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CHAPTER ONE

Population Dynamics in Spatial Habitats

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All organisms are discrete entities that mainly interact with neighboring individuals of their own or other species. This discrete nature and spatial confinement is most evident for sessile organisms such as terrestrial plants, marine macrophytes, corals, and other organisms that live attached to surfaces. However, even motile organisms have their greatest impacts in a rather confined region—the region through which they move. These simple observations have profound implications for the dynamics and outcome of both intraspecific and interspecific interactions. In particular, local interactions and local movement/dispersal mean that population densities do not change in response to average conditions across a large habitat, as is assumed in classical nonspatial models, but rather in response to the local conditions experienced by each individual. This chapter presents three simple approaches for dealing with this spatial aspect of species interactions and population biology. The three approaches that we discuss are Levins's metapopulation-like model, cellular-automaton-like models, and reaction-diffusion models. The first and last approaches can be analytically tractable, whereas the middle one is best approached via computer simulation. The first two consider interactions in a spatially subdivided habitat, whereas the last considers interactions in a spatially continuous habitat. These approaches are the building blocks upon which much of this book is based. Our goal is to abstract the essential features of space that come from the discrete

nature of individual organisms. There are many additional complexities to space that are not considered in this chapter but are discussed in subsequent chapters.

LEVINS'S MODEL FOR A SINGLE SPECIES

Levins's (1969) model provides a simple description of the dynamics of a single species living in a habitat composed of distinct sites. Although it has given many insights into communities that are subdivided into local populations, its use as a literal metapopulation model has been criticized (e.g., Hanski 1991, chap. 2; Harrison, Thomas, and Lewinsohn 1995; Harrison and Taylor 1997). For instance, it is viewed as a poor descriptor of metapopulations because it assumes that a single propagule can instantly transform an empty site into a local population that is at its carrying capacity, because local sites cannot differ in their carrying capacities or other measures of their quality, and because there is no possibility of local conditions "rescuing" a local site from extinction.

An alternative use of Levins's framework is to view it as a model for the occupancy of sites by single individuals as opposed to populations. To understand this interpretation of Levins's model, consider a habitat that is divided into sites that are just large enough to contain a single adult individual of a sessile species. A propagule of this species, upon entering an empty site, would occupy it. Individuals occupying sites could produce propagules that would be dispersed throughout the habitat. The rate of propagule production by a site is c (for colonization rate). In addition, there is some mortality rate, m, that is the chance of a currently occupied site becoming empty. To keep track of the dynamics of this species, it is necessary to know p, the proportion of all possible sites that are occupied at a given instant in time. If it is assumed that propagules disperse randomly among all possible sites, these assumptions can be converted into a simple model:

$$\frac{dp}{dt} = cp(1 - p) - mp. \tag{1.1}$$

This states that the rate of change in site occupancy (dp/dt) depends on the rate of propagule production (cp) multiplied by the proportion of currently open sites ($1 - p$). The rate of mortality (mp) is subtracted from this to get the net change in site occupancy.

This simple model of site occupancy has several interesting features. First, a species can persist in a habitat if $c > m$. Populations grow in a logistic fashion. When $c > m$, the proportion of occupied sites will approach an equilibrium, $\hat{p}$ (at which $dp/dt = 0$), with

$$\hat{p} = 1 - \frac{m}{c}. \tag{1.2}$$

This equilibrial density is globally stable (Hastings 1980), meaning that p will approach $\hat{p}$ from any starting density, and any perturbation, as long as $p > 0$. The most important and interesting aspect of this model is that no species is capable of completely filling its habitat at equilibrium. The proportion of all viable sites that are left unoccupied at equilibrium in a habitat occupied by a single species is $\hat{s}$, where

$$\hat{s} = 1 - \hat{p} = \frac{m}{c}. \tag{1.3}$$

Equation 1.3 shows that an unavoidable result of living in a spatial habitat is that a proportion of sites will be empty. The greater the mortality rate of a species relative to its colonization rate, the greater would be the amount of open space.

