r's; (B) unequal K's, unequal r's, equal slopes $(r_1/K_1 = r_2/K_2)$; (C) unequal K's, equal r's, (D) unequal K's, unequal r's, $r_1/K_1 < r_2/K_2$; (E) Unequal K's, unequal r's, $r_1/K_1 > r_2/K_2$; (F) nonlogistic growth. See text for discussion.

may either leave unchanged (A, B), increase (C, D), or even decrease (E, F) the total number of individuals. The consequences of optimal habitat selection for population size vary in a predictable manner with the shapes and relative positions of the patch-specific growth curves.

As a concrete example, assume that the population in each patch follows a logistic growth law: $\phi_i = r_i(1 - N_i/K_i)$ (see Figs. 2A-E). For $\varepsilon \to \infty$ it can be shown (Freedman and Waltman, 1977) that

$$N_1^* = N_2^* = \frac{N_T^*}{2} = \frac{r_1 + r_2}{r_1/K_1 + r_2/K_2},$$

and that the change in total population size from zero dispersal is

$$K_1 + K_2 - N_T^* = \frac{(K_1 - K_2)(r_2 K_1 - r_1 K_2)}{r_1 K_1 + r_2 K_2}$$

(correcting a typographical error in Freedman and Waltman (1977)). The total population size is unchanged by optimal habitat selection if (1) $K_1 = K_2$ (Fig. 2A), or (2) $r_1/K_1 = r_2/K_2$ (the two growth curves have the same slope; Fig. 2B). Total population size increases under optimal habitat selection if $r_2/K_2 > r_1/K_1$ (recall that we are assuming $K_1 > K_2$) (Fig. 2C and D), and decreases if $r_2/K_2 < r_1/K_1$ (Fig. 2E). Adding δN individuals to patch i decreases the per capita growth rate in patch i by an amount $\delta N(-r_i/K_i)$, so the quantity r_i/K_i measures the strength of density-dependence in patch i. The equilibrial size of a passively dispersing population is biased toward the K of the patch with stronger density-dependence. If this patch happens to be the patch with higher K, the total population size at equilibrium is greater than $K_1 + K_2$ (Fig. 2E). In other words, passive dispersal can lead to a total population greater than the summed carrying capacities of the two patches.

This conclusion holds more broadly than for just the logistic growth model. For instance, in Fig. 2F we contrast two qualitatively different non-logistic growth functions for patch 1; one is convex downward (cx), and the other is concave (cv). Let us assume that the per capita rate of mortality, m, is the same in both patches and is independent of density, and that a resource essential for reproduction is restricted to patch 1. Competition for this essential resource leads to negative density-dependence in the per capita birth rate, $b(N_1)$. The growth rates in the two patches are $\phi_1 = b(N_1) - m$ and $\phi_2 = -m$. Different descriptive assumptions or competitive mechanisms can produce different shapes for $b(N_1)$ (see, e.g., Ayala *et al.*, 1973; Schoener, 1976). With optimal habitat selection, no individuals should be found in patch 2 at all, and the average number of individuals present per patch is $K_1/2$. With passive dispersal, patch 2 is a sink. By inspection of the figure, it can be seen that with a convex growth curve and rapid, passive dispersal, the population equilibrates with each patch containing nearly K_1 individuals, so the total population is nearly twice that expected given an optimal habitat distribution. Conversely, with a concave growth curve the population is in balance at an average number per patch well below $K_1/2$, so the total population is less than K_1.

In Fig. 1 and Appendix I we assumed that each patch in isolation could support a population. Let us briefly consider a source–sink model in which the population declines exponentially in patch 2 ($r_2 < 0$). The growth equation for patch 1 is the same as in model (4), and the growth of patch 2 is described by $dN_2/dt = N_2(r_2 - \varepsilon) + \varepsilon N_1$. At equilibrium, $N_2^*/N_1^* = \varepsilon/(\varepsilon - r_2)$ and $\phi_1(N_1^*) = -\varepsilon r_2/(\varepsilon - r_2)$. Let $r_1 = \phi_1(0)$. With $r_2 < 0$, a non-trivial equilibrium exists if $r_1 > -\varepsilon r_2/(\varepsilon - r_2)$. Two sufficient conditions for the persistence of the population are $r_1 > \varepsilon$ and $r_1 > |r_2|$. If neither of these conditions hold, the species may become locally extinct if its rate of decline in the sink and its dispersal rate are sufficiently large.

With logistic growth in patch 1,

$$N_1^* = K_1\left(1+\frac{r_2}{r_1}\left(\frac{\varepsilon}{\varepsilon-r_2}\right)\right),$$

and

$$N_T^* = N_1^* + N_2^* = K_1\left(1+\frac{r_2}{r_1}\left(\frac{\varepsilon}{\varepsilon-r_2}\right)\right)\left(1+\frac{\varepsilon}{\varepsilon-r_2}\right).$$

If the species exhibits an optimal habitat distribution, at equilibrium all individuals are in patch 1, and the total population is K_1. The effect of passive dispersal on population size is shown in Fig. 3. In this source–sink model, the change in population size resulting from an optimal habitat distribution depends upon the degree of spatial variation in fitness. Passive dispersal into a sink can increase total population size if the absolute magnitude of the per capita rate of decline in the sink is less than the intrinsic rate of increase in the source population. The effect is particularly pronounced if per capita growth rates in the source are a convex function of density (Fig. 2F).

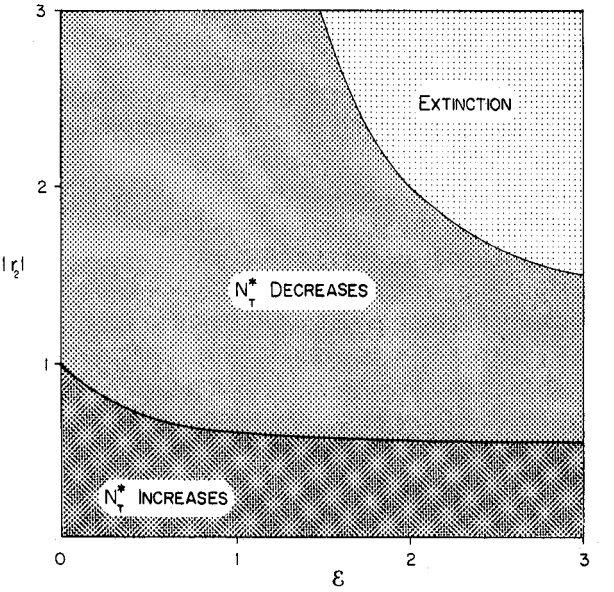

FIG. 3. Effect of dispersal on population size. The model is logistic growth in patch 1, with $r_1 = 1$, exponential decline in patch 2 at rate r_2, and passive dispersal at rate ε. The total population size is compared to K_1, the size expected given an optimal habitat distribution.

