

Numerically Exploring Habitat Fragmentation Effects on Populations Using Cell-Based Coupled Map Lattices

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Received September 8, 1997

We examine habitat size, shape, and arrangement effects on populations using a discrete reaction–diffusion model. Diffusion is modeled passively and applied to a cellular grid of territories forming a coupled map lattice. Dispersal mortality is proportional to the amount of nonhabitat and fully occupied habitat surrounding a given cell, with distance decay. After verifying that our model produces the results expected for single patches of uniform habitat, we investigate heterogeneous and fragmented model landscapes. In heterogeneous single-patch systems near critical patch size, populations approach Gaussian spatial distributions with total population constrained by the capacity of the most limiting cell. In fragmented habitat landscapes, threshold effects are more complex and parametrically sensitive. The results from our experiments suggest the following: the ability to achieve persistence in hyper-dispersed patchy habitats by adding similarly fragmented patches requires meeting threshold reproduction rates; persistent metapopulations in which no local population is individually persistent appear when dispersal distances and reproduction rates are both high, but only within narrow parameter ranges that are close to extinction thresholds; successful use of stepping-stone patches to support metapopulation systems appears unlikely for passively diffusing species; elongated patches offer early colonization advantages, but blocky patches offer greater population resilience near extinction thresholds. A common theme running through our findings is that population viability estimates may depend on our ability to determine when population and habitat systems are approaching extinction threshold conditions. © 1999 Academic Press

Key Words: spatial models; dispersal; population persistence; critical patch size; critical patch arrangement; spatial heterogeneity; reaction–diffusion; metapopulations.

INTRODUCTION

Fragmented landscapes often occur as habitable areas of varying size, shape, and dispersion embedded in a matrix that is largely inhospitable. Consequently, the manner in which organisms disperse strongly affects population abundance and distribution in a particular habitat complex. The importance of population diffusion

has led to a concerted effort directed toward the development of spatially explicit models of population dynamics in recent decades. Among the earliest works in this field are the pioneering studies on random dispersal by J. G. Skellam (1951), Kierstead and Slobodkin (1953), and Segel and Jackson (1972). Their reaction–diffusion methods are based on the probabilistic concept of random “walks” (Fisher, 1937) or quasi-random walks undertaken by dispersing organisms. In the aggregate,

these dispersal assumptions lead to deterministic population diffusion models similar to molecular diffusion models from physics (see Pielou, 1977, and Okubo, 1980). In a parallel early effort, metapopulation analysis (Levins, 1970) provided an alternative approach for studying habitat fragmentation effects by focusing on stochastic patch extinction and colonization rates, and the resulting frequency of patch occupancy (e.g., Hanski and Gyllenberg, 1993), rather than modeling dispersal explicitly.

Unfortunately, realistic landscapes of fragmented populations have proven difficult to study by reaction–diffusion and metapopulation analysis methods. Reaction–diffusion modeling typically involves continuous time and space partial differential equations to describe rates of population change (Holmes *et al.*, 1994). While this approach has analytical advantages, continuous-variable calculus imposes important modeling limitations, particularly for fragmented habitats (Levin, 1974). Discrete-space differential equation systems are better suited to modeling diffusion in fragmented habitat, but complicated mathematics have prevented analysis beyond simple patch arrangements (see Allen, 1987). Similarly, metapopulation models typically require highly idealized spatial structure (Hastings and Harrison, 1994).

The spatial limitations of these early methods have led to a variety of new approaches for analyzing fragmented populations, including cellular automata models (e.g., Wolfram, 1984), percolation models (e.g., Gardner *et al.*, 1989), demographic models (e.g., Lande 1987), and others (see Kareiva, 1990, for a review). The diversity of approaches involved, however, has produced inconsistent theoretical results (Hastings and Harrison, 1994) that seem contradictory to conservation biologists (de Roos and Sabelis, 1995). One factor that contributes to this variety in theoretical results is the absence of a single model formulation that simultaneously addresses both intra- and interpatch diffusion for realistic landscapes.

The purpose of this paper is to explore the effects of spatial habitat structure on population persistence, abundance, and distribution using numerical analysis of coupled map lattices (Kaneko, 1993) based on cells rather than on patches. Using this approach to model spatial structure allows us to examine both within-patch and between-patch population dynamics with a single discrete-time, discrete-space, reaction–diffusion model. There are two main contributions from this study. First, with a single model, variations in assumptions about population dynamics are captured parametrically instead of structurally. Second, although most of the

single-patch results we present are fairly obvious from earlier reaction–diffusion work, many of the multi-patch results are not so obvious and have implications for conservation biology and habitat protection.

THE MODELING APPROACH

The extension by Levin (1974), Allen (1987) and others of single-patch reaction–diffusion models to multi-patch formulations converts a single partial differential equation to a system of ordinary differential equations. This patch-level approach allows the examination of habitat complexes, but does not account for variations in population density within each patch. We observe that this procedure can be refined into a method suitable for both single- and multi-patch environments, without loss of intrapatch information, by subdividing patches into smaller units that we call habitat cells. The two-dimensional habitat complexes depicted in Fig. 1, for example, can all be modeled equally well using cell-based reaction–diffusion as long as some degree of diffusivity exists among the cells.

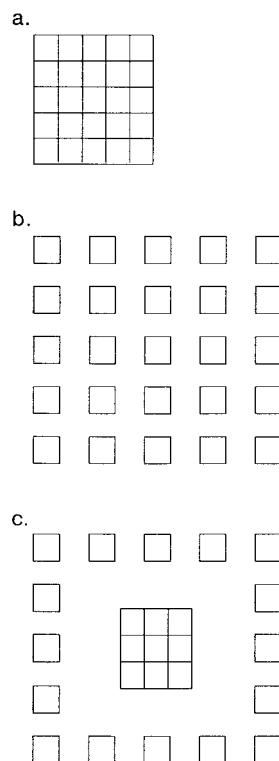


FIG. 1. Three examples showing (a) single patch, (b) hyperdispersed, and (c) mainland–island habitat complexes consisting of 25 habitat cells.

For species that establish and defend distinct breeding territories, those territories define the spatial scale at which reproduction and dispersal processes occur within the population. Discretizing habitat by replacing patches with a network of appropriately sized grid cells converts the habitat complex into a lattice representation based on breeding territories. For more continuously distributed species, any habitat approximation error resulting from the use of cells can be reduced by refining grid resolution in a procedure typical of finite numerical approximation methods.

By further discretizing reaction–diffusion models with respect to time, we convert our cellular-level system of ordinary differential equations into a coupled map lattice (Kaneko, 1993) consisting of an enlarged system of difference equations that offers a more realistic formulation for many species (Levin, 1974; Holmes *et al.*, 1994). Just as cellular models can represent breeding territories more realistically than continuous-space models, discrete time intervals are more appropriate than continuous-time modeling for species with distinct breeding and dispersal periods. For other species, discrete time steps still offer many computational advantages (Cushing, 1988).

In this study, we model populations that exhibit periodic reproduction and diffusion with adults and juveniles dispersing identically as

$$v_{it} = \sum_{j=1}^N [1 + f_j(v_{j,t-1})] v_{j,t-1} g_{ji} \quad \forall i, t, \quad (1)$$

where i and j each index all cells in the complex, v_{it} is the population in cell i at time t , $f_j(v_{j,t-1})$ is the net per capita rate of reproduction within cell j (not accounting for mortality associated with dispersal), and g_{ji} is defined as the proportion of organisms in cell j expected to emigrate to cell i per time step. We derive Eq. (1) and related age- or sex-structured spatial population models from a discrete-space, continuous-time reaction–diffusion model in the Appendix.

Based on previous work (Bever *et al.*, 1997), a typical species in this regard is the black-footed ferret (*Mustela nigripes*). Like many species, ferrets reproduce in the spring of each year, followed in late summer or early autumn by wide-ranging dispersal of many juveniles and adults. Dispersing juveniles mature over the winter to become breeding adults the following spring (Clark, 1989; Seal *et al.*, 1989). Extensions to species with more complex population dynamics are considered under Discussion.

