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PARTIAL DIFFERENTIAL EQUATIONS IN ECOLOGY: SPATIAL INTERACTIONS AND POPULATION DYNAMICS[1]

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Abstract. Most of the fundamental elements of ecology, ranging from individual behavior to species abundance, diversity, and population dynamics, exhibit spatial variation. Partial differential equation models provide a means of melding organism movement with population processes and have been used extensively to elucidate the effects of spatial variation on populations. While there has been an explosion of theoretical advances in partial differential equation models in the past two decades, this work has been generally neglected in mathematical ecology textbooks. Our goal in this paper is to make this literature accessible to experimental ecologists.

Partial differential equations are used to model a variety of ecological phenomena; here we discuss dispersal, ecological invasions, critical patch size, dispersal-mediated coexistence, and diffusion-driven spatial patterning. These models emphasize that simple organism movement can produce striking large-scale patterns in homogeneous environments, and that in heterogeneous environments, movement of multiple species can change the outcome of competition or predation.

Key words: coexistence; differential equations; diffusion; diffusive instabilities; dispersal; ecological models; invasions; reaction–diffusion; spatial models; spatial patterning.

Introduction

Ecologists are paying increasing attention to spatial processes in a wide variety of practical contexts. For example, landscape ecology and conservation biology focus on the consequences of habitat fragmentation, and geographic information systems have recently made available large stores of spatially structured data. Prior to this fascination with the spatial dimension by applied ecologists, several theoretical community ecologists explored the consequences of dispersal and habitat heterogeneity with respect to patterns of diversity (e.g., Levin 1978, Wiens 1986). One of the major mathematical tools for analyzing spatiotemporal processes is partial differential equations or PDEs. While mathematical ecology is now richly imbued with theoretical insights obtained from PDE models, most experimental ecologists have not kept up with these theoretical advances. One reason for the weak connection between PDE models and experimentation is unfamiliar jargon and notation. While ordinary differential equations and difference equations are usually described in basic introductory ecology textbooks, PDEs are often not described even in mathematical ecology texts (see, for example, Poole 1974, Vandermeer 1981, Yodzis 1989). To help remedy this situation, we present here an introduction to the major theoretical insights gleaned from PDE models in ecology, paying particular attention to both the applications and limitations of the theory. We do not strive for mathematical rigor, but rather try to present the mechanistic underpinnings of classical PDE models in a biologically intuitive way.

While PDEs that are sufficiently realistic to be used in ecological models are usually more difficult to solve than ordinary differential equations, they are advantageous because they allow modellers to incorporate both temporal and spatial processes simultaneously into equations governing population dynamics. Although PDE models obviously cannot describe all ecological situations (see *Discussion*), they do lend insight into numerous fundamental population processes, five of which we emphasize here: dispersal, ecological invasions, the effect of habitat geometry and size, dispersal-mediated coexistence, and the emergence of spatial patterns.

[1] For reprints of this Special Feature, see footnote 1, p. 1.

Models for Dispersal

In the classical applications of PDE models to population ecology, organisms are assumed to have Brownian random motion, the rate of which is invariant in time and space. This assumption leads to the diffusion model (Okubo 1980, Edelstein-Keshet 1986, Murray 1989):

$$\frac{\partial u(x, y, t)}{\partial t} = D\left(\frac{\partial^2 u}{\partial x^2} + \frac{\partial^2 u}{\partial y^2}\right), \tag{1}$$

where $u(x, y, t)$ is the density of organisms at spatial coordinates x, y, and time t, and D is the diffusion coefficient that measures dispersal rate, with units $\frac{\text{distance}^2}{\text{time}}$. When animals are released at a central point and disperse via diffusion in a two-dimensional environment, their resulting distribution in space is Gaussian with a mean squared displacement (MSD) of $4tD$. One can also use Eq. 1 to predict the location of a single individual at some particular distance from a starting point. For example, the average time for an organism to travel a distance, L, away from its starting position is

$$\frac{L^2}{2D}\ln\left(\frac{L}{a}\right).$$

Thus, the average organism takes a very large time to travel long distances via diffusion. On the other hand, the diffusion model (Eq. 1) also predicts that there is a small finite probability that an individual travels an arbitrarily long distance in arbitrarily short time. These seemingly contradictory predictions depend upon whether we are concerned with the average behavior of individuals or with events that are possible, although highly improbable.