3. A Two-Patch Predator-Prey Model

3.1. *Passive Dispersal and Exponential Prey Growth*

To refine our understanding of when selection on dispersal might decrease population size, we will analyze the following model of a predator–prey interaction with predator dispersal into a sink:

$$\begin{aligned} dP_1/dt &= P_1(\mathbf{a}R - C_1) - \varepsilon P_1 + \varepsilon P_2, \\ dP_2/dt &= P_2(-C_2) + \varepsilon P_1 - \varepsilon P_2, \\ dR/dt &= R(r - \mathbf{a}P_1). \end{aligned} \tag{7}$$

The number of predators in patch i is P_i, and R is the number of prey in patch 1. By assuming that the prey is restricted to patch 1, we do not necessarily assume that the predator literally has nothing to eat in patch 2. Instead, we are assuming that the availability of any alternative food in patch 2 is not significantly affected by consumption, and that this food is of such low quality that the predator in patch 2 cannot persist without immigration. The quantity $\mathbf{a}R$ is the predator's functional response (i.e., the instantaneous rate of prey capture by an individual predator, expressed as a function of prey density; Holling, 1959a); $\mathbf{a}R$ is assumed to rise monotonically with R. Predators disperse passively and symmetrically between the source and sink patches at a rate scaled by ε. Without the predator, the prey grows exponentially. (This assumption is relaxed below.) The term $\mathbf{a}RP$ describes both births in the predator and deaths in its prey. This may appear unnecessarily restrictive, for a predator may need to consume multiple prey to produce a single offspring. This could be incorporated by replacing $\mathbf{a}RP$ in the predator growth equation with $b\mathbf{a}RP$, where the constant b converts the consumption of prey into predator births. However, if we measure prey abundance in units of b, we can proceed without this additional parameter.

There are two sources of stability (or instability) in this model–(i) the predator's functional response, and (ii) coupling with the sink habitat. When $\varepsilon = 0$, the dynamics of patch 1 are described by the well-known model: $dP_1/dt = P_1(\mathbf{a}R - C_1)$ and $dR/dt = R(r - \mathbf{a}P_1)$. (For comprehensive reviews of predator–prey models, see Murdoch and Oaten, 1975, and May, 1981.) The equilibrium ($P_1^* = r/\mathbf{a}$ and $R^* = C_1/\mathbf{a}$) is locally stable if $\dot{\mathbf{a}} = d\mathbf{a}/dR > 0$ and unstable if $\dot{\mathbf{a}} < 0$ ($\dot{\mathbf{a}}$ is evaluated at the equilibrium): stability hinges entirely on the character of the functional response. Predator satiation or time invested in handling prey may lead to saturation in the total attack rate with increasing prey density and thus a decreasing rate of capture per prey, or $\dot{\mathbf{a}} < 0$. Conversely, learning and other behavioral changes may make the predator more efficient at capturing prey

as prey density increases. In like manner, if refuges are present in limited supply, proportionally more prey may be vulnerable to predation at higher prey densities. Such mechanisms imply $\dot{\mathbf{a}} > 0$ and stabilize the predator–prey interaction. However, at high densities saturation should occur and $\dot{\mathbf{a}}$ will change sign at some prey density (a Type III functional response). In this case, the local stability of the interaction depends upon the ability of the predator to depress prey to levels sufficiently low that $\dot{\mathbf{a}} > 0$ holds.

Levin (1977) has cautioned that this tight relation between the functional response and the stability of the predator–prey interaction is not a general property of predator–prey models, for either direct density-dependence within predator or prey or their relative spatial distributions can also influence stability. Elsewhere (Holt, 1984, Appendix) I have shown that coupling with a sink habitat by passive dispersal stabilizes the neutrally stable Lotka–Volterra model ($\dot{\mathbf{a}} = 0$). The sink in a sense provides a source of time-lagged recruitment for the predator population in patch 1. This effect is maintained in the more general model (7). The details of the local stability analysis are presented in Appendix III. Not surprisingly, the sign and magnitude of $\dot{\mathbf{a}}$ figure importantly. The qualitative conclusions can be summarized as follows.

(1) $\dot{\mathbf{a}} \geqslant 0$ for all R^*: If in an isolated patch the interaction is stabilized by the predator's functional response, or is neutrally stable, coupling to a sink by passive dispersal produces a stable system.

(2) $\dot{\mathbf{a}} < 0$. The sink stabilizes the intrinsically unstable interaction in patch 1 if (i) the strength of the destabilizing trend in the functional response, as measured by $|\dot{\mathbf{a}}|/\mathbf{a}^2$, is sufficiently small; (ii) the patches are partially coupled (i.e., neither $\varepsilon \cong 0$, nor $\varepsilon \to \infty$); (iii) prey productivity (r) and predator mortality (C_1 and C_2) are sufficiently low. These qualitative results are stated more precisely and proven in Appendix III.

As a concrete example, let $\mathbf{a}$ be the disk equation of Holling (1959b): $\mathbf{a} = a'/(1 + a'hR)$. The parameter a' is the rate of prey capture per unit search time, and h is the time required to subdue and consume a single prey individual, during which other prey cannot be sought. Figure 4 displays several slices through the parameter domain allowing local stability. For local stability, handling times must be small, particularly if r is high. Empirical estimates of h for insect predators are typically well below 0.02 (Hassell, 1981), suggesting that predator sinks could provide a stabilizing influence in some natural predator–prey interactions. I should caution that this stability analysis is strictly local. Numerical studies suggest that for the disk equation, the equilibrium may not be globally stable against large perturbations for parameter choices near the threshold line separating parameter regions of local stability from regions of local instability.