EXPERIMENTS

In the sections that follow, we use Eq. (1) to first investigate the effects of patch size, shape, and heterogeneity on populations occupying a single patch. This is followed by a series of experiments on fragmented habitat complexes. As noted before, fragmented landscapes often occur as habitable areas embedded in a traversable but largely inhospitable matrix. For this exploratory study, we assume passive random-walk dispersal across such landscapes. We use square habitat cells in our examples, but other shapes could easily be used.

The choice of spatial and temporal units requires scale consistency in this reaction–diffusion process. That is, each g_{ji} diffusion parameter should reflect the probability of emigrating from breeding territory j to territory i a full breeding season later by any of a multitude of possible routes. For simplicity, we assume identical diffusion from each cell in any given complex, calculated in the following manner. Organisms disperse on average from the center of each cell outward in uniformly random directions. Dispersal probabilities decline with distance (x) according to

$$p_x(x) = \sigma^{-1} \exp[-(x - \theta)/\sigma]; \quad x > \theta, \sigma > 0$$

as defined by a mean dispersal distance (σ) from the center of the home cell, using a minimum dispersal distance (θ) of zero. Diffusion proportions (g_{ji}) are estimated by numerical approximation over distances and angles defined by the boundaries of each destination cell (indexed by i) relative to the center of each source cell (indexed by j). Using an exponential distribution for dispersal distance (as in Fahrig, 1992) results in a globally connected cellular lattice.

To observe the effects of within-patch heterogeneity clearly and simply, we hold reproduction constant by setting $f_j(v_{j,t-1})$ to a per capita net rate of reproduction r identical for every cell and every time period. In the absence of dispersal, this produces discrete exponential growth up to a constant adult population carrying capacity limit b_i that can be varied across cells by adding the constraint

$$v_{it} \leq b_i \quad \forall i, t. \quad (2)$$

In each time step, cellular populations from Eq. (1) in excess of Eq. (2) are treated as mortality resulting from dispersal into fully occupied territories. This procedure produces a probability of dispersal mortality across a varied landscape that is proportional to the amount of

nonhabitat and fully occupied habitat around a given cell, with distance decay.

Populations Isolated in a Single Patch

Effects of patch size. Using continuous-variable random walk models, Skellam (1951) and others have demonstrated that for a single patch of habitat a critical patch size generally exists, below which theoretical populations are expected to perish. In our examples, this threshold is defined by the combination of patch size, intrinsic rate of population growth (r), and mean dispersal distance (σ , hence all g_{ji}). By varying one parameter while holding the others constant, a critical set of threshold values can be determined. Below the threshold point, total population in the model declines toward zero from any initial population level.

To verify consistency of our model with reaction-diffusion theory, we demonstrate this critical threshold effect by considering a species with a mean dispersal distance (σ) of 2.50 cell-side units, an r -value (r) of 0.495,¹ and a carrying capacity (b_i) of ten organisms per cell (as may be appropriate) for social predators or cooperative breeders). By incrementally increasing the size of square patches, we initially observe a persistent population with a 7×7 cell patch (Fig. 2). This patch is at the critical size threshold for the given reaction-diffusion conditions, meaning that removal of any cell from the patch results in an equilibrium population of zero. Similarly, if mean dispersal is increased to 2.51 (with $r = 0.495$) or if the r -value is decreased to 0.494 (with $\sigma = 2.50$), the equilibrium population drops to zero.

Patch size also affects population growth and resulting density. Population growth curves for square patches of different sizes under otherwise identical conditions are shown in Fig. 3. Initial populations of ten organisms per cell were placed in the nine central cells of each patch so that population declines would be visible for patches below the critical size threshold.

For the 7×7 cell patch near critical threshold conditions, we observe a “ j -shaped” exponential growth curve up to 327.5 population units, at which point the population behaves as though the carrying capacity limit had been reached. Given the constant r -value used in this model, the shape of the growth curve for this patch is not surprising except that the equilibrium population is only about 66% of total expected carrying capacity. It is also noteworthy that about 2,000 time steps are required

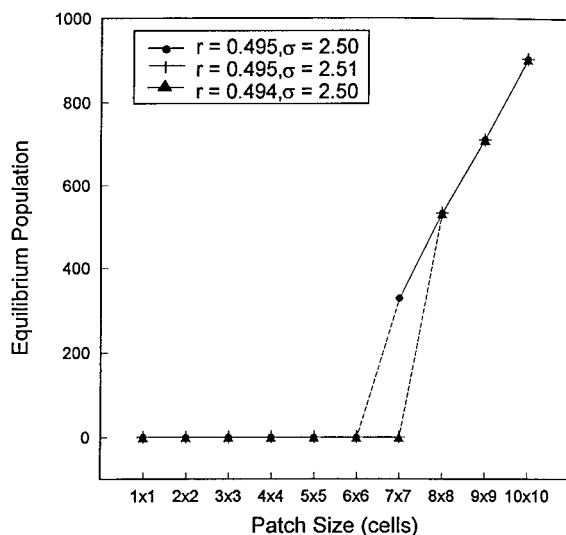


FIG. 2. Equilibrium populations in square patches ranging in size from 1 to 100 cells under three slightly different sets of reaction-diffusion parameter values.

before the resulting upper limit is reached. Consequently, the overall “realized” per capita rate of population growth is about 0.00065 per time step—considerably less than the r -value used in the simulation. This realized rate of growth approaches zero as the critical extinction threshold is approached with greater precision. Below equilibrium in this system, dispersal mortality is entirely due to diffusion into nonhabitat. Cellular capacity only becomes limiting at equilibrium.

Three other effects of patch size are particularly noticeable in Fig. 3. First, as patch size increases above the critical size threshold of 7×7 cells, the patch population growth curves become sigmoid rather than exponential in shape, despite constant net rates of within-cell reproduction, as dispersal mortality results more and more from movement into fully occupied cells. Second, the overall realized rate of population growth increases with patch size, although the realized rate is always less than the parameterized r -value due to the loss of organisms dispersing beyond the patch and perishing. Third, as patch size increases, the resulting equilibrium population more closely approaches total expected patch carrying capacity, reaching about 88% of total carrying capacity in the 9×9 cell patch, and about 92% of carrying capacity in the 11×11 cell patch.

A final patch size effect concerns the distribution of populations within the patch. Our results indicate that cellular populations are more variable, with systematic reductions toward the patch boundary, as critical patch size is approached (Fig. 4a). As patch size increases, the

¹ Some of the parameter values used here exceed the levels of precision that can be observed in the field due to the mathematical nature of critical thresholds in deterministic models.