This model is mathematically simple and analytically tractable because of the simplifying assumption that it makes about dispersal. By assuming that all propagules are randomly dispersed across the entire habitat, this model eliminates the effects of local dispersal. However, by assuming that the habitat is subdivided into sites the size of an adult, the model implicitly addresses the discreteness of individuals and the idea of occupying space. Even though space is not treated explicitly, this model and its derivatives have been remarkably versatile in uncovering certain effects of space both on single species and

on multispecies interactions (e.g., Skellam 1951; Levins and Culver 1971; Horn and MacArthur 1972; Armstrong 1976; Hastings 1980; Shmida and Ellner 1984; Bengtsson 1989, 1991; Tilman 1994; Chapters 8 and 10).

CELLULAR AUTOMATA AND INDIVIDUAL-BASED MODELS

The crucial simplifying assumption above was that of global, random dispersal of propagules. To model local dispersal we still envision a physically homogeneous habitat that is subdivided into sites, each of the size just capable of supporting a single adult. Each individual has a probability, m, of mortality per unit time. Each produces propagules at a rate of c. However, the propagules produced by each individual disperse locally. Local dispersal can be as simple as equiprobable movement to all adjacent cells (four adjacent cells for a habitat divided into a square grid, or six for a hexagonal grid) or a more complex pattern of dispersal to a greater number of nearby sites. A model in which propagules are randomly dispersed to adjacent sites is called a stochastic cellular automaton model. In the limit, as the number of sites that form the neighborhood of an individual is increased, the behavior of this model approaches that of Levins's model (Eq. 1.1).

In our simulations, individual organisms are distributed across a two-dimensional array of hexagonal sites (Figure 1.1). At any instant of time (of length dt), each occupied site sends propagules to a set of neighboring sites with a probability of $c\,dt$. If a propagule lands on an occupied site, the propagule is lost. If it lands on an empty site, the site is colonized and is now fully occupied. This process is repeated every time step, dt, to determine the dynamics of growth of the species.

Because this is a simulation model, there is no closed-form mathematical solution for the general case. However, the model shares many features with Levins's model and has the advantage of being easily modified. This allows its underlying assumptions to be manipulated in an exploration of the importance of these assumptions to the dynamics, equilibrium densities, and spatial patterns predicted by the model. Below