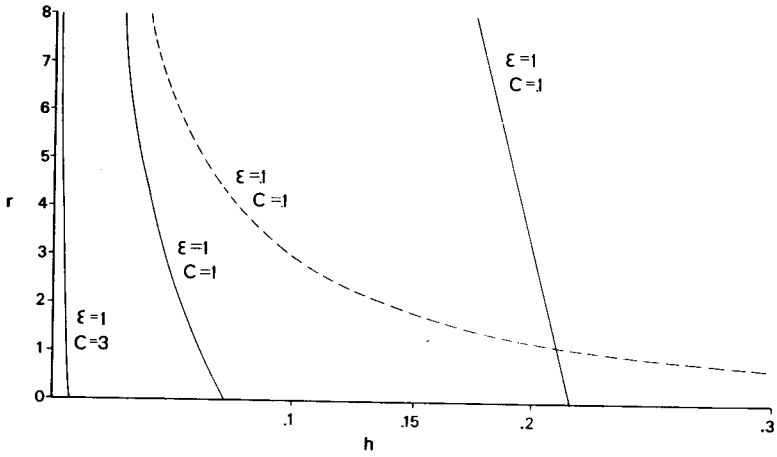

FIG. 4. Local stability domains in the predator–prey model (7), with the "disc" equation for **a**. It is assumed that $C_1 = C_2 \equiv C$. The system is locally stable to the left of each line, and locally unstable to the right. For the solid lines, $\varepsilon = 1$; for the dashed line, $\varepsilon = .1$.

(3) $\dot{\mathbf{a}}$ mixed in sign. The above results assume that the sign of $\dot{\mathbf{a}}$ is independent of R. For a wide enough range in R, it seems more likely that the sign of $\dot{\mathbf{a}}$ would shift from positive to negative as R increases, given that $\dot{\mathbf{a}}$ was positive at low prey densities. Coupling to a sink may in this case destabilize an intrinsically stable ($\dot{\mathbf{a}} > 0$) interaction. Prey density rises with increasing ε, and the system may be destabilized because prey density leaves the region within which the functional response is fundamentally stabilizing and becomes high enough for the functional response to be destabilizing. This effect is reminiscent of the "diffusive instability" observed in some reaction–diffusion models (Levin, 1976; Okubo, 1980).

The crucial feature of this model is that prey are consumed by only a fraction of the total predator population, a fraction that is not a fixed proportion but is instead a variable driven by the system's dynamics. This additional degree of freedom may permit the system to dampen oscillations that otherwise would be caused by a destabilizing functional response. Dispersal into patch 2 reduces the effective predator growth rate when prey are abundant and the predator is increasing in numbers. Conversely, dispersal from patch 2 back into patch 1 provides a "buffer" reducing the effective rate of decline of the predator in patch 1 when prey are rare.

3.2. *Optimal Habitat Distribution*

(a) *The direction of natural selection.* It is clear that if the predator–prey interaction in patch 1 is stable at a point equilibrium, the only evolutionarily stable habitat distribution is for all predators to be in

patch 1. A predator's fitness in patch 1 is $F_1 = \mathbf{a}R - C_1$ and in patch 2 is $F_2 = -C_2$. At equilibrium, $aR^* = C_1 + \varepsilon C_2/(\varepsilon + C_2)$, hence $F_1 \geqslant 0 > F_2$, and selection should favored reduced ε. This prediction can be borne out by an explicit analysis of competition between predators differing only in dispersal rate (see Appendix II). The only evolutionarily stable (Maynard Smith, 1983) rate of dispersal is $\varepsilon = 0$.

(b) *Effect on the size of the predator population.* In this model, the evolution of an optimal habitat distribution by predators tends to minimize the number of predators present at equilibrium. The total number of predators is $P_T^* = P_1^* + P_2^* = (r/\mathbf{a})(1 + \varepsilon/(\varepsilon + C_2))$. With a linear functional response, $\mathbf{a}$ is constant and P_T^* declines monotonically as ε diminishes. With a saturating Type II functional response, as ε decreases, R^* grows smaller and $\mathbf{a}$ increases; the change in per prey attack rates accentuates the decrease in P_T^*. With an accelerating functional response ($\dot{\mathbf{a}} > 0$), the terms $r/\mathbf{a}$ and $(1 + \varepsilon/(\varepsilon + C_2))$ change in opposite directions with diminishing ε, so the overall effect of the evolution of an optimal habitat distribution on population size is not clear. However, for most attack models the net effect appears to be in the same direction. For example, consider the attack model $\mathbf{a} = \beta R^X$, where $\beta > 0$ and $X \geqslant 0$, and let $C_1 = C_2 \equiv C$. At equilibrium, $P_T^* = rC^{-\mu'}\beta^{-\mu}(1 + \varepsilon/(\varepsilon + C))^{\mu}$ ($\mu = (1 + X)^{-1}$ and $\mu' = X(1 + X)^{-1}$), and we see that once again P_T^* declines monotonically with decreasing ε.

For an intuitive understanding of why this occurs, note that at equilibrium the fraction of the total number of predators found in patch 1 is $d \equiv (1 + \varepsilon/(\varepsilon + C_2))^{-1}$. The attack rate of a predator in patch 1 is $\mathbf{a}R$ and in patch 2 is 0. The average attack rate per prey, per predator, is thus $\tilde{a} \equiv ((d)\,\mathbf{a}R + (1 - d)\,0)/R$, or $\tilde{a} = \mathbf{a}d$. Hence, $P_T^* = r/\tilde{a}$. The evolution of an optimal habitat distribution amounts to an increase in the average attack rate; this short-term increase in individual predator fitness eventually reduces the number of predators present at equilibrium, because each predator can garner a larger fraction of the prey's per capita productivity.

(c) *Effect on stability.* If predation dispersal into a sink stabilizes an intrinsically unstable interaction, optimal habitat choice by individual predators necessarily destabilizes the predator–prey interaction. In model (7), this generates oscillations of increasing amplitude, ultimately leading to predator extinction.