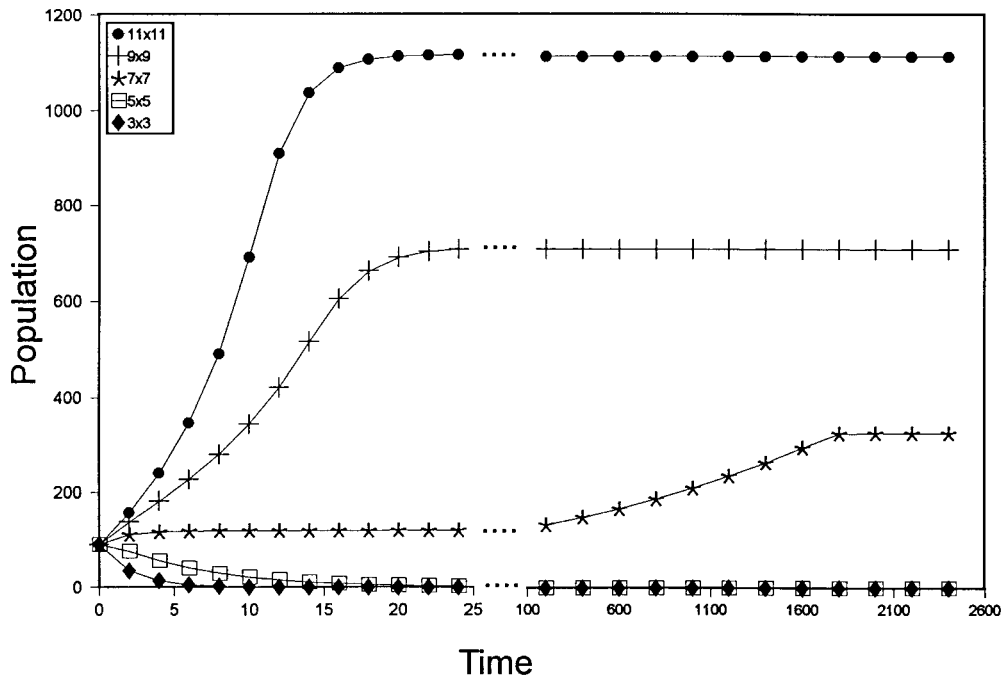


FIG. 3. Population growth curves from centrally located initial populations of 90 organisms for five sizes of square habitat patches with reaction-diffusion parameters $r = 0.495$ and $\sigma = 2.50$.

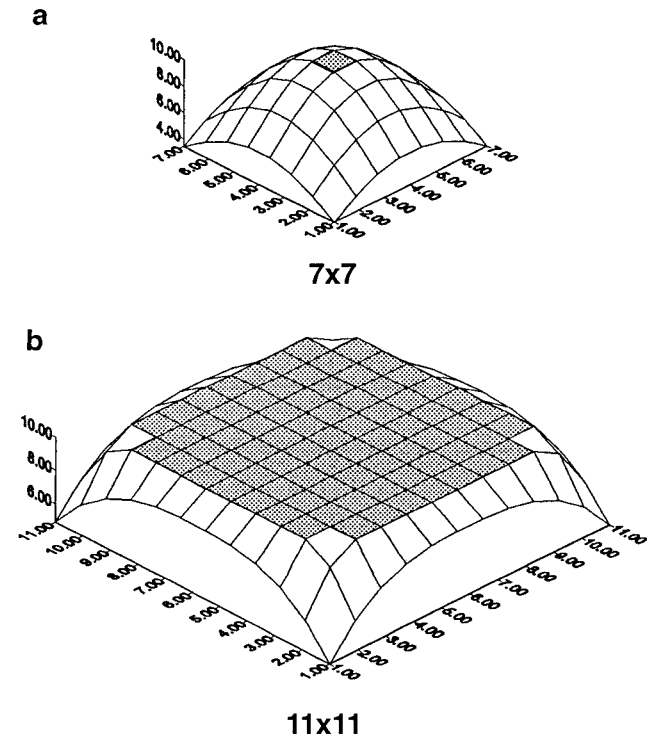


FIG. 4. Cellular populations at equilibrium for (a) a 7×7 and (b) an 11×11 cell patch with reaction-diffusion parameters $r = 0.495$ and $\sigma = 2.50$, and centrally located initial populations of 90 organisms. Shading indicates cells at carrying capacity.

addition of centrally located cells results in a more uniform pattern of cellular populations (Fig. 4b). All of these patch size effects are consistent with existing reaction-diffusion theory (see the review by Holmes *et al.*, 1994).

Effects of patch shape. When dispersal is equally likely in all directions, Game (1980) and others have suggested that circular shapes are preferred for single patches of remnant habitat. Ludwig *et al.* (1979) confirm that critical patch area is smallest for circular areas under such assumptions. Using uniformly distributed dispersal as we do here, we would expect elongations in patch shape to affect overall population growth and persistence in our model. To confirm this, we consider three patches, each comprising 144 cells one unit in size, but arranged with increasing elongation. The curves in Fig. 5 result from a centrally located initial population of one unit (with $r = 0.495$ and $\sigma = 2.50$). As the shape elongates from a square to the 4:1 length-to-width ratio, early growth lags, the slope of the growth curve declines, and the upper population limit drops. When the shape is elongated further (16:1), the patch drops below a critical shape threshold and cannot support any long-term population. This result again conforms with existing reaction-diffusion theory (see Holmes *et al.*, 1994).

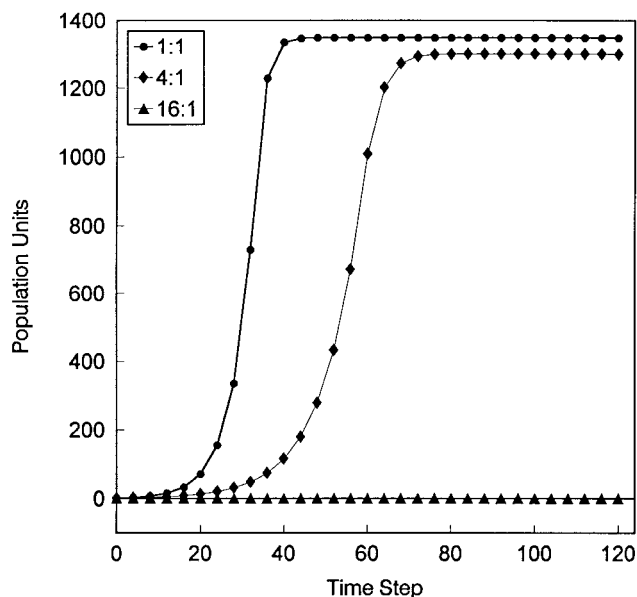


FIG. 5. Population growth curves from centrally located initial populations of one unit with reaction-diffusion parameters $r = 0.495$ and $\sigma = 2.50$ for 144-cell rectangular patches of three different shapes, indicated by length-to-width ratio.

Effects of intrapatch heterogeneity. Intrapatch heterogeneity can be introduced into the model by varying the cellular carrying capacity limits (b_i), allowing us to begin exploring habitat configurations that have proven difficult to address with earlier models. The individual effects of variations in cellular capacity depend on that cell's location within the modeled complex, and on the associated reaction-diffusion system. In a 7×7 cell patch with $r = 0.495$ and $\sigma = 2.50$, setting the carrying capacity of the center cell to one, with all of the others set to ten, results in an equilibrium population of 32.75 distributed as in Fig. 6a. Because the patch is very close to threshold conditions, a 90% lower capacity in just the center cell has the same effect as a 90% reduction in all cells. Although a 90% reduction in carrying capacity within a patch at threshold conditions does not drive the expected population to zero in this deterministic model (unlike the removal of a single cell), the effects are large and the resulting population would be much more likely to perish in a stochastic simulation (Goodman, 1987). Corner cells in a square patch are the least "connected" in a diffusion sense, so that lowering the capacity of a corner cell has less effect, resulting in an equilibrium population of 114 (shown in Fig. 6b). Near threshold conditions, the locations of habitat quality changes have a significant effect on total abundance within the patch.

Populations Occupying Multiple Patches

One of the principal strengths of this model is that similarities and differences in population responses to both contiguous and fragmented habitat complexes can be examined without requiring any structural change in the formulation. In the following experiments, we examine these similarities and differences by investigating the effects of habitat fragmentation.

Fragmentation of contiguous habitat. Allen (1987) shows the existence of a critical number of patches in a reaction-diffusion model of one-dimensional locally coupled patchy habitats. This suggests that we should be able to observe persistence thresholds for fragmented habitat complexes similar to those we have observed for contiguous patches.

Earlier, we described a 7×7 cell patch that slightly exceeded critical habitat threshold conditions for a population with $r = 0.495$ and $\sigma = 2.50$. With carrying capacity set at ten population units per cell, the equilibrium patch population was 327.5. If we fragment that contiguous block of cells into 49 one-cell patches arranged in a hyperdispersed fashion, as in Fig. 1b, dispersal-related mortality increases markedly. Consequently, the habitat complex can no longer support a persistent population under those reaction-diffusion conditions. Indeed, population persistence is not observed until the r -value is increased to 2.172 (resulting in an equilibrium population of 296.2) or until mean dispersal is reduced below 0.53 (implying that almost two-thirds of the organisms remain in their home cell from time period to time period, resulting in an equilibrium population of 312.2). Because none of the patch populations in the complex are individually persistent, we would expect such a complex to function as a metapopulation (Harrison and Taylor, 1997).