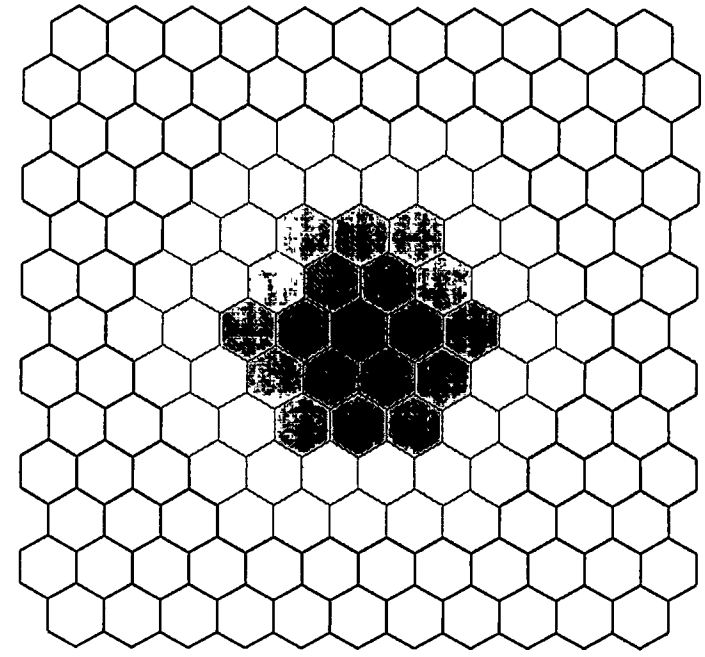
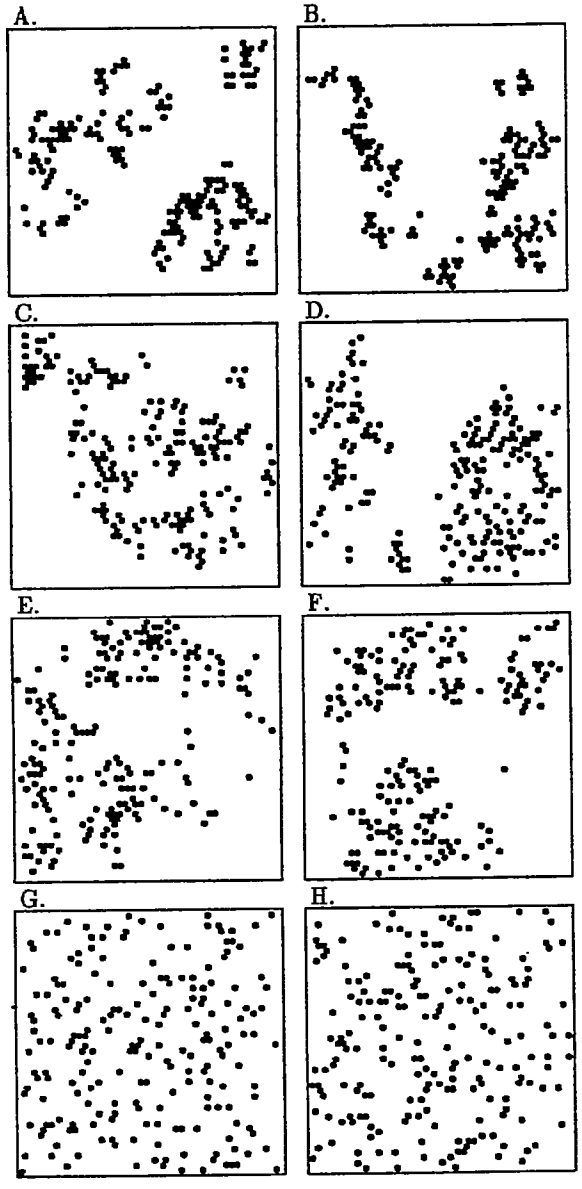


FIGURE 1.1. The hexagonal grid of sites used in our simulations of a spatially explicit habitat (see also Chapters 8 and 10). The shading illustrates the various rings of neighbors to which the propagules produced by the blackened site may be dispersed. The lightest shading is the fourth ring. Each hexagonal cell is the size of the area occupied by a single adult individual of this species.

we highlight some of the unique features that arise when Levins's model is modified into a spatial simulation that includes neighborhood dispersal.

An unavoidable outcome of local interactions (for a single species the interaction is that an occupied site cannot be invaded by a propagule) and local dispersal is clumping (see Durret and Levin 1994a,b for a thorough analysis). This is easily shown in cellular automaton models (Figure 1.2). Each of the six cases we illustrate is for a physically uniform habitat containing 10^4 sites (i.e., a 100×100 habitat). In each case the species becomes clumped, but the extent of this clumping, the



size of the clumps, and the average distance between clumps depend on the mortality rate, the colonization rate, and the distance (number of adjacent rings) over which dispersal occurs. A field biologist encountering a clump of individuals of a species in nature, and noting a nearby habitat in which the species was rare or absent, might immediately wonder what environmental characteristics caused such clumping. These results show that there need not be any environmental cause. Because clumping is predicted to be an unavoidable result of local interaction and local movement in any spatial habitat—even if it is homogeneous—the appropriate null model for organism distributions might be a clumped dispersion (as opposed to the often-used random or Poisson distribution).

As discussed by Holmes (Chapter 5), another unavoidable result of local dispersal is decreased equilibrial abundances compared to the analytical Levins model. This occurs because local dispersal reduces the effective dispersal rate. With local dispersal, there is a greater chance of propagules falling on the same site and thus of all but the first propagule being ineffective. This decrease in the effective dispersal rate with local dispersal is further magnified by clumping. If most individuals of a species live in clumps, most of their propagules will fall in or near the clump and thus experience higher rates of site occupancy than occur on average, across the full habitat. This effect is illustrated in Figure 1.3, which shows how equilibrial population density (proportion of sites occupied) depends on the distance over which propagules are dispersed. Note that propagules have to be dispersed over a range greater than the