A numerical example is shown in Fig. 5. Following a large perturbation, a clone of predators with $\varepsilon = 1$ returns to equilibrium through a succession of damped oscillations. A second clone of predators with $\varepsilon = 0$ then invades and quickly replaces the original clone; the stabilizing influence of the sink wanes, the population goes through oscillations of increasing size, and eventually both the predator and its prey for all practical purposes become

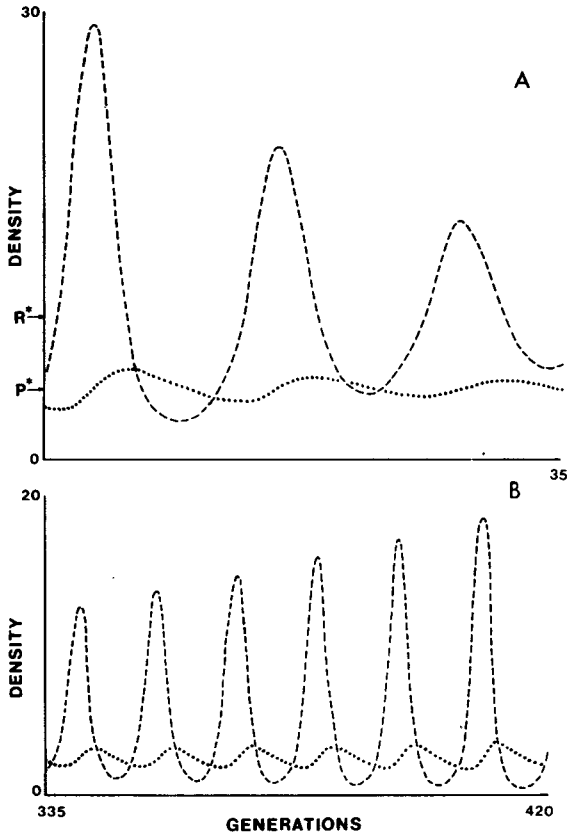

FIG. 5. Numerical example of optimal habitat selection destabilizing a predator–prey interaction. Model (7) is used with the disk equation for **a**. In (A), the predator (dotted line; summed numbers over both habitats) and its prey (dashed line) show oscillatory convergence to equilibrium following a perturbation. The parameters are: $a = 1$, $h = .001$, $\varepsilon = 1$, $C_1 = C_2 = 0.1$, $r = 2.5$, and $b = 0.2$. The initial conditions are $P_1 = 2$, $P_2 = 1.5$, and $R = 5$. After equilibrium is reached, a second clone of predators with $\varepsilon = 0$ (but otherwise identical to the resident) invades and eventually displaces the first clone (not shown). In B, this second clone has become fixed, and the predator and its prey go through oscillations of increasing amplitude, eventually leading to extinction. The numerical solutions were obtained with a modified Runga–Kutta technique, using the DVERK routine in the International Mathematical and Statistical Library.

extinct. The idea that selection may undermine the stability of a predator–prey interaction is not new (e.g., Rosenzweig, 1973; Gilpin, 1975). The novel twist here is that this destabilization may occur because of the evolution of optimal habitat selection by the predator.

(d) *The effect of self-damping in the prey.* We have assumed that there is no density-dependence in the prey other than that exerted through the predator's functional response. Self-limitation in the prey contributes an additional stabilizing factor to the system. Moreover, if the predator in patch 1 when isolated underexploits its prey, optimal habitat selection may increase the size of the total predator population. This can easily be seen if we allow the prey in model (3) to have logistic growth, $dR/dt = R(r - R/K - \mathbf{a}P)$, and assume $\mathbf{a}$ to be constant. To simplify matters, let $C_1 = C_2 = C$, and define $\hat{R} = C/\mathbf{a}$ (the equilibrium number of prey when $\varepsilon = 0$). With these assumptions, $P_T^* = (r/\mathbf{a})(1 + \varepsilon/(\varepsilon + C))(1 - R^*/K)$, where $R^* = (C + \varepsilon C/(\varepsilon + C))/a$. $\partial P_T^*/\partial\varepsilon > 0$ if $\mathbf{a}K > 2(C^2 + 2\varepsilon C)/(\varepsilon + C)$. The right side of this inequality increases monotonically with ε, rising from $2C$ to $4C$. Hence, $\partial P_T^*/\partial\varepsilon < 0$ for all ε if $K/2 < \hat{R}$. Prey production is maximal at $R^* = K/2$. If the predator, when restricted to the source patch, is unable to depress prey numbers below $K/2$, it suffers a reduction in abundance if its average attack rate is lowered by spillover into a sink habitat. Indeed, as $\varepsilon \to \infty$, $P_T^* \to 0$. In this case optimal habitat selection increases P_T^*, and a suboptimal predator may not be able to persist at all. By contrast, if $K/4 > \hat{R}$, $\partial P_T^*/\partial\varepsilon > 0$ at all ε, and optimal habitat selection necessarily lowers P_T^*. In the intermediate case, $K/2 > \hat{R} > K/4$, the evolution of lower dispersal rates reduces P_T^* only over a range of values of ε. In short, optimal habitat selection tends to reduce the population size of efficient predators, and to increase the population size of inefficient predators.

Stable limit cycle behavior may result if an efficient predator with a destabilizing functional response interacts with a self-regulating prey population (May, 1975). This might select for labile habitat selection by the predator. In patch 1, predator fitness ($F_1 = \mathbf{a}R - C_1$) varies with R; in patch 2, predator fitness is constant ($F_2 = -C_2$). An unbeatable movement strategy is: if $\mathbf{a}R - C_1 > -C_2$, forage in patch 1; if $\mathbf{a}R - C_1 < -C_2$, hold out in patch 2. A necessary condition for the predator to move to patch 2 is $C_2 < C_1$. So if the patch with prey is also a riskier place to be, fluctuations in prey numbers (including those produced by the predator–prey interaction itself) may select for switches in habitat use by the predator. The ultimate effect of this on population size and stability is not at all obvious.

Passive coupling with a sink may therefore increase the total number of predators sustained by a prey population and stabilize an otherwise unstable predator–prey interaction. The evolution of an optimal habitat distribution eliminates the predator sink, may decrease predator numbers, and may even destroy the stability of the system. As in the two-patch density-dependent growth model analyzed earlier, an optimal habitat distribution for individuals does not as a rule maximize total population size.