Consistent with the notion of a critical number of patches (Allen, 1987), the population fails to persist at $r = 2.171$ and $\sigma = 2.50$ unless more habitat is added to the complex. With the addition of a single similarly displaced one-cell patch off the lower right-hand corner of the complex, a persistent equilibrium population of 293.3 organisms results. Thus, for a population occupying hyperdispersed one-cell patches in a square arrangement, with the reaction-diffusion parameters given above, the critical number of patches is 50.

As we increase the overall size of the hyperdispersed habitat complex from 7×7 to 9×9 and 11×11 one-cell patches (with $r = 2.172$ and $\sigma = 2.50$), the population growth curves shift, as in the single-patch case (Fig. 3), from j -shaped to sigmoid (Fig. 7). However, the rate of population growth is slower than in the contiguous

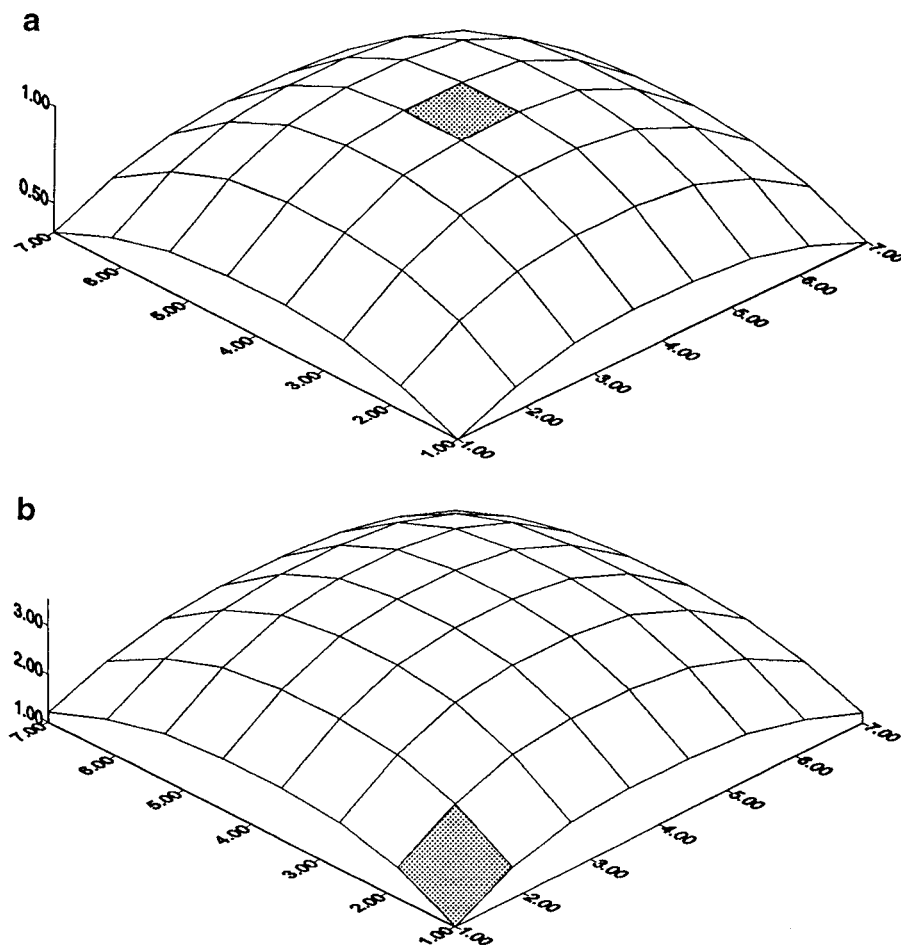


FIG. 6. Cellular populations at equilibrium in a 7×7 cell patch with reaction-diffusion parameters $r = 0.495$, $\sigma = 2.50$, and carrying capacities of ten population units in all cells except (a) the center cell and (b) a corner cell (shaded), for which carrying capacities are set to one.

habitats portrayed in Fig. 3 (from similar initial populations). Equilibrium populations approach total available carrying capacity as the habitat complex is expanded beyond threshold conditions, although again at a slower rate than observed with contiguous habitats.

Pronounced differences in critical threshold effects between contiguous and patchy habitats can also be observed, however. For example, after noting that a four- to five fold increase in r -value is enough to largely compensate for the effects of fragmenting our 7×7 cell habitat complex, we might expect that (with $r = 0.495$ and $\sigma = 2.50$) population recovery could be achieved by providing a four- to five fold increase in the size of the fragmented complex. In our experiments, though, even a 200×200 hyperdispersed complex of one-cell patches was inadequate to support a persistent population in these circumstances.

On one hand, we have a set of results that support Allen's (1987) finding that, for a given r -value and dispersal rate, it is always possible to reach persistent equilibrium population conditions by adding enough similarly configured habitat. On the other hand, we have a set of results suggesting instead that, because the ratio of habitable cells to uninhabitable cells and their geometry remain unchanged, no amount of additional similarly arranged habitat would be enough. This dilemma is resolved by examining the dispersal parameters.

In our 7×7 hyperdispersed complex, the center patch is best positioned to supply successful dispersers given our dispersal assumptions. From that patch, organisms have a probability of about 0.34 of dispersing into any one of the 49 patches in the complex. They have a probability of about 0.60 of dispersing into nonhabitat areas within the complex perimeter and perishing, and a

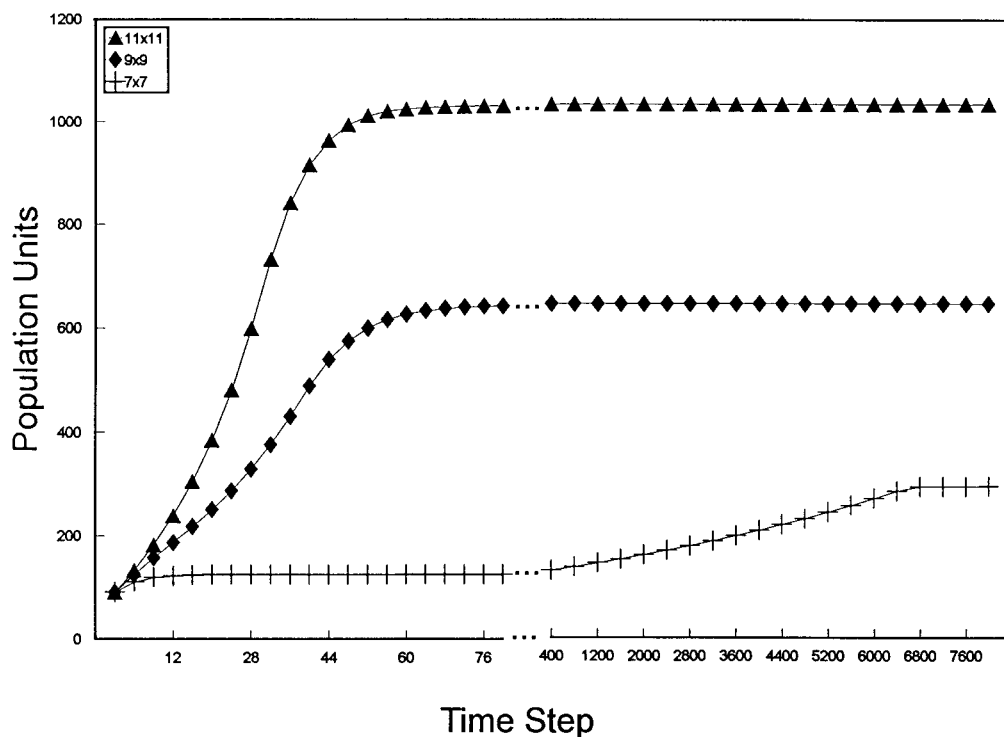


FIG. 7. Population growth curves from centrally located initial populations of 90 organisms with reaction-diffusion parameters $r = 2.172$ and $\sigma = 2.50$ for varying sizes of habitat complexes fragmented into hyperdispersed arrangements of one-cell patches.