FIGURE 1.2. Clustering as a function of dispersal range. Parts A–F are snapshots of typical arrangements of individual organisms after three thousand simulated years, starting from an initially random arrangement, for dispersal ranges of one, two, or three rings around the parent. Parts G and H show a random arrangement, as occurs if there is global dispersal. Occupied cells are shown as black hexagons, and empty cells are not shown. In all cases, parameters were chosen to give equilibrial proportional site occupancy of about 0.08. For A, C, and E, $m = 0.05$, with c of 0.084, 0.068, and 0.065, respectively. For B, D, and E, $m = 0.03$ with c of 0.049, 0.041, and 0.03, respectively.

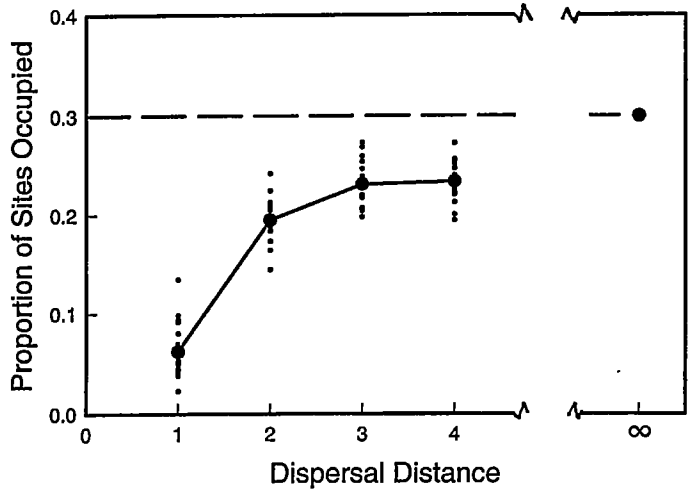


FIGURE 1.3. A comparison of different ranges of neighborhood dispersal with the global dispersal of Levins's model. Holding $m = 0.05$ and $c = 0.0714$ gives an equilibrial abundance of this species of 0.3 in the Levins model, but local dispersal of range 1 (dispersal to adjacent six cells) leads to much lower equilibrial abundances in the spatial simulator. As the dispersal range increases, the equilibrial abundance increases, approaching that of the Levins model when dispersal is across all possible sites.

adjacent four rings of neighbors for equilibrial density to approach that of the Levins analytical model.

Another feature of many spatially explicit models is called percolation. Percolation refers to how something responds to "clogs" in its habitat. For instance, consider water draining through a bed of gravel. The spaces between pieces of gravel form channels through which water may flow. If items are added that clog these channels, flow will eventually cease. However, the probability that any water will flow is not linearly dependent on the number of clogs added but is more like a step function. The average level of clogging at which flow ceases is called the percolation threshold. Populations living in spatial habitats can also exhibit a percolation threshold, but the dynamics are more complex because the "flow" is caused

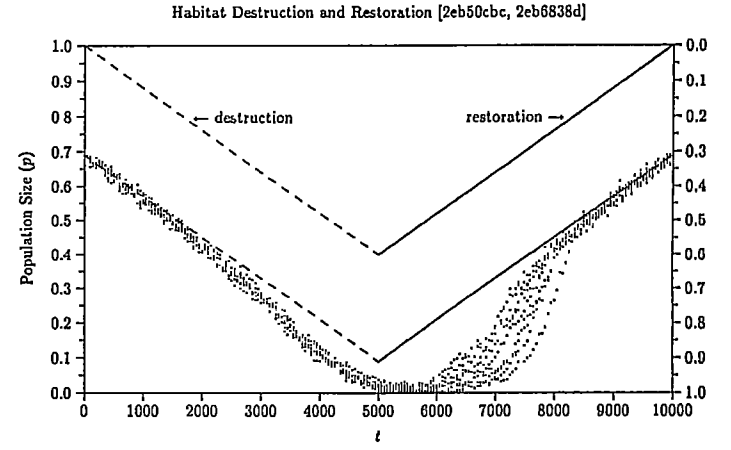


FIGURE 1.4. A composite of twelve replicate runs of random habitat destruction and restoration. The upper dashed line shows the amount of habitat destroyed, and the lower dashed line shows the decrease in the population size of this species analytically predicted by the Tilman et al. (1994) model. The solid lines similarly show habitat restoration and the recovery predicted by the Tilman et al. (1994) model. Dots show the results of runs in our spatial simulator, with simulations being sufficiently slow that each dot approximates the equilibrial outcome of those conditions.

by a combination of dispersal and reproduction, and individuals can survive, even if not reproducing, in completely isolated sites.