Although the diffusion equation is simplistic, it has been used to effectively describe the movement of numerous animals in mark–recapture studies (e.g., Dobzhansky and Wright 1943, Johnston and Heed 1976, Taylor 1980, Kareiva 1982). The model works best when the environment itself is homogeneous (Kareiva 1983) and the individuals in the population have similar movement rates. When animals orient toward external stimuli or are carried by the wind or water currents, drift or convection terms are added to Eq. 1, resulting in the model (Helland et al. 1984, Banks et al. 1988):

$$\frac{\partial u}{\partial t} = D\left(\frac{\partial^2 u}{\partial x^2} + \frac{\partial^2 u}{\partial y^2}\right) - w_x\frac{\partial u}{\partial x} - w_y\frac{\partial u}{\partial y}, \tag{2}$$

where w_x and w_y are drift velocities.

A more fundamental departure from simple Brownian motion derives from the fact that animals do not zig-zag back and forth wildly like molecules, but instead tend to continue forward in the direction of their existing path. This process, referred to as a correlated random walk, leads to a PDE model known as the telegraph equation:

$$\frac{\partial u}{\partial t} = \frac{s^2}{2\lambda}\left(\frac{\partial^2 u}{\partial x^2} + \frac{\partial^2 u}{\partial y^2}\right) - \frac{1}{2\lambda}\frac{\partial^2 u}{\partial t^2}, \tag{3}$$

where $\frac{1}{2\lambda}$ is a measure of the correlation between directions of travel from one step to the next and s is the velocity of the organisms (Goldstein 1951, Othmer et al. 1988, Holmes 1993). The mean squared displacement for animals obeying the telegraph model is

$$\text{MSD} = s^2\left[\frac{t}{\lambda} - \frac{1}{2\lambda^2}(1 - e^{-2\lambda t})\right]. \tag{4}$$

Comparison with the MSD for simple diffusion shows that, with time, the Brownian random component dissipates the effects of the correlated motion and the two MSDs converge (with $D = s^2/2\lambda$, see Holmes 1993). This model is distinguished from the simple diffusion model not only by correlated direction of motion but also by finite animal velocity. This leads to a bounded distribution of animals because no animal is able to go an infinite distance in a finite amount of time.

There are several PDE models of movement that can be derived by including much more behavioral detail than is implicit in the diffusion or telegraph equations. These models, which can be traced back to work by Patlak (1953), incorporate the precise distributions of turning angles, pauses in motion, changes in velocity and step length, and drift in a particular direction (Okubo 1980, Doucet and Wilschut 1987, Turchin 1989*b*, 1991). Such details of animal movement are much more likely to be important if behaviors of interest occur during short times after movement commences, such as for pollen dispersal (Morris 1993).

It is straightforward to alter these PDE models so that they also encompass interactions between conspecifics. For example, if animals are either attracted to one another or repelled from one another, then the simple diffusion model can be replaced by a biased random motion model (Gurney and Nisbet 1975):

$$\frac{\partial u}{\partial t} = D\frac{\partial^2 u}{\partial x^2} + \frac{\partial}{\partial x}\left(ku\frac{\partial u}{\partial x}\right), \tag{5}$$

where u is population density, and k is a measure of the tendency to move away from conspecifics when $k > 0$ and is a measure of the tendency to move towards conspecifics when $k < 0$. The strength of attraction (or avoidance) is ku and thus is a linear function of density. This model leads to clumping of organisms if the aggregation component dominates the random component and a distribution of animals that is bounded, implying that animals spread at a finite speed. The

microscale animal motion that produces this model is movement preferentially toward neighboring areas of more favorable density or movement merely in response to density at the animal's current location (Gurney and Nisbet 1975, Shigesada et al. 1979, Okubo 1986). Because k is a constant in Eq. 5, this model describes animals that aggregate toward (or avoid) conspecifics at all population densities. In reality, organisms often aggregate at low densities and avoid one another at high densities. Such a density-dependent response can be modelled by replacing Eq. 5 with (Aronson 1980, Turchin 1989*a*)

$$\frac{\partial u}{\partial t} = D\frac{\partial^2 u}{\partial x^2} + \frac{\partial^2 \psi(u)}{\partial x^2}, \tag{6}$$

where $\psi(u)$ is a function that is negative at low density and positive at high densities. In particular, $\psi(u)$ allows for a much more general relationship between aggregation tendency and density.