probability of about 0.06 of dispersing beyond the perimeter of the complex and perishing. If all 49 cells had similar circumstances, the population would have to support an r -value of about 1.94 to persist in this model.² Because the other 48 patches are actually less well-positioned than the center patch, an r -value slightly greater than 2.17 is required. Furthermore, adding similarly configured habitat patches around the complex can only increase the probability of successful dispersal from the center cell by about one-fourth of 0.06, suggesting a minimum r -value for persistence somewhat greater than 1.82.³ Consequently, we can expect some possibility of reaching persistence by adding hyperdispersed one-cell patches for populations with r -values between roughly 1.82 and 2.17 given our dispersal assumptions. Below an

r -value of about 1.82, no amount of additional similarly configured habitat will suffice.

Island systems. In a mainland-island system connected by population diffusion, with an island being too small to independently support a persistent population, we generally expect the island population to have little influence on the size of the overall population. In an archipelago system with no "mainland," on the other hand, we generally expect each island to have a stronger influence on overall population size (again assuming that each island is too small to independently support a persistent population). Stepping-stone systems arise because, in many cases, a practical limit to dispersal distance exists such that organisms are unable to move directly between all patches in the habitat complex, forming an incompletely coupled lattice.

One of the more interesting questions regarding these island systems concerns the relative importance to population persistence of small, isolated patches as diffusion varies. We investigate the effects of increasing diffusion in mainland-island and archipelago systems using three separate habitat patches, arranged diagonally, and decreasing in size (Fig. 8). If this complex is inhabited by a species with a mean dispersal distance of 2.00, an r -value of 0.571, and a carrying capacity of ten organisms

² We estimate this from $[(0.34(1+r)) = 1]$ by observing that the total number of organisms (per capita) following reproduction $(1+r)$ times the fraction that will disperse successfully (0.34) has to equal one surviving organism or more (per capita) in order for the population to persist in our model.

³ We calculate this from $[(0.34 + (0.25)(0.06))(1+r) = 1]$ by estimating that if hyperdispersed one-cell habitat patches were added throughout the region extending beyond the present complex perimeter, organisms would be able to disperse successfully from the center cell at a rate of about 25%.

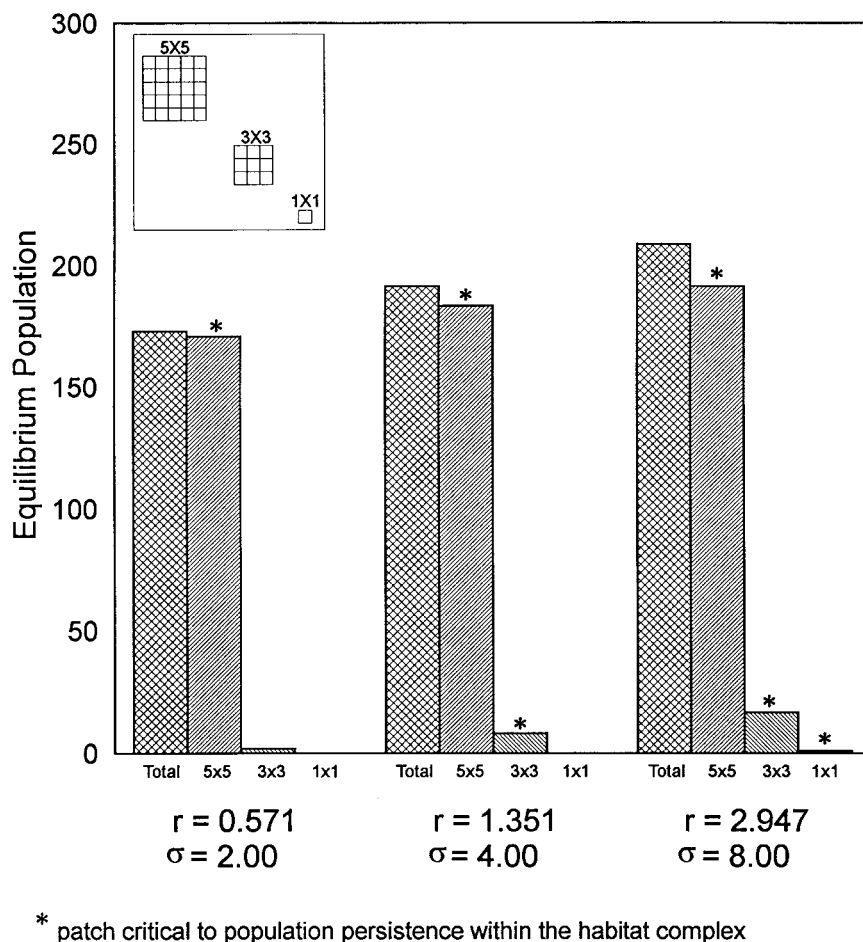


FIG. 8. Total and patch-level equilibrium populations and critical patches in a three-patch complex with a total expected carrying capacity of 350 organisms (see inset) under three sets of threshold-level reaction-diffusion parameters reflecting increasing mean dispersal and net reproduction.

per cell, the complex just exceeds critical threshold conditions for supporting a persistent population of 173.0 organisms. The two smaller islands, however, are not essential for persistence. The 5×5 patch alone exhibits an equilibrium population (170.6 organisms). Because a mean dispersal distance of 2.00 units results in little inter-patch diffusion, this complex functions as a mainland-island system with very small expected populations on the islands, which would “wink” (Gilpin, 1987) in and out of existence in a stochastic simulation with population modeled in integer units.

For species that exhibit greater mean dispersal distances, a true archipelago system emerges. Consider, for example, a species with a mean dispersal distance of 4.00 units. In this habitat complex, such a species would need an r -value of 1.351 just to achieve critical threshold conditions for persistence. A carrying capacity of ten organisms per cell would then produce an equilibrium population of 191.7 organisms. For this species, however,

the 5×5 cell patch is no longer able to independently support persistence; instead, both of the two larger islands are required, while the smallest island remains unessential and barely populated. At a mean dispersal distance of 8.00 units (and an increase to $r = 2.947$ to meet critical threshold conditions), all three islands become essential to support a persistent equilibrium population of 208.7 organisms. Near persistence thresholds for this habitat chain, a shift toward metapopulation conditions occurs as mean dispersal distance increases to encompass most or all of the complex.

Under passive diffusion assumptions, high reproduction rates are required to support such metapopulations, and only slightly higher rates cause a shift back to a mainland-island system. At a mean dispersal distance of 4.00 units, increasing the r -value from 1.351 (the persistence threshold as a metapopulation) to 1.355 allows the population on the largest island to persist independently. At a mean dispersal distance of 8.00 units,

TABLE 1

Equilibrium Populations in Stepping-Stone Island Systems with Varying Population Growth Rates

Habitat layout	Population growth			
	$r = 0.401$	$r = 0.400$	$r = 0.399$	$r = 0.398$
	Equilibrium population			
Total without stepping stone	322.6	0	0	0
Total with stepping stone	330.2	326.0	321.9	0
Stepping stone	1.9	1.9	1.8	0

Note. All simulations were run with mean dispersal distance (σ) set to 1.5 units with a maximum dispersal distance of 3.5 units. The habitat layout is shown in the figure inset.

increasing the r -value from 2.947 to 2.977 causes similar results. These narrow ranges in r -values and their proximity to extinction thresholds suggest that classic metapopulation dynamics (Harrison and Taylor, 1997) would occur only rarely in passively diffusion populations.