Consider a large, physically uniform spatial habitat occupied by a single species. For this habitat, let there be the potential destruction of individual sites. Destruction of a site means that no organisms can live there and that all propagules that enter the site die. The analytical Levins model predicts that a species would have its equilibrial abundance decrease, in a simple linear manner, as the amount of habitat destruction is increased (dashed line in Figure 1.4; see Tilman et al. 1994 and Chapter 10) and that its equilibrial abundance similarly would increase linearly as the habitat is restored to various degrees (solid line in Figure 1.4). Does this also occur in an explicitly spatial habitat? To find out, we imposed random destruction of

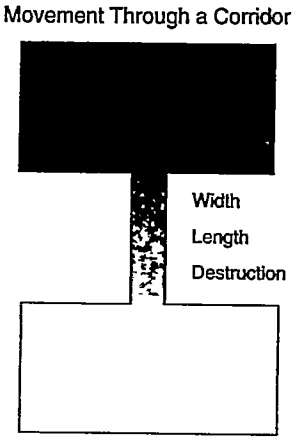


FIGURE 1.5. The ability of a species to move from a source area into a new viable habitat may depend on the length, width, and extent of destruction of a corridor, as illustrated here. See Figure 1.6 for results.

individual sites in our spatial simulator and kept all other model assumptions identical to those of the Levins analytical model. For each case, we ran the simulator while slowly increasing the number of sites destroyed, keeping the system always near equilibrium and continuing the process until the full range of habitat destruction had been covered. Then we started to slowly restore the area by randomly choosing destroyed sites and making them viable again, in similarly sized steps. We repeated these analyses many times, with each replicate simulation being separately randomized.

The resulting pattern differs markedly from the predictions of the analytical model (Figure 1.4). Each dot in Figure 1.4 shows the predicted quasi-equilibrium population abundance for a particular amount of habitat destruction or restoration. Abundance declined with destruction much as predicted analytically, but it did not increase as predicted by the Levins model following restoration. There was a marked "lag" between habitat restoration and the restoration of the population. This was not a time lag, because each point on the graph is close to the long-term equilibrial abundance of the species

for that particular habitat condition. Rather, this was caused by the actual spatial pattern of destroyed and viable sites created by each run of random habitat destruction. As a habitat is restored, random restoration can create many viable sites that are totally inaccessible to current residents because there are no corridors connecting them with occupied sites. As restoration progresses, corridors are eventually created, allowing the species to occupy some formerly inaccessible sites. As this occurs, occupancy of the habitat comes closer to that predicted by the analytical model. This hysteresis results from what is called a percolation effect (Djordjevic 1992; O'Neill, Gardner, and Turner 1992). Percolation of living organisms differs from that of fluids because the percolation threshold depends on the biology of the organisms as well as the pattern of destruction. The variance observed in the responses depends on the actual spatial patterning of the random habitat destruction.

As anticipated by percolation theory, we have found a critical proportion of a habitat that can be destroyed, called D_c, at which point the habitat undergoes an abrupt change. When destruction is less than D_c there are paths that allow a species to migrate through the habitat; when destruction is greater than D_c there is almost no chance of migration from one side to the other of the habitat. The ability to migrate is of little importance as a habitat is being increasingly fragmented because there are few new open sites to which a species can move. However, when a habitat is being restored, the lack of corridors can prevent recolonization of viable sites. The nonlinear effect shown in Figure 1.4 thus is an insight provided by spatially explicit models that was not anticipated by the simpler analytical portraits of spatial processes.