In addition to modifying their movement in response to conspecifics, many organisms also alter their movement in response to environmental heterogeneity. This complication leads to a suite of models analogous to Eqs. 5 and 6, except with some feature of the physical environment driving the movement. In particular, a PDE model of animals that assess and move to higher quality neighboring areas or move based on quality at their current location is

$$\frac{\partial u}{\partial t} = \frac{\partial}{\partial x}\left[u\frac{d}{dx}\phi(E)\right], \tag{7}$$

where E is the environmental potential that increases as habitat quality decreases, and the function $\phi(E)$ describes how environment alters movement behavior (Shigesada et al. 1979, Shigesada 1980, Okubo 1986, Brew 1987). This model leads to a distribution of animals that is aggregated where E is lowest, i.e., where habitat quality is greatest. Another, perhaps intuitive, model of animals that vary their movement rate in response to habitat quality is

$$\frac{\partial u}{\partial t} = \frac{\partial}{\partial x}\left[D(E)\frac{\partial u}{\partial x}\right]. \tag{8}$$

It is important to note, however, that Eq. 8 mechanistically models animals that move according to the average quality between their current location and neighboring sites and produces a homogeneous distribution of animals. It can be argued that this type of spatial averaging movement is an accurate model for many animals. One method for modelling animals that have such averaging movement, yet still aggregate on high quality areas, is to describe the population as two subpopulations, one that is moving and the other that is permanently or temporarily not moving (Morris and Kareiva 1991). In this case, the probability of changing from the moving into nonmoving class or vice versa is dependent on environmental quality.

In summary, these permutations on simple diffusion models emphasize that although most applications of PDEs to spatial processes assume diffusion, there is a rich theoretical framework for addressing more complex movement behavior. To a certain extent, however, models of movement alone are of limited interest. In the following sections, we discuss reaction–diffusion models in which movement is combined with population dynamics and multispecies interactions. It is via these types of models that PDEs have produced many salient results concerning the dynamics arising from the interplay of movement and population interactions. Reaction–diffusion models take the form:

$$\frac{\partial u}{\partial t} = \frac{\partial}{\partial x}\left[D(u)\frac{\partial u}{\partial x}\right] + \frac{\partial}{\partial y}\left[D(u)\frac{\partial u}{\partial y}\right] + f(u), \tag{9}$$

where the first two terms represent diffusive movement and the last term, $f(u)$, is the reaction term and describes population growth dynamics. Although this theory typically assumes that dispersal takes the form of Brownian random motion, many of the same results discussed below still emerge if diffusion is modified to include less simple behavior (as in Eqs. 3, 5, 6, and 7).

Invasion Models

The first formal mathematical attempts to model ecological invasions via reaction–diffusion equations were made by Skellam (1951) who modelled the expansion of muskrat populations in Europe. His model of invading animals with diffusion movement and Malthusian growth predicts that the area occupied by an invader will increase linearly with time (cf. Okubo 1980). This model has been used successfully to describe the historical range expansion of European Starling, English Sparrow, and House Finch (Okubo 1988), the Collared Turtle Dove (Hengeveld 1989), the Himalayan thar (Caughley 1970), the grey squirrel in England (Okubo et al. 1989), and the California sea otter (Lubina and Levin 1988).

Reaction–diffusion invasion models exhibit more striking behavior when population growth is not exponential but instead is regulated by density-dependent mortality. These models produce travelling waves of invaders that spread out from their "beachhead" at a constant velocity and shape (Fig. 1). Travelling waves are a common feature of many reaction–diffusion models. The classic reaction–diffusion model of ecological import is the Fisher model (Fisher 1937), which represents logistic population growth plus Brownian random dispersal (Skellam 1951):

$$\frac{\partial u}{\partial t} = ru\left(1 - \frac{u}{K}\right) + D\left(\frac{\partial^2 u}{\partial x^2} + \frac{\partial^2 u}{\partial y^2}\right), \tag{10}$$

where r is a population's intrinsic rate of growth and K is the carrying capacity. This model produces waves of invaders that travel at a velocity $c(t)$, which approaches $\sqrt{4rD}$ as the invasion progresses (Kolmogoroff et al. 1937, Uchiyama 1978). The invasion velocity quickly approaches this asymptotic speed; at time t, after the release, $c(t) \approx \sqrt{4rD} - b(\frac{1}{t} \ln t)$, where b is some constant (Bramson 1978).