Using the exponential distribution for dispersal distances, as we do here, unrealistically portrays the diffusion process as occurring over infinite distances in each time step. Instead, organisms are generally limited to some finite maximum dispersal distance over a fixed time interval. This leads to the possibility that small “stepping-stone” habitat patches might be used to provide diffusion between two larger but otherwise isolated habitat complexes. Based on our analysis of archipelago systems, we might expect that in some, but not all cases, a stepping-stone patch could provide important connectivity for population diffusion.

To investigate the effects of stepping-stone patches, we constructed a model with two 5×5 cell habitat patches placed edge-toward-edge at a distance of three cells apart. Centered between these two patches, we placed a one-cell stepping-stone patch (see figure inset in Table 1). Diffusion parameters were calculated as before using an exponential distribution with a mean dispersal distance of 1.50 cell-side units, but truncated at a maximum distance of 3.50 units. Because diffusion coefficients are estimated from the center of each source cell in our numerical analysis, no diffusion occurs directly between the two larger patches.

With an r -value of 0.401 and carrying capacities set to ten organisms per cell, the two large patches can each independently support an equilibrium population of 161.3 organisms at threshold conditions (Table 1). Adding the stepping-stone patch increases the total complex population from 322.6 to 330.2 organisms, with 1.9 of those in the stepping-stone patch. Without the stepping-stone, reducing the r -value to 0.400 drops both of the larger patches below critical threshold conditions.

With the stepping-stone in place, an r -value of 0.400 merely reduces the equilibrium population to 326.0 organisms.

While this demonstrates that stepping stone patches can support persistence of modeled metapopulations, the usefulness of such patches appears to be quite limited. A slight reduction in net reproduction from 0.400 to 0.398 causes the metapopulation to perish even with the stepping stone patch. Thus, the range in r -value for which this well-placed stepping stone patch supports persistence is only from 0.399 to 0.400. Similarly, if we increase the distance between the two larger patches from three to five units (with $r = 0.400$), and again place the stepping stone halfway between the larger patches,

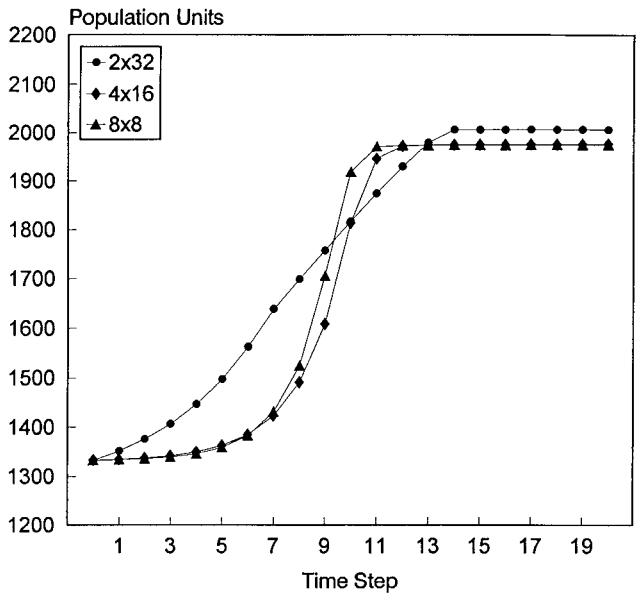


FIG. 9. Population growth curves beginning from equilibrium conditions in an existing habitat complex with reaction-diffusion parameters $r = 1.75$ and $\sigma = 2.50$ following the introduction of an unoccupied, centrally placed 64-cell patch of habitat varying in shape.

TABLE 2

Equilibrium Populations at Various Mean Dispersal Distances (σ) for Isolated and Combined Habitats of Different Configuration at $r=1.75$ with Cellular Capacities Set to Ten

σ	140-Cell ring	8 $\times$ 8 patch	Ring with 8 $\times$ 8 patch	4 $\times$ 16 patch	Ring with 4 $\times$ 16 patch	2 $\times$ 32 patch	Ring with 2 $\times$ 32 patch
2.5	1332.9	640.0	1976.0	640.0	1978.3	640.0	2007.0
3.5	0	640.0	676.3	640.0	688.1	614.3	818.9
4.5	0	638.5	678.8	631.8	679.8	0	0
5.5	0	633.0	680.6	607.2	659.4	0	0
6.5	0	613.4	666.2	516.5	568.4	0	0
7.5	0	555.0	608.5	0	0	0	0
8.5	0	0	0	0	0	0	0

the metapopulation perishes. These results suggest that successful use of stepping stone patches in population recovery strategies would be improbable under our dispersal assumptions.

Effects of patch shape on colonization. Diamond and May (1976), Game (1980), and others have suggested that new patches introduced into existing habitat complexes would be colonized more quickly given an elongated shape rather than circular or square shapes. Hamazaki (1996) points out, however, that elongated perimeters could enhance emigration as much or more than immigration, confounding the expected results. With equal-size cells and equivalent diffusion for all cells, our examples retain the property that cell-to-cell diffusion is identical for both emigrating and immigrating organisms. Consequently, we can use our model to examine the effects of patch shape on colonization in the absence of immigration or emigration bias by placing unpopulated patches identical in size but of different shapes within a populated habitat complex.

We constructed a habitat complex of 140 cells connected together to approximate the circumference of a circle about 36 cell-sides in diameter to represent the surrounding existing habitat for this analysis. With $r = 1.75$, $\sigma = 2.50$, and capacities set to ten organisms per cell, this ring of habitat supports an equilibrium population of 1333 organisms. We used this population as our initial condition.

We placed three different shapes of unpopulated habitat 64 cells in size into the center of the existing habitat ring and modeled population growth (Fig. 9). For the first several time steps, the most elongated patch (a 2×32 cell rectangle) clearly demonstrates a more rapid net gain in population. The moderately elongated patch (a 4×16 rectangle), on the other hand, shows somewhat slower population growth than the square shaped patch, although both show more rapid gains than

the 2×32 cell patch shortly before approaching equilibrium conditions. Finally, the most elongated patch does appear to support a slightly greater long-term population than the less elongated patches. However, nearly all of the additional long-term population actually occurs in the surrounding ring, probably because the 2×32 cell patch places several cells much closer to the surrounding ring than do the other two patches.

To test whether these effects of patch shape are sensitive to the reaction-diffusion parameters, similar analyses were performed with $r = 2.25$ and $\sigma = 2.50$ and 3.50 . All results were similar to those shown in Fig. 9. Elongated new patches in existing habitat complexes appear to consistently show some early colonization advantages under passive diffusion assumptions, while blockier patches tend to support faster population growth later on.

Whether newly colonized elongated or blocky patches will support higher overall equilibrium populations likely depends on the arrangement of the surrounding habitat, and on mean dispersal distance. Moreover, once the new patch becomes necessary for persistence, serving as the “mainland” of the habitat system, our square patch is able to support population increases in the complex as mean dispersal distance increases, while the better “connected” elongated patches are not (Table 2). Although these increases are slight, they suggest that the addition of a blockier critical habitat patch can significantly increase resilience to variations in dispersal distance.

DISCUSSION

Organisms movement fundamentally affects the distribution and abundance of a species within and among habitat patches. In this paper, we numerically examine the effects of habitat size, shape, and arrangement on

spatially structured populations. We use a discrete reaction–diffusion model (derived in the Appendix) applied to a cellular grid of habitat and nonhabitat territories forming a coupled map lattice. Dispersal is modeled passively for adults and juveniles using a procedure that produces a probability of dispersal mortality proportional to the amount of nonhabitat and fully occupied habitat surrounding a given cell, with distance decay. Successful dispersal from one habitat cell to another is determined primarily by distance. The key feature of this approach is that any landscape arrangement can be accommodated, with population diffusion represented by straightforward proportion parameters.