5. Discussion

There are many ways in which Darwinian selection can produce evolutionary change inimical to a population or species as a whole. For example, density-independent or density-dependent selection restricted to a single stage in a complex life cycle may decrease total population size (Charlesworth, 1980; Prout, 1980), and feedback from other species may indirectly reduce population numbers (Levins, 1975; Roughgarden, 1979). We have seen that in two distinct models, the evolution of an optimal habitat distribution may decrease the total number of individuals sustained by the environment. However, it is important to remember that it may also increase or leave unchanged population size. In the predator–prey model with logistic prey growth, an optimal habitat distribution of efficient predators tends to reduce their population size: by contrast, such a distribution increases the numbers of inefficient predators. In the two-patch density-dependent growth model, selection on dispersal can decrease population size below the size sustained with passive dispersal if there is strong, negative density-dependence in the patch with higher K, whereas an increased population results if density-dependence is stronger in the low K patch. This variety in potential outcomes makes it more difficult to predict how the evolution of dispersal might influence the size and stability of natural populations.

In agreement with these results, other analyses of the evolution of dispersal in a spatially homogenous environment have concluded that optimal dispersal rates do not necessarily maximize population size (cf. Hamilton and May, 1977; Comins *et al.*, 1980; Motro, 1982; Hastings, 1983). These studies buttress our findings and suggest that selection on dispersal may often reduce population size. Gadgil (1975) observed that in a temporally constant but spatially variable environment, selection favors low dispersal rates. He also claimed that dispersal typically depressed the total population size of a species. If so, the evolution of low dispersal rates would tend to increase population size. In his analysis he used discrete-time logistic models in which K varied among patches, but r did not. In the logistic model discussed above, at high ε and with $r_1 = r_2$, $N_T^* < K_1 + K_2$; this matches Gadgil's result. In the more general case where r's as well as K's vary spatially, we have seen that random dispersal may also increase or leave unchanged total population size. Vance (1980) explored logistic growth in a patchy environment and observed that dispersal tended to increase population size. In contrast to our models, in Vance's model different patches have identical deterministic properties and differ only in the chance vicissitudes of environmental stochasticity. An interesting task for future work will be to explore the relation between dispersal and population size in models that combine temporal and spatial heterogeneity.

Asmussen (1983) has recently analyzed a discrete-time two-patch density-dependent haploid selection model with dispersal between the patches and showed that the evolution of reduced dispersal rates increased population size. In this model it is assumed that two genotypes are present, each respectively more fit (higher K) in one of the two patches. By contrast, in our models individuals are assumed to differ only in their propensity to move among habitats. The model of Asmussen (1983) combines elements of both local selection (i.e., selection resulting from within-patch differences in fitness) and dispersal selection. Elsewhere (Holt, 1983b) I have discussed the influence of one-way immigration on the size and stability of peripheral populations. In a haploid model in which residents were genetically different from immigrants and had a higher carrying capacity, as long as a polymorphism persisted the peripheral population was shown to equilibrate at the higher K of the locally favored allele. This result complements that of Asmussen (1983). The population-level consequences of selection acting upon dispersal and habitat selection are thus quite sensitive to the existence of spatially patterned genetic variation within a species.

The effects discussed above occur when the dynamics of neighboring patches are coupled by short-distance dispersal. At larger spatial scales, habitat selection can also affect the geographic range occupied by a species in several distinct ways, both to increase and to decrease it. Consider first some ways in which optimal habitat selection might permit a species to be found over a broader geographical range than would suboptimal habitat use. In both of the models treated above, if habitats of greatly varying quality are closely juxtaposed, a passive disperser may have a negative per capita growth rate at all densities and thus go extinct. By contrast, an optimal habitat selector should persist and equilibrate at the carrying capacity of those patches in which its fitness is positive at low densities. In like manner, consider the effects of a local disturbance which drives a population in a spatially variegated environment to low numbers but does not cause its extinction, possibly because some individuals ride out the perturbation in refuges. If following the population crash individuals tend to move to habitats in which they have the highest per capita growth rate, the population as a whole will grow faster than if individuals were to move at random among habitats. This enhancement of a population's ability to rebound from low numbers could play a significant role in population persistence in fluctuating environments. At a species border, where population sizes are typically low, such behavior could be of particular importance in allowing a species to persist. Optimal habitat selection may therefore permit a species to persist in the face of substantial local spatial or temporal heterogeneity. Such a species might have the capacity to exist over a much

wider geographical range than could a species with passive dispersal behavior.

On the other side of the coin, optimal habitat selection may for other reasons greatly restrict the geographical range of a species. Many biogeographers believe that the current ranges of species are determined in large measure by sporadic (or even unique) dispersal episodes (see review in Brown and Gibson, 1983). In a temporally constant environment, habitat selectors should be poor long-distance colonizers. In the "ideal free distribution" (Fretwell, 1972) predicted by habitat selection theory, no individuals should be found in habitats where their expected fitness is less than one. This implies that relatively narrow bands of unsuitable habitat can block the spread of a species. Moreover, if a physical disturbance leads to local extinction, the paucity of potential colonists could imply a long time before recolonization. By contrast, with passive dispersal, source populations generate a steady supply of potential colonists traversing unsuitable habitats, colonists which are available to colonize new habitable areas or to replace extinct populations.

Finally, natural selection might act differently in a population with optimal habitat selection than it does in a population with suboptimal dispersal behavior. In the former, habitats in which per capita growth rates are negative should be avoided entirely, and generalist phenotypes will tend to be replaced by specialists (Rosenzweig, 1981). If certain habitats are entirely avoided, selection cannot improve the ability of the species to utilize those habitats. In a population with suboptimal habitat selection, natural selection may act to improve the ability of individuals to survive and reproduce in habitats which initially cannot sustain a population without regular immigration. Over evolutionary time, a species with suboptimal dispersal behavior might be expected to become more tolerant of spatial variation in habitat quality, and might better be able to track shifts in the relative abundance of alternative habitat types. This should tend to decrease the selective advantage of optimal habitat selection, because differentials in fitness across habitats are reduced. By contrast, as selection hones the ability to utilize a preferred habitat at the expense of the ability to utilize other habitats, the selective advantage of optimal habitat selection correspondingly increases. This sets up a positive feedback between natural selection for habitat selection behavior and selection for phenotypic specialization to particular habitats. Optimal habitat selection may lock a species into habitat specialization; nonoptimal habitat selection, which may follow from constraints preventing the perfection of dispersal behavior by natural selection, may permit a species to be labile in its habitat utilization over evolutionary time. This could ultimately allow a species to be found

over a broader geographical range than a species which was specialized *ab initio* and could freely elect to remain canalized to the habitat in which it was initially most fit. Natural selection, by favoring habitat selection by individuals, may thus have the long-term evolutionary consequence of restricting the geographical range eventually occupied by a species.