If the Fisher model (Eq. 10) is analyzed in two dimensions, circular waves form and spread outward (Fig. 1). The asymptotic travelling wave speed calculated in the one-dimensional case is also the asymptotic wave speed in the two-dimensional case. Opposite to the one-dimensional case, the wave speed is slower shortly after the release of the organisms and increases toward the asymptotic velocity as the invasion progresses. For a refined version of Fisher's model in which an Allee effect is added to the growth function (see below), wave speed has been shown to be a function of the shape of the wavefront. For segments of the front with higher curvature and that thus protrude ahead, the wave speed is slower. For segments with lower curvature and that thus lag behind, the wave speed is greater. This effect drives noncircular wavefronts toward circularity (Lewis and Kareiva 1993). Fisher's model has been used to make predictions of range expansion using microscale data on individual movement for a variety of animals: working well with cabbage butterflies, muskrats, grey squirrels, and neolithic farmers (Ammerman and Cavalli-Sforza 1984, Okubo et al. 1989, Andow et al. 1990) and underestimating speeds for cereal leaf beetles (Andow et al. 1990).

Surprisingly, the simple equation for the asymptotic invasion velocity for the Fisher model is not restricted to logistic population growth, but more generally arises as

$$\text{asymptotic velocity} = \sqrt{4f'(0)D}, \qquad (11)$$

where $f(u)$ is a general class of population growth functions of which the logistic equation is only one specific example. $f'(0)$, the rate of population growth at very low population density, is the first derivative of the population growth function evaluated at zero population density, $u = 0$. Loosely speaking, Eq. 11 holds as long as the population growth function satisfies two criteria: (1) the growth rate is positive when the population is below the carrying capacity and (2) the maximum per capita growth rate is found when the population is small (Hadeler and Rothe 1975, Fife 1979).

A formula similar to Eq. 11 for the wave speed of an invasion has been obtained in models for invasions into a heterogeneous environment. The analysis was done by examining the dynamics associated with Eq. 10 in a one-dimensional environment, and allowing both r and D to vary periodically in space (Shigesada et al. 1986). A travelling wave of invasion is still obtained, but its asymptotic velocity is

$$\sqrt{4r_a D_h},$$

where r_a is the arithmetic mean for intrinsic rate of increase and D_h is the harmonic mean for the diffusion coefficient. Two interesting points emerge from this analysis. First, the invasion velocity is determined by the rates of population growth and diffusion. Second, the presence of the harmonic mean for diffusion indicates that spatial variation in dispersal can greatly deter spread, since harmonic means are much lower than arithmetic means when variability is substantial. Although Shigesada et al.'s (1986) results pertain to a specific model and specific form of heterogeneity, they suggest it is possible to obtain general guidelines for summarizing the effects of heterogeneity on spatial processes such as invasions.

A final refinement in invasion theory involves the relaxation of restrictions on population growth functions, in particular, by the addition of an Allee effect

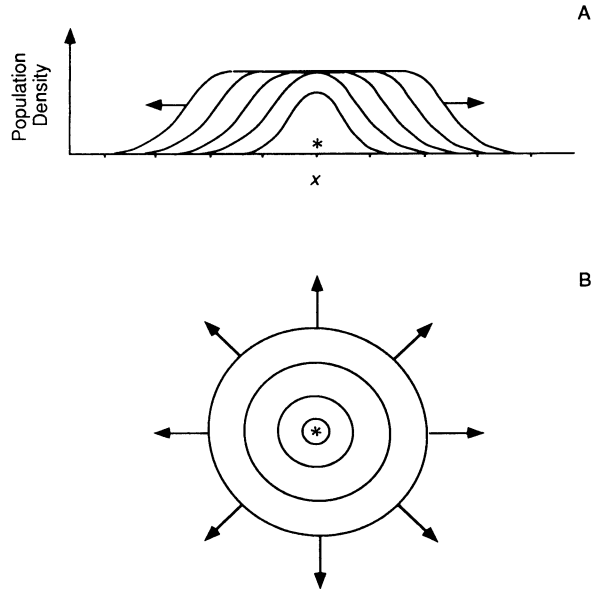

FIG. 1. Examples of travelling waves. Populations that disperse outward and reproduce can spread as a wave into previously unoccupied areas. (A) shows successive snapshots of such a wave moving in one dimension, for example, along a coastline or river. Organisms are released at the asterisk and each line is a snapshot of the population density along the x axis. The lines that are farther from the release point are later in time. (B) is similar to (A) but represents spread in two dimensions. The circles represent the extent of the organisms' range at successive times. Note that in the strict mathematical sense, these are not travelling waves because the shape (i.e., curvature) changes with time.