Through a series of experiments on the effects of patch size and shape on single-patch populations, we confirm that results from our discrete model conform to those generally expected from reaction–diffusion models, as summarized by Holmes *et al.* (1994). These effects include the existence of critical patch size thresholds (Fig. 2), increases in overall population growth rate and more fully occupied habitat as patch size increases above the critical size threshold (Fig. 3), Gaussian-like equilibrium population distributions in uniform habitat (Fig. 4), and the observation that critical patch size is smaller for square patches than for elongated shapes (Fig. 5). Furthermore, we confirm the prediction that increasing dispersal out of the patch leads to larger critical patch sizes. In our model, increases in mean dispersal distance have this effect, and must be compensated for by increases in r -value or patch size for populations to be maintained. As we noted, these outcomes are consistent with those previously reported from reaction–diffusion studies, providing a reasonable basis for experiments with heterogeneous habitat patches and fragmented patch complexes.

In non-uniform single-patch systems, our experiments on the effects of heterogeneous habitat capacities demonstrate that the location of habitat quality changes become quite important near extinction threshold conditions. At critical patch size, equilibrium populations appear to approach Gaussian-like distributions of abundance in space with total population constrained by the capacity of the most limiting (in a reaction–diffusion sense) cell (Fig. 6).

Similar effects can be shown for populations in fragmented habitat landscapes using otherwise identical models (e.g., Fig. 7). Our experiments clearly demonstrate, however, that these systems have inherently more complex behavior that is parametrically sensitive. For example, the existence of a critical size threshold in single-patch systems guarantees that population persistence can be achieved by increasing the size of the patch

regardless of the magnitude of the other system parameters. For hyperdispersed patchy habitats, on the other hand, we show that the ability to achieve persistence by extending the size of the complex (i.e., providing more similarly fragmented habitat) depends upon the reaction–diffusion parameters in the system. At sufficiently low r -values in our model, such an approach cannot succeed regardless of the increase in size of the complex. Because these systems could be made persistent by adding a single individually persistent patch, population recovery efforts might best be focused on strategies that increase existing patch sizes when those options are available.

We see parametric sensitivity in our results for other fragmented landscapes, as well. For an island-chain habitat complex, metapopulation conditions in which no single patch is individually persistent arise only when both dispersal distances and reproduction rates are relatively high (Fig. 8), and only within very narrow parameter ranges that are close to extinction thresholds. These results offer one explanation for Harrison and Taylor's (1997) finding that classical metapopulations appear to be scarce. In terms of population distribution, persistent archipelago complexes do not appear to be substantially different from mainland-island systems. Empirically, they may be indistinguishable, and stochastically, extinctions complex-wide may be nearly as frequent as extinctions from large islands.

Similarly, our results suggest that successful use of stepping-stone patches to support metapopulation systems would be unlikely for passively diffusing species (Table 1). Once again, these results tend to suggest that when options are available, more emphasis should be placed on increasing habitat patch sizes and providing blockier patch shapes within existing fragmented landscapes to achieve persistence. This is consistent with Adler and Nuernberger's (1994) finding that clustering identical patches in a fragmented habitat complex enhances population persistence. Although elongated patches added to existing complexes do offer early colonization advantages (Fig. 9), blocky patches appear to offer greater long-term population resilience to fluctuations in reaction–diffusion behavior near threshold conditions (Table 2).

Naturally, our interpretations are predicated on the simple assumptions we have made about population behavior. Passive diffusion identical for adults and juveniles, a completely uninhabitable matrix between habitat patches, and constant per capita reproduction are fairly strong assumptions, even though similarly simple assumptions have been used successfully to model populations of many species, including humans, with reaction–diffusion equations (e.g., Ammerman and

Cavalli-Sforza, 1984). The effects of relaxing these assumptions in reaction–diffusion models are generally well understood for single-patch systems (see Holmes *et al.*, 1994: 22–23). For example, factors that decrease diffusion from patches generally lead to smaller critical patch sizes. Such factors would include advective diffusion toward available habitat, density-dependent dispersal, and nest site fidelity. Likewise, less hostile boundary conditions generally lead to smaller critical patch sizes.

Capturing more realistic dispersal behavior by adding age-structure as we discuss in the Appendix is tempting, but requires a substantial increase in data that is difficult to obtain and is highly variant. Wennergren *et al.* (1995) question the usefulness of such added complexity. We also note that increasing age complexity to separately represent adult and juvenile dispersal results in a model that differs only parametrically from the simpler model used for our experiments (see the Appendix).

Density-dependent population growth rates do tend to regulate population sizes, but do not affect critical patch size in the absence of Allee effects (Murray, 1989). Furthermore, population growth occurs within breeding territories in our model. While Eq. (1) can incorporate density-dependent growth effects, those effects are modeled in our experiments at the population scale as a consequence of dispersal related mortality instead. This is consistent with the notion that mortality during dispersal can have a stabilizing influence on populations (Ruxton *et al.*, 1997). Unlike models in which dispersal-related mortality is not apparent (e.g., Levin, 1974; Hastings, 1982, 1992; Bascompte and Solé, 1994; and Hassell *et al.*, 1995), it is portrayed explicitly in our model through the cumulative dispersal parameters (see the Appendix).

While relaxation of our simple assumptions generally leads to predictable effects for single-patch systems, this was not the case for multi-patch systems. As we have shown, multi-patch systems display more complex outcomes. However unlikely, results such as observing increases in total population when patch emigration rates are increased (Table 2) suggest that further research on these systems under broader reaction–diffusion assumptions is warranted. In our deterministic experiments, equilibrium populations are insensitive to initial populations, and persistence is insensitive to carrying capacity. Consequently, stochastic reaction–diffusion modeling could also be an important area for further work. Stochastic extinction models (e.g., Goodman, 1987) have generally shown that initial population level and carrying capacity are both important factors in estimating persistence times. Sensitivity to initial populations might

also occur deterministically with high enough reproduction rates, if chaotic or catastrophic population transients develop (as in Hastings and Higgins, 1994). Although we did not observe such transients, further work in this area seems particularly warranted for classical metapopulations given their apparent rarity and their manifestation at high reproduction and dispersal rates.

Despite the simplicity of our assumptions, the results from our experiments suggest that reaction–diffusion effects pervade many basic ecological concepts. Based on observing that population density depends on patch size, effective carrying capacity may be so entangled with reproduction, dispersal, and spatial heterogeneity that empirical estimates of the capacity in a given habitat complex could be flawed (perhaps significantly) if patch sizes, shapes, and arrangements are not considered. Another implication, based on observing density declines toward habitat edges in uniform patches, is that reductions in population densities at habitat perimeters among species considered to be habitat interior specialists may be partially explained by dispersal patterns rather than selection for interior habitats. Similarly, slow population growth and spread rates during early observations of invading species could be due to poorly suited habitat, Allee effects, or could also result from habitat configuration and initial population placement combined with random diffusion.

Perhaps the most troubling inference from our study is that estimating population viability may require knowledge of when habitats are approaching critical threshold conditions. Unfortunately, traditional monitoring protocols are not designed to detect when such conditions have been reached (see Kareiva and Wennergren, 1995). Population dynamics at or near critical thresholds, at least as modeled here, show long periods of quasi-stability that when monitored would belie a trajectory toward extinction. Consequently, conservation biologists may be handicapped until innovative monitoring methods can be developed. Our results suggest that slow rates of population change in response to habitat enlargements or reductions, and strongly kurtotic abundance distributions in large habitat patches, may indicate threshold population conditions, but further work is needed on this important problem.