APPENDIX I

Properties of Density-dependent Growth in Two Patches Linked by Passive Dispersal

1. *Isoclines and Equilibria of Model* (1)

Freeman and Waltman (1977) discuss some formal properties of this model when ε is small.

We first show that $K_2 < N_2^* \leqslant N_1^* < K_1$ for all $\varepsilon > 0$ and $K_2 < K_1$, with the equality holding only in the limit $\varepsilon \to \infty$. Assume that $\phi_i(N_i) < 0$ for all $N_i > K_i$, and $|d\phi_i/dN_i| < \infty$. We argue by contradiction. Assume $N_1^* < N_2^*$ and let ϕ_i denote $\phi_i(N_i^*)$. Substituting from (4) into (5), at equilibrium $2N_1^*\phi_1/(N_1^* - N_2^*) = 2\varepsilon > 0$. Thus $\phi_1 < 0$, which requires that $K_2 < K_1 < N_1^* < N_2^*$. But this implies in turn that $\phi_2 < 0$, so $N_1^*\phi_1 + N_2^*\phi_2 < 0$ and the total population cannot be in equilibrium. Therefore, $N_1^* > N_2^*$, $\phi_1 > 0$ (i.e., $N_1^* < K_1$), and $\phi_2 < 0$ (i.e., $N_2^* > K_2$). Hence, $K_2 < N_2^* < N_1^* < K_1$. Equation (3) at equilibrium may be written $(N_1^*\phi_1 - N_2^*\phi_2)/2\varepsilon + (N_2^* - N_1^*) = 0$, so in the limit $\varepsilon \to \infty$, $N_2^* = N_1^*$.

The isocline of species 1 in the phase plane (N_1, N_2) is described by $G_1(N_1, N_2) = \phi_1(N_1) + \varepsilon(N_2/N_1 - 1) = 0$, and the isocline of species 2 by $G_2(N_1, N_2) = \phi_2(N_2) + \varepsilon(N_1/N_2 - 1) = 0$ (see Fig. 6). Because ϕ_i decreases monotonically with increasing N_i, the isoclines have positive slope. The isocline G_i intersects the N_i-axis at a density $N_i < K_i$ and intersects the 45° line $N_1 = N_2$ at $N_i = K_i$. The isoclines intersect only once. To see this, consider the straight line $N_2 = aN_1$ connecting the origin and a given equilibrium (N_1^*, N_2^*) (the dashed line in Fig. 6). Along this line, $G_1 > 0$ if $N_1 < N_1^*$, and $G_1 < 0$ if $N_1 > N_1^*$. Similarly, along this line $G_2 > 0$ if $N_2 < N_2^*$ and $G_2 < 0$ if $N_2 < N_2^*$. In other words, both isoclines intersect the line $N_2 = aN_1$ just once. Were there a second point equilibrium along the isocline $G_1 = 0$, then on geometrical grounds it is clear that the isocline $G_2 = 0$ would have to cross $N_2^* = aN_1^*$ a second time (e.g., dotted line in Fig. 1B). Since this does not happen, the non-trivial equilibrium must be unique.

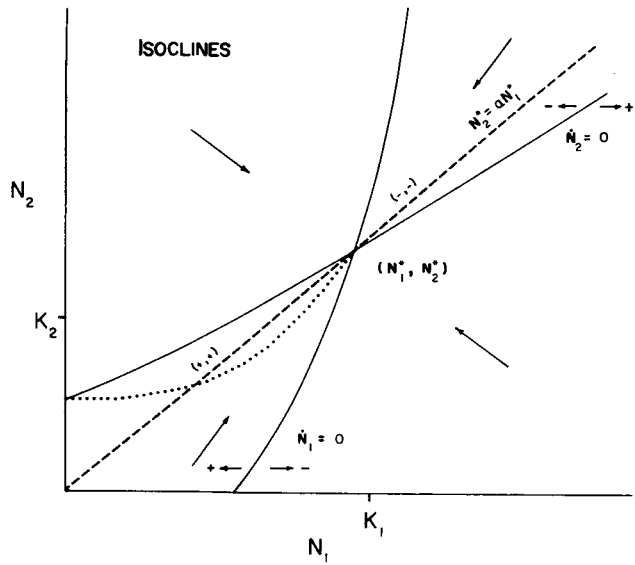

FIG. 6. Zero-growth isoclines for two-patch, density-dependent growth with passive dispersal between patches. See Appendix I for details. The long arrows show trajectory directions in the positive quadrant.

2. *Local stability*

The Jacobian of system (1) is

$$\begin{pmatrix} \phi_1 + N_1^* \dot{\phi}_1 - \varepsilon & \varepsilon \\ \varepsilon & \phi_2 + N_2^* \dot{\phi}_2 - \varepsilon \end{pmatrix}$$

The characteristic equation is $\lambda^2 + a_1 \lambda + a_2 = 0$, where

$$a_1 = -[(\phi_1 - \varepsilon + N_1^* \dot{\phi}_1) + (\phi_2 - \varepsilon + N_2^* \dot{\phi}_2)],$$

and

$$a_2 = (\phi_1 - \varepsilon + N_1^* \dot{\phi}_1)(\phi_2 - \varepsilon + N_2^* \dot{\phi}_2) - \varepsilon^2.$$

From (1), at equilibrium $\phi_1 - \varepsilon = -\varepsilon N_2^*/N_1^*$, and similarly $\phi_2 - \varepsilon = -\varepsilon N_1^*/N_2^*$. After substitution, we find that $a_1 > 0$ and $a_2 > 0$. Therefore, the equilibrium is locally stable. Moreover, because the separatrix $a_1^2 - 4a_2 > 0$, the equilibrium is a stable node. No closed trajectories exist. This may be shown by using the Dulac extension of Bendixson's negative criterion (Andronov *et al.*, 1966; cited in Schoener, 1976). If a multiplier $B(N_1, N_2)$ exists such that (1) B is continuous and has continuous derivatives for all positive N_1 and N_2, and (2) $\partial/\partial N_1(BdN_1/dt) +$

$\partial/\partial N_2(BdN_2/dt)$ has constant sign, the Dulac–Bendixson criterion is satisfied. Let $B = 1/N_1N_2$. B is continuous and has continuous derivatives for $N_1, N_2 > 0$. Note that $\partial/\partial N_1(BdN_1/dt) = \dot{\phi}/N_2 - \varepsilon N_1^{-2} < 0$, and similarly $\partial/\partial N_2(BdN_2/dt) = \dot{\phi}_2/N_1 - \varepsilon/N_2^{-2} < 0$. The Dulac–Bendixson criterion holds: there are no limit cycles in this model. Because the trajectories are bounded (see Fig. 6), this ensures global stability.