Percolation effects point out some potential problems with corridors that link together the remnant fragments of once continuous ecosystem types. For a corridor to be effective, a species has to be able to migrate through it. This may not be a major problem, even for narrow corridors, if an organism can adjust its behavior to stay within the corridor (as may occur for some animals). However, plants move through a corridor by reproducing at one site and sending propagules out to adjacent sites. If a corridor is too narrow, a plant may lose too

many propagules across its edges and be unable to spread down the corridor. If the corridor is too fragmented, passage may also be stopped. We used our spatial simulator to determine the effects of corridor length, width, and degree of destruction on the probability of movement through a corridor (Figure 1.5; Tilman, Lehman, and Yin 1997). To do this, we assumed we had a large source pool of propagules and investigated the probability that propagules would make it through a corridor to populate an empty intact habitat. We defined the critical amount of destruction for a given corridor as the amount of destruction, D_c, at which propagules were successful in half of the cases (where each case is a try in an independently randomly destroyed corridor). Corridor width (Figure 1.6A; Tilman et al. 1997) had a much greater effect on D_c than did corridor length (Figure 1.6B). We also found an interesting and alarming result: A habitat must be more pristine to serve as a corridor than to serve as a viable long-term refuge. This occurs because it is easier to maintain a species within a highly fragmented habitat than it is to facilitate migration through the same corridor. For example, in simulations reported in Figure 1.6 we found that species could survive in a habitat that had suffered up to 50% habitat destruction. However, the species could not migrate through a corridor that had suffered this much destruction. This means that virgin habitats, such as nature reserves and national parks, in addition to being reserves, might be considered to be viable corridors for repopulating surrounding areas that have suffered some habitat destruction, whereas habitats that have suffered destruction, fragmentation, or both might be considered to be most useful as supplemental reserves but *not* as corridors.

REACTION-DIFFUSION MODELS: SPATIALLY EXPLICIT MODELS FOR CONTINUOUS SPACE AND TIME

The traditional mathematical approach to spatial population models has involved the analysis of so-called reaction-diffusion models. In an ecological context "reaction" pertains to the process of population change or species interaction in the absence of any dispersal. Thus a reaction term in a reaction-