APPENDIX: MODEL DERIVATION

Allen (1987, following Levin, 1974) describes a random walk (passive) reaction–diffusion model for a fragmented complex of N habitat patches (cells in our usage) as

$$\begin{aligned} \frac{dv_i}{dt} &= v_i f_i(v_i) + \sum_{j=0}^N D_{ij}(v_j - v_i); \quad i = 1, \dots, N \\ D_{ij} &= D_{ji} \geq 0 \quad \forall i, j \\ v_0 &= 0 \end{aligned} \quad (3)$$

where v_i is the population in cell i as a function of time t , $f_i(v_i)$ is the per capita rate of reproduction (net of mortality unassociated with dispersal, as discussed below), and D_{ij} is defined as cell-to-cell passive diffusivity constant that determines the net cell i population gain from (or loss to) cell j based on the difference between the cell populations. Nonhabitat is indexed by $j = 0$.⁴ In this model, dispersing organisms can successfully cross inter-cellular regions of nonhabitat, but some of them perish and are treated as dispersers into nonhabitat. Because the nonhabitat population v_0 is fixed at zero, D_{i0} times $-v_i$ defines the population loss due to unsuccessful dispersal from any cell i . Only those cells (indexed by i) which are “connected” by positive diffusivity ($D_{ij} > 0$) with at least one other cell (some $j \neq i > 0$) comprise the habitat complex. This definition of cell connectivity imposes no constraint on the particular arrangement of habitat in the complex.

Expanding the diffusion summation in Eq. (3) and recollecting the terms into separate summations for emigration and immigration, we get

$$\frac{dv_i}{dt} = v_i f_i(v_i) - \sum_{\substack{j=0 \\ j \neq i}}^N D_{ij} v_i + \sum_{\substack{j=1 \\ j \neq i}}^N D_{ij} v_j \quad \forall i, \quad (4)$$

where we now distinguish the diffusivity constants D_{ij} and D_{ji} to indicate the direction of dispersal.

Reproduction and diffusion are modeled as simultaneous processes in this equation, so that reproduction in each cell can implicitly contribute to the diffusion summation terms. In a simple age-structured population comprising only adults and juveniles, four cases are possible: only adults disperse, only juveniles disperse, adults and juveniles disperse identically, or adults and juveniles disperse, but not identically. To clarify this, we define g_{ij} as the proportion of potential dispersers in cell i expected to move to cell j per unit of time, so that g_{i0} is the proportion that perish in nonhabitat while dispersing from cell i per unit of time, and $g_{ii} = [1 - \sum_{j=0, j \neq i}^N g_{ij}]$ is the proportion expected to remain in cell i per

unit of time. Then, if only adults disperse, Eq. (4) becomes

$$\frac{dv_i}{dt} = v_i f_i(v_i) - \sum_{\substack{j=0 \\ j \neq i}}^N v_i g_{ij} + \sum_{\substack{j=1 \\ j \neq i}}^N v_j g_{ji} \quad \forall i$$

and we see that all $D_{ij} = g_{ij}$. If only juveniles disperse, Eq. (4) becomes

$$\frac{dv_i}{dt} = v_i f_i(v_i) - \sum_{\substack{j=0 \\ j \neq i}}^N v_i f_i(v_i) g_{ij} + \sum_{\substack{j=1 \\ j \neq i}}^N v_j f_j(v_j) g_{ji} \quad \forall i$$

and we see that all $D_{ij} = f_i(v_i) g_{ij}$. This is equivalent to

$$\frac{dv_i}{dt} = v_i f_i(v_i) \left[1 - \sum_{\substack{j=0 \\ j \neq i}}^N g_{ij} \right] + \sum_{\substack{j=1 \\ j \neq i}}^N v_j f_j(v_j) g_{ji} \quad \forall i,$$

which simplifies to

$$\frac{dv_i}{dt} = \sum_{j=1}^N v_j f_j(v_j) g_{ji} \quad \forall i.$$

If both adults and juveniles disperse identically (as we assume in our experiments), Eq. (4) becomes

$$\frac{dv_i}{dt} = \sum_{j=1}^N v_j f_j(v_j) g_{ji} - \sum_{\substack{j=0 \\ j \neq i}}^N v_i g_{ij} + \sum_{\substack{j=1 \\ j \neq i}}^N v_j g_{ji} \quad \forall i$$

or

$$\begin{aligned} \frac{dv_i}{dt} &= v_i f_i(v_i) - \sum_{\substack{j=0 \\ j \neq i}}^N v_i [1 + f_i(v_i)] g_{ij} \\ &\quad + \sum_{\substack{j=1 \\ j \neq i}}^N v_j [1 + f_j(v_j)] g_{ji} \quad \forall i \end{aligned} \quad (5)$$

and we see that all $D_{ij} = [1 + f_i(v_i)] g_{ij}$. We note that a restriction $g_{ij} = g_{ji}$ is not required because we have decoupled immigration from emigration. By eliminating the reliance on population gradients, less restrictive types of diffusion can be modeled (as in Holmes *et al.*, 1994). For the latter two cases in which juveniles are dispersing with stationary probabilities, we also note that the D_{ij} diffusivity parameters in gradient-based models should only be treated as constants when the net per capita cellular reproduction rate functions (f) are also constant. We discuss the case in which both adults and juveniles disperse, but not identically, below.

Converting the single-stage population system of Eq. (5) to discrete time results in

⁴ Allen (1987) includes $j = N + 1$ in the diffusion summation as an additional designator for nonhabitat to conveniently account for population movement along a locally coupled linear lattice. This additional nonhabitat index is not needed here.

$$v_{it} = v_{i,t-1} + v_{i,t-1} f_i(v_{i,t-1}) - \sum_{\substack{j=0 \\ j \neq i}}^N v_{i,t-1} [1 + f_i(v_{i,t-1})] g_{ij} + \sum_{\substack{j=1 \\ j \neq i}}^N v_{j,t-1} [1 + f_j(v_{j,t-1})] g_{ji} \quad \forall i, t$$

plus a finite difference approximation error term. For species with distinct breeding seasons, an approximation error would be more correctly assigned to Eq. (5). The discrete-time equation simplifies to

$$v_{it} = \sum_{j=1}^N [1 + f_j(v_{j,t-1})] v_{j,t-1} g_{ji} \quad \forall i, t,$$

which is Eq. (1) in the main body of the paper. Equation (1) has also been used by Kot and Schaffer (1986) as the foundation for integrating to a discrete-time, continuous-space formulation. In discrete-time simulation analysis, the values f_j (net reproduction) and g_{ij} (dispersal) could be defined as random functions $f_j(V, H, t-1)$ and $g_{ji}(V, H, t-1)$, respectively, where V is the vector of cell populations and H is a matrix of cellular habitat conditions in the complex. Such functions could be systematically as well as stochastically time-variant.

Multiple life stages could also be used to depict age- or sex-specific reproduction and dispersal processes (van den Bosch *et al.*, 1992). For species that mature in a single breeding season, and whose adults and juveniles both disperse but not identically, we can derive the following equation in place of Eq. (1):

$$v_{it} = \sum_{j=1}^N v_{j,t-1} f_j^A(v_{j,t-1}) [g_{ji}^A + f_j^J(v_{j,t-1}) g_{ji}^J] \quad \forall i, t.$$

Here we have partitioned net reproduction f_j and dispersal g_{ji} into separate terms for adult survivorship f_j^A and dispersal g_{ji}^A , and for net natality f_j^J and juvenile dispersal g_{ji}^J . More generally, Eq. (1) can be viewed as a special case of

$$v_{ikt} = \sum_{j=1}^N \sum_{m=1}^M h_{jkm} v_{jm,t-1} g_{jik} \quad \forall i, k, t,$$

where k and m index each of the M life stage classes, and the random function h_{jkm} defines the per capita rate per time step of organisms in class k produced by the organisms in class m in cell j . The population state variables and dispersal parameters would be redefined by life stage class accordingly. This formulation, which we

do not pursue in this paper, could directly capture habitat- or density-biased diffusion (e.g., Allen, 1983), stratified diffusion (Shigesada *et al.*, 1995), correlated random walks (Goldstein, 1951), stochastic variation, and similar phenomena.

ACKNOWLEDGMENTS

We are grateful to Mike Knowles for assistance with figures, to Jill Heiner for computer programming support, and to John Hof for early comments on the study and manuscript. We are also grateful to our anonymous reviewers for many improvements in both the study and this report.

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