Appendix II

Evolutionarily Stable Dispersal Rates

An evolutionarily stable strategy (ESS) is defined to be a phenotype that, when near fixation in a population, can resist invasion by alternative phenotypes (Maynard Smith, 1983). A monomorphic ESS is unique if it can always increase when rare relative to other strategies.

Consider first density-independent growth in model (6). With constant F_i, this model is a pair of coupled linear differential equations with constant coefficients. The population in each patch eventually grows exponentially at a rate $\tilde{r}$ determined from the dominant eigenvalue of the coefficient matrix,

$$\begin{pmatrix} F_1 - \varepsilon & \varepsilon \\ \varepsilon & F_2 - \varepsilon \end{pmatrix}.$$

Let $Q = (F_1 - F_2)/2$. The realized asymptotic rate of growth in both patches is a function of the dispersal rate:

$$\tilde{r}(\varepsilon) = \frac{F_1 + F_2}{2} - \varepsilon + (\varepsilon^2 + Q^2)^{1/2}.$$

Because $\partial\tilde{r}/\partial\varepsilon < 0$ if $F_1 \neq F_2$, we can conclude that if two clones are identical except for dispersal rates, the one with lower ε grows at a faster rate and so is favored by natural selection. If $F_1 > F_2$, eventually patch 1 contains more individuals than patch 2. With passive dispersal, more individuals leave patch 2 than return, so there is a net flux of individuals from high-fitness to low-fitness environments. This is why passive dispersal is disadvantageous in a spatially heterogeneous environment.

This may all seem to be beside the point, inasmuch as patch-specific fitnesses are not constants in models (1) and (7). But, the argument we have just sketched suffices to determine evolutionarily stable dispersal rates in these models, too. For instance, in the two-patch predator–prey model (7), alternative dispersal strategies interact indirectly through the prey. To

model competition between asexual clones of predators that differ only in dispersal rates we expand model (7) as follows (the hat denotes the second clone):

$$dR/dt = R(r - \mathbf{a}(P_1 + \tilde{P}_1)),$$
$$d\tilde{P}_1/dt = \tilde{P}_1(\mathbf{a}R - C_1 - \varepsilon') + \varepsilon'\tilde{P}_2,$$

and

$$d\tilde{P}_2/dt = \tilde{P}_2(-C_2 - \varepsilon') + \varepsilon'\tilde{P}_1;$$

the equations for dP_1/dt and dP_2/dt are unchanged. Setting all five equations equal to zero, it is readily shown that a polymorphic equilibrium with both predators present in positive numbers cannot exist if $\varepsilon \neq \varepsilon'$. Assume that (P_1^*, P_2^*, R^*) is locally stable. The dynamics of the second clone, when it is sufficiently rare to leave unchanged the equilibrium number of prey, is described by

$$d\tilde{P}_1/dt = \tilde{P}_1(\mathbf{a}R^* - C_1 - \varepsilon') + \varepsilon'\tilde{P}_2$$
$$d\tilde{P}_2/dt = \tilde{P}_2(-C_2 - \varepsilon') + \varepsilon'\tilde{P}_1$$

where $\mathbf{a}R^* = (1 + \varepsilon + \varepsilon C_2/(\varepsilon + C_2))$. This pair of linear differential equations is formally equivalent to (6), with $F_1 = \mathbf{a}R^* - C_1$ and $F_2 = -C_2$. We therefore know that the invading clone has a higher growth rate than the resident if $\varepsilon' < \varepsilon$, as this implies $\tilde{r}(\varepsilon') > \tilde{r}(\varepsilon)$. At equilibrium, however, the density of prey in patch 1 and the densities of the resident predator in the two patches are adjusted so that the resident predator's realized rate of growth is $\tilde{r}(\varepsilon) = 0$. Hence, the invasion succeeds if $\varepsilon' < \varepsilon$, and fails if $\varepsilon' > \varepsilon$. It follows that $\varepsilon = 0$ is the only ESS.

This basic approach generalizes to any model which leads to a stable equilibrium. A resident population with a rate of dispersal ε achieves equilibrium because of direct density-dependence, changes in resource levels, etc. Its patch-specific fitnesses, F_i, therefore, must be such that $\tilde{r}(\varepsilon) = 0$. When the invader differing only in ε is rare enough, its patch-specific fitnesses are independent of its own density. Therefore, the initial stages of invasion can be represented by (6), with constant F_i, and the invader increases in numbers if $\tilde{r}(\varepsilon') > 0$, which requires that $\varepsilon' < \varepsilon$. This argument applied to (7) shows that $\varepsilon = 0$ is the only ESS given density-dependent growth in the patches. Hastings (1983) uses a more sophisticated analysis to show that this result is general for a single species distributed over n patches. The argument presented here suggests that an identical result holds in multispecies models, so long as the initial patch-specific fitnesses of the invader can be represented to a first approximation

as constants, and the invader and resident differ only in their dispersal rates. James Bull (personal communication) has derived a similar result in a discrete-time growth model.

As noted in the text, this result may appear to contradict the finding by Hamilton and May (1977; see also Comins *et al.* 1980; and Motro, 1982) that high rates of dispersal are evolutionarily stable even in temporally stable environments. One difference between their model, and the models of Hastings (1982) and this paper, results from a key difference in how dispersal is assumed to influence local population size. In the Hamilton–May model, the sites at which adults live have a fixed carrying capacity (e.g., one adult per site) and juveniles compete for access to that site. Thus, adult density at a site is largely independent of the rate at which juveniles disperse into or out of that site. By contrast, in our models dispersal out of a site inevitably reduces equilibrial density.