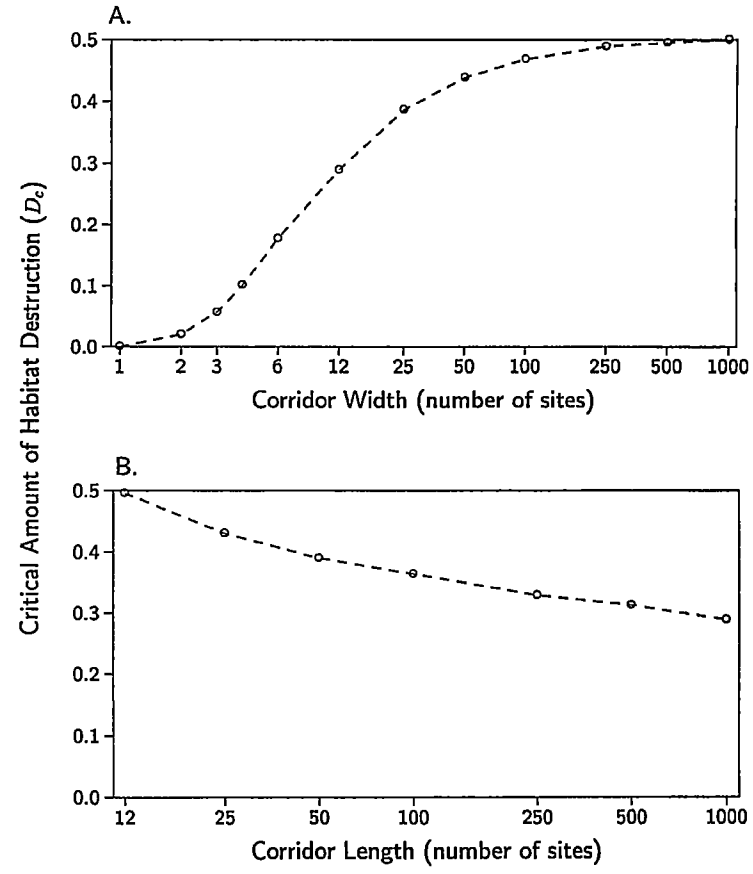


FIGURE 1.6. Numerous runs of a spatial simulator approximating the conditions of Figure 1.6 were performed to determine the critical amount of habitat destruction beyond which a population could not move, on average, through a corridor. (A) Even a strip of virgin habitat cannot function as a corridor if it is too narrow, and it can never function as a corridor, for this species, if it is more than 50% destroyed. (B) Corridor length also influences the extent of habitat destruction for which a corridor can remain viable.

diffusion model could be the logistic equation, or an exponential growth function, or any of ecology's many permutations on models of interspecific interactions. The "diffusion" term in these equations describes the movement of individuals and is usually the mathematical term corresponding to simple passive diffusion, that is,

$$D\frac{\partial^2 N(x,t)}{\partial x^2}, \tag{1.4}$$

for a one-dimensional model, where D is the diffusion coefficient (or dispersal rate) and $N(x,t)$ is the density of individuals at position x and time t. Notice that the entire term equals zero if N is uniform in space or if the spatial variation in N is described by a straight line. Reaction-diffusion equations are widely used in ecology, but their origin is rarely explained, leaving the mistaken impression that they require advanced mathematics to be understood. In fact, Equation 1.4 is easy to appreciate intuitively if we think about a one-dimensional environment—for example, that approximated by nearly sedentary organisms confined to the shoreline of a pond. Consider the shoreline to be divided into many discrete sites along its length, and suppose for the moment that there is no reproduction within the sites—all changes in population come from migration among sites. Concentrate on a given site and suppose that migration is purely random; that is, suppose each individual has a fixed probability of leaving the site in any given interval of time and leaves by moving either to the east or to the west with equal probability. Thus in each small unit of time, part of the population of the given site is lost when a fixed proportion of its occupants move west and an equal proportion move east. However, this loss is counteracted by individuals immigrating from neighboring sites. Because the same processes are assumed to operate in all sites concurrently, a fixed proportion of the number of individuals in the site to the west enters from the west, and similarly for the east. To put these words into mathematical symbols, let N_e be the number of individuals in the site to the east, N_w the number in the site to the west, N_c the number in the given site at the center, and

D the fixed proportion of individuals that leave in each direction per unit time. Then the change in population of the given site in a small unit of time is

$$\frac{\Delta N}{\Delta t} = DN_e + DN_w - 2DN_c. \tag{1.5}$$

Now notice that we can think of N_c as the density at some position x, N_w as the density at $x - (\Delta x/2)$, and N_e as the density at $x + (\Delta x/2)$. This means Equation 1.5 can be rewritten as

$$\frac{\Delta N}{\Delta t} = D\left\{\left[N\left(x+\frac{\Delta x}{2}\right) - N(x)\right] - \left[N(x) - N\left(x-\frac{\Delta x}{2}\right)\right]\right\}. \tag{1.6}$$

After some algebra this is equivalent to

$$\frac{\Delta N}{\Delta t} = D\frac{\Delta(\Delta N)}{\Delta x^2}. \tag{1.7}$$

This is a discrete approximation to

$$\frac{\partial N}{\partial t} = D\frac{\partial^2 N}{\partial x^2}. \tag{1.8}$$

Although this is clearly not a rigorous argument, it provides some intuition to the simple diffusion equation (see Levin and Pacala, Chapter 12, for rigorous mathematical derivations).