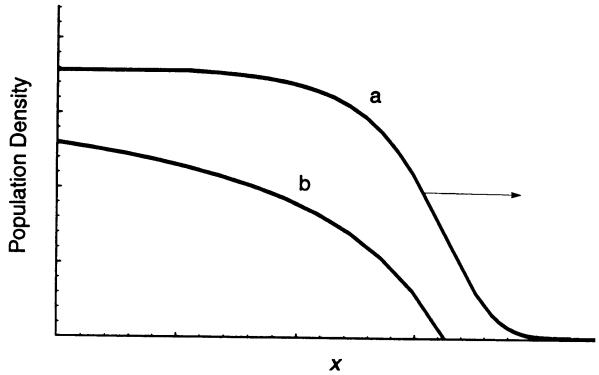

FIG. 2. Comparison of the travelling wave fronts produced by a Fisher invasion model (front a) with density-independent dispersal and that produced by an invasion model with density-dependent dispersal via the directed random motion model (front b).

(Allee 1931), whereby at low densities population growth is negative. Models involving population growth with an Allee effect plus diffusive dispersal also produce travelling waves of invaders (Aronson and Weinberger 1975, Fife and McLeod 1977, Lewis and Kareiva 1993). The key differences that arise with an Allee effect are threefold: (1) some threshold density must be exceeded before an invasion takes hold, (2) the initial spatial arrangement of invaders influences the fate of an invasion, and (3) assuming the threshold density has been exceeded, the velocity of spread is reduced in proportion to the Allee effect.

Earlier we mentioned that although most reaction–diffusion models assume simple diffusion, some of the key ideas would emerge without restricting dispersal to Brownian random motion. Invasion theory offers a good example. If the Fisher model (Eq. 10) is modified such that the assumption of diffusive movement is replaced by a correlated random walk (i.e., telegraph equation), travelling waves of invaders still result, but the waves travel at the slower asymptotic wave speed of

$$s\sqrt{\frac{8r\lambda}{(r+2\lambda)^2}}.$$

This wave speed departs from the classical wave speed in proportion to the product of the intrinsic rate of population growth and the correlation between directions of travel (Holmes 1993). Estimation of the two parameters for organisms as varied as bacteria, butterflies, and muskrats suggests that the actual magnitude of differences between wave speed predictions from diffusive as opposed to correlated walk movement typically are <5%.

Other "nondiffusive" extensions of invasion models have examined convective movement (Lewis and Kareiva 1993) and density-dependent dispersal, such as in Eq. 5 with $k > 0$. In the latter case, travelling waves are also obtained with an asymptotic velocity of $\sqrt{2rk}$ (Aronson 1980). Here, k represents the degree to which high density increases avoidance. As might be expected, increasing avoidance, k, increases the invasion wave velocity. However, the shape of the travelling wave is strikingly different when animals disperse via density-dependent motion (Fig. 2).

Species invasions have also been modelled in more complex settings that include interactions between species. To date such extensions generally have been restricted to pairwise interactions. For instance, consider the spread of two competing species using the coupled PDEs:

$$\frac{\partial u}{\partial t} = D_u\frac{\partial^2 u}{\partial x^2} + (r_u - \alpha_{uu}u - \alpha_{uv}v)u$$

$$\frac{\partial v}{\partial t} = D_v\frac{\partial^2 v}{\partial x^2} + (r_v\ \alpha_{vv}v - \alpha_{vu}u)v, \qquad (12)$$