Appendix III

Stability of the Two-Patch Predator–Prey Model

The equilibrium densities of model (7) are found from

$$P_1^* = r/\mathbf{a}, \qquad P_2^* = P_1^* \varepsilon/(\varepsilon + C_2), \qquad \text{and} \qquad R^* = \psi/\mathbf{a},$$

where $\psi = C_1 + \varepsilon C_2/(\varepsilon + C_2)$. A full solution requires an explicit function for $\mathbf{a}$. Note, however, that relative densities (e.g., the ratio P_1^*/P_2^*) are independent of $\mathbf{a}$. The local stability of model (7) is determined from the eigenvalues of the Jacobian:

$$\begin{pmatrix} \mathbf{a}R - C_1 - \varepsilon & \varepsilon & \mathbf{a}P_1 + RP_1\dot{\mathbf{a}} \\ \varepsilon & -\varepsilon - C_2 & 0 \\ -\mathbf{a}R & 0 & r - \mathbf{a}P_1 - P_1 R\dot{\mathbf{a}} \end{pmatrix} \tag{A1}$$

The population sizes and $\dot{\mathbf{a}}$ are evaluated at equilibrium. Let $\Delta \equiv \varepsilon + C_2 + \varepsilon^2/(\varepsilon + C_2)$, and $q \equiv \dot{\mathbf{a}}/\mathbf{a}^2$. The quantity q is a measure of the effect of the functional response on stability (stabilizing if $q > 0$, destabilizing if $q < 0$). If the eigenvalues of (A1) have negative real parts, the equilibrium is locally stable.

The characteristic equation of (A1) is a cubic, $\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0$, where $a_1 = \Delta + r\psi q$, $a_2 = \psi r[1 + (\Delta + \psi)q]$, and $a_3 = \psi r(\varepsilon + C_2)(1 + \psi q)$. Define $a_4 \equiv a_1 a_2 - a_3$. The Routh–Hurwitz criterion for each eigenvalue to have negative real part is for $a_i > 0$, $i = 1$ to 4 (May, 1975). If $a_2 > 0$, it

follows that $a_3 > 0$, so the conditions that must be checked for stability are $a_1 > 0$, $a_2 > 0$, and

$$a_4 = \psi r[\varepsilon^2/(\varepsilon + C_2) + q[\Delta(\Delta + \psi) + \psi r - \psi(\varepsilon + C_2)] + q^2\psi r(\Delta + \psi)] > 0$$

We consider separately (i) $\dot{\mathbf{a}} \geqslant 0$, and (ii) $\dot{\mathbf{a}} < 0$.

(i) $\dot{\mathbf{a}} \geqslant 0$. It is readily seen that $a_1 > 0$, $a_2 > 0$, and $a_4 \geqslant 0$; the equality in the latter case holds only when $\dot{\mathbf{a}} = 0$ and $\varepsilon = 0$, which describes an isolated patch with a linear functional response—the neutrally stable Lotka–Volterra model.

(ii) $\dot{\mathbf{a}} < 0$. We examine in turn the effect of:

(a) The magnitude of $\dot{\mathbf{a}}$. Each of the stability criteria is satisfied if $\varepsilon > 0$, and if $|q|$ is sufficiently small. Conversely, from $a_2 > 0$, a necessary upper bound for $|q|$ is $(\Delta + \psi)^{-1}$.

(b) The degree of patch coupling. As $\varepsilon \to 0$, the dynamics of the two patches become progressively decoupled, and the inherent instability of patch 1 should emerge at sufficiently low ε. A formal demonstration of this follows from the observation that as $\varepsilon \to 0$, $a_2 > 0$ and $a_4 > 0$ cannot both hold. The equilibrium is always locally unstable if ε is sufficiently small.

Conversely, as $\varepsilon \to \infty$, the two predator populations merge into a single population, one in which the effective encounter rate with prey is reduced by one-half; if the functional response is destabilizing, the merged populations should be unstable. For a formal proof, note that $a_2 < 0$ if $\varepsilon > \frac{1}{2}(|q|^{-1} - C_1 - C_2)$.

(c) The magnitude of r. Prey productivity influences stability through its effect on P_1^* and P_2^*. R^* is independent of r; $\mathbf{a}$ is a function of R and thus is also independent of r. From $a_1 > 0$, an upper bound on r is $\Delta/\psi\,|q|$. The expression $a_4/\psi r$ is linear in r and has negative slope (if $a_2 > 0$). For stability to occur for any r, it must therefore be true that $a_4 > 0$ when $r \cong 0$. From this, we have

$$|q| < (\psi + (\varepsilon + C_2)(\Delta/\varepsilon)^2)^{-1} \tag{A2}$$

as a necessary condition for local stability. At low rates of density-independent mortality, relative to dispersal rates, this inequality is approximately $|q| < (4\varepsilon)^{-1}$.

If $a_4 > 0$, this implies that $a_2 > 0$ and, since r is assumed to be small, $a_1 > 0$. This demonstrates that if (A2) holds, all $a_i > 0$ for sufficiently low r. Both a_1 and $a_4/\psi r$ decline linearly with r. Given that (A2) holds, the maximum r consistent with $a_4 \geqslant 0$ is

$$r_{\max} = \frac{|q|[\Delta(\Delta+\psi) - \psi(\varepsilon + C_2)] - \varepsilon^2/(\varepsilon + C_2)}{|q|^2 \psi(\Delta+\psi) - |q|\psi}. \tag{A3}$$

It can be shown that $r \leqslant r_{\max}$ implies $a_1 > 0$.

This leads to the following recipe for assessing stability. For a given ε, C_1, and C_2, find R^* from $\mathbf{a}R^* = C_1 + \varepsilon C_2/(\varepsilon + C_2)$. Evaluate $|q|$ at this R^*. If (A2) is violated, the equilibrium is unstable, independent of r. If (A2) holds, the system is locally stable if $r < r_{\max}$.

The implicit dependence of q on ε, C_1, and C_2 makes it impossible to specify in general the ranges of these parameters consistent with local stability. For the disk equation discussed in the text, however, we have the convenient property that $|q| = h$. Substitution into (A2) and (A3) leads to the conditions illustrated in Fig. 4.

ACKNOWLEDGMENTS

I thank the University of Kansas for its financial support. I appreciate the suggestions of J. Brown, J. Bull, H. Freedman, A. Hastings, H. Horn, and M. Rosenzweig, and the efforts in manuscript preparation by T. Shistar, Coletta Spencer and Jan Elder.

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Printed by the St. Catherine Press Ltd., Tempelhof 41, Bruges, Belgium