A simple reaction-diffusion model that includes logistic growth at any given site (the last term in the equation) and passive diffusive spread is

$$\frac{\partial N}{\partial t} = D\frac{\partial^2 N}{\partial x^2} + rN\left(1 - \frac{N}{K}\right), \tag{1.9}$$

with the x, t notation for each N dropped in order to "unclutter" the equations. Indeed, Equation 1.9 is the "Fisher equation," which has played a prominent role in the theory of

ecological invasions. The population grows as a wave that spreads through a habitat at a speed determined by both D and r (see Lewis, Chapter 3). Although the vast majority of reaction-diffusion spatial theory assumes simple passive diffusion, there are important papers that explore more complex diffusive processes such as density-dependent diffusion, taxis, and convection (see Okubo 1980 for a review). Reaction-diffusion models gain their power from the fact that they treat space as a continuous variable and from the arsenal of analytical and numerical tools that can be brought to bear on partial differential equations. They represent the most compact description of spatially explicit population dynamics. For a biological audience, the main drawback of reaction-diffusion models is their unfamiliarity and the mistaken impression that they are "sophisticated mathematics." A more justified drawback is that analytical results using these equations are hard to come by, except for the simplest models and formulations. When reaction-diffusion models cannot be handled analytically, numerical methods end up effectively treating space as a series of coupled points (or discrete cells) linked by diffusion, with ordinary differential equations describing the dynamics at each point (or cell). In other words, we discretize partial differential equations to solve them numerically and thus should not avoid discrete spatial models on principle alone.

Two themes stand out from the theory of reaction-diffusion models in the context of spatial ecology. First, they have been used with great success to identify how patterns corresponding to predictable spatial variation in population density can be formed in uniform environments. In single species models, these patterns are typically in the form of traveling waves associated with invasions, whereas for interacting species the patterns can end up being fixed in space, so that there are standing waves or standing checkerboards, and so forth. The pattern formation revealed by reaction-diffusion equations has spawned a rich theoretical literature (e.g., see Murray 1989) that provides an excellent guide to similar processes of pattern formation in discrete cellular automata models (Hassell and Wilson, Chapter 4).

The second theme is the illustration of a tight connection between the ability of a population to persist as a function of the size and geometry of habitat islands, and the similar situation for isolated habitat fragments that find themselves surrounded by hostile lands. The mechanism underlying this influence of habitat shape and size is the effect of perimeter-to-area ratios on processes governed by dispersal across habitat boundaries. For instance, the simplest prediction is that if the radius of a circular habitat gets too small, diffusive losses to the surrounding world will outweigh population growth and the population declines deterministically to zero. This phenomenon has been hypothesized as a mechanism explaining plankton patchiness in marine systems and the absence of plankton patches beneath some finite small threshold size—the so-called *critical patch size* (Kierstead and Slobodkin 1953). Recent extensions of this theory involve more complex behaviors than diffusion (such as mixed boundary conditions) and less arbitrary external conditions than absolute instant mortality (Cantrell and Cosner 1993). These more sophisticated models obviously yield different predictions, but it is still variation in perimeter-to-area ratios that underlies the major effects of habitat size and shape.

SUMMARY

All organisms are discrete entities that interact and disperse locally. Space unavoidably causes individuals to differ in both the intra- and interspecific interactions and the resource levels they experience. Although detailed spatially explicit models of population growth and interaction can be cumbersome, three simple approaches can abstract different aspects of the essence of spatial ecology. The Levins model is only implicitly spatial because it assumes global dispersal, but this makes it analytically tractable. It often provides a reasonable approximation to explicitly spatial simulators, such as cellular automata. One of its interesting predictions is that no species living in a spatial habitat can occupy all sites at equilibrium, which has profound implications for biodiversity (Tilman 1994; Lehman and Tilman, Chapter 8). Cellular automata are created when

Levins-like models are made one step more realistic by including local dispersal in a habitat divided into distinct cells or sites. A range of new phenomena appear, chief among them spatial patterning. A species living in a homogeneous but spatial habitat will take on a spatially clumped distribution. The degree of local dispersal influences the equilibrial population size of species (including ability to persist). Local dispersal also leads to percolation effects that may mean that habitats need to be of higher quality to function as corridors than as preserves. An alternative approach is to consider space a continuous variable and to approximate dispersal as a diffusion process. Such reaction-diffusion models can be analytically tractable and also provide a rigorous framework within which to numerically explore the implications of different assumptions about the nature of species interactions, the dispersal process, and habitat boundaries and conditions. These three approaches are the major tools with which spatial ecology is explored throughout this book.