where u and v are the densities of the two species, D_u and D_v are species-specific diffusion rates, r_u and r_v are species-specific intrinsic rates of increase, and the α's represent interspecific and intraspecific competition coefficients (rescaled so that carrying capacity does not appear in the equation). According to the model, if the two species are introduced into unoccupied habitat, they can spread across the environment as two travelling waves with the wave of the faster reproducer moving ahead of the slower (Bramson 1988). A more biologically plausible example corresponds to an exotic species, v, invading an area completely occupied by a native competitor, u. In this case, species v invades with a wave speed less than its maximum possible speed since it spreads into territory already occupied by species u. The wave speed of species v is denoted by c_v and is given by (Bramson 1988)

$$2\sqrt{\left(r_v - \alpha_{vu}\frac{r_u}{\alpha_{uu}}\right)D_v} \geq c_v < 2\sqrt{r_vD_v},$$

where $\left(r_v - \alpha_{vu}\frac{r_u}{\alpha_{uu}}\right)$ represents the population growth rate of species v in the presence of species u when u is at carrying capacity. The term $2\sqrt{r_vD_v}$ is the maximum wave speed of species v and this speed occurs when the species spreads into empty territory. Okubo et al. 1989 used the same ideas to model the invasion of grey squirrels across the habitat occupied by red squirrels in Britain.

Similar analyses have been applied to predator–prey interactions using a Lotka-Volterra predator–prey model with diffusion (Murray 1975, Dunbar 1983, Murray 1989):

$$\frac{\partial u}{\partial t} = D_u \frac{\partial^2 u}{\partial x^2} + ru\left(1 - \frac{u}{K}\right) - \alpha_{uv} uv$$

$$\frac{\partial v}{\partial t} = D_v \frac{\partial^2 v}{\partial x^2} - \mu v + \alpha_{vu} uv, \qquad (13)$$

where μ is the per capita mortality rate of predators in the absence of prey, α_{uv} represents the rate at which predators consume prey, and α_{vu} represents the rate at which predators convert prey into new predators. If both predator and prey colonize an environment that is initially empty, their spread cannot be mathematically represented by a travelling wave. The spread in this case does form wave-like patterns (which eventually damp out), resembling those found in some zooplankton–phytoplankton interactions (Dubois 1975). If the prey already exists uniformly across the environment and a predator is released, the situation is analogous to the spread of disease through susceptible hosts, and travelling waves of predator and prey will ensue (Fig. 3b). Again the asymptotic wave speed of the predators (as they eat the prey) is determined by the predator population growth rate and diffusion rate.

Reaction–diffusion models have also been used to investigate the spatial spread of diseases (Kendall 1948, 1965, Bailey 1967, Mollison 1972, Atkinson and Reuter 1976, Aronson 1984, Källen et al. 1985; Fig. 3). Epidemiological models that investigate the spatial invasion of a disease have been applied to cholera, plague (Noble 1974), rabies (Murray et al. 1986, Murray and Seward 1992), and moth viruses (Dwyer 1993).

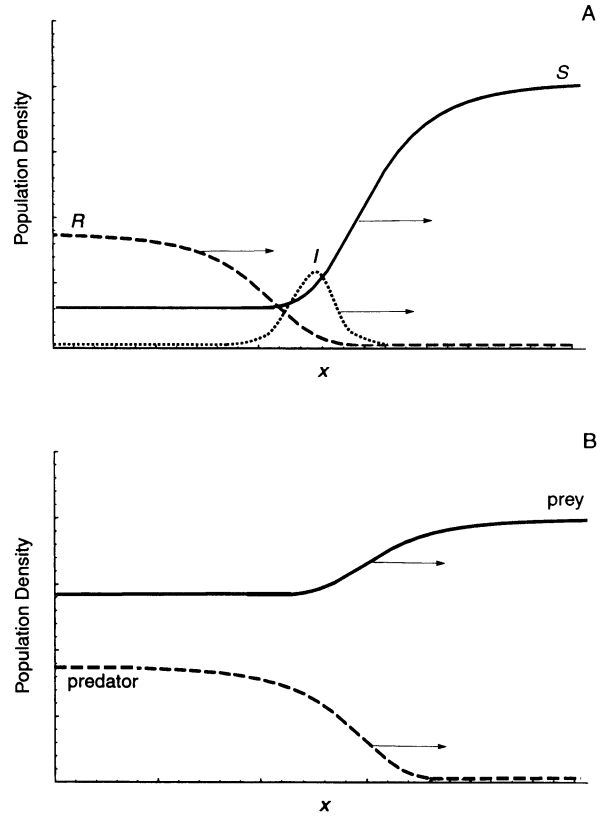

FIG. 3. Examples of travelling wave fronts produced by an SIR disease model and a Lotka-Volterra predator–prey model. The waves move through space with constant shape and velocity in the direction shown by the arrows. In the disease model, an infection (I) spreads spatially through a population of susceptible (S) individuals. The infected individuals eventually die or become immune (R). In the predator–prey model, predators invade a region occupied by prey.

POPULATION DYNAMICS IN "ISLAND" HABITATS

PDEs provide a natural framework for investigating the influence of patch size and geometry on the population dynamics of organisms living within habitat patches (McMurtrie 1978, Okubo 1984). Primarily, PDE models have been used to look for critical patch sizes, that is, the smallest patch that can (minimally) sustain a population. It exists because as the patch decreases in size, the ratio of perimeter to interior area increases and the relative impact of the edge increases until at some critical patch size the patch is too small to sustain the population. As expected, the critical patch size depends on a number of factors, including the population dynamics in the patch, the rate at which organisms leave the patch, the degree to which the region outside the patch is lethal, and patch geometry.

The basic critical patch model is known as the KISS model after Kierstead and Slobodkin (1953) and Skellam (1951) and was originally developed to investigate the size of nutrient patches needed to sustain phytoplankton blooms. The model assumes an exponentially growing population that disperses randomly within and out of a patch into lethal habitat:

$$\frac{\partial u}{\partial t} = D\left(\frac{\partial^2 u}{\partial x^2} + \frac{\partial^2 u}{\partial y^2}\right) + ru. \qquad (14)$$

The KISS model predicts a critical patch area given by

$$\text{critical patch area} = c_0 \pi^2 \left(\frac{D}{r}\right), \qquad (15)$$

where $c_0 = 1.84$ for a circular patch and $c_0 = 2$ for a square patch (Okubo 1984, Murray 1989). A natural extension of these models is to incorporate population growth that is depressed by high population densities. However, although such density-dependent effects depress population growth rates compared to exponential growth, the density-dependent population growth is irrelevant to critical patch size because near the critical patch size, the patch sustains a very low population density and density-dependent effects are minimal. On the other hand, density-dependent population growth

does dramatically influence the size of the population in the patch. For example, a population with logistic growth will have a maximum population density close to zero when the patch is slightly larger than the critical size given by Eq. 15. The maximum population density will approach the population's carrying capacity as the patch approaches infinite size. In general, for populations that (1) are within patches with lethal boundaries, (2) have reaction–diffusion dynamics, and (3) have convex growth functions, i.e., $f(u) > 0$ for $0 < u <$ carrying capacity, $f(u) < 0$ for $u >$ carrying capacity, and $f'(u)$ at a maximum at $u = 0$, the critical patch area is given by

$$A_{cr} = c_0\pi^2[D/f'(0)], \tag{16}$$

where c_0 is a constant determined by the shape of the patch (Skellam 1951, Landahl 1959, Okubo 1984; see Platt and Denman 1975 for an application). In general, the critical patch area is smallest for circular areas and increases as the area deviates from circularity (Ludwig et al. 1979, Cantrell and Cosner 1989, 1991*a, b*).

Much research has focused on similar analyses applied to less restrictive idealizations of habitat islands and organism growth and movement. In particular, relaxing the restriction of a completely hostile patch boundary results in a smaller critical patch size. For example, if the zone outside the patch is only partially hostile or organisms avoid leaving the patch, then as expected, the critical patch size is diminished so that a smaller patch can sustain a population (Ludwig et al. 1979, see also Okubo 1984). Partially hostile boundaries are represented by so-called mixed boundary conditions, and model situations where some individuals will cross the boundary but others won't, situations where organisms will cross the boundary sometimes but not at other times, situations where organisms readily cross the boundary but the region outside the patch is only partially hostile, or any combination of the above. In lieu of distinct boundaries, it is also possible to analyze a situation in which habitat quality is positive in some central region and becomes more and more hostile away from the center. In this circumstance, the population will have a peak density in the region where the growth rate is positive and the population density falls away from the positive region. The results for this case with random diffusion are not dramatically different from the case of a patch with lethal boundaries, i.e., critical area $\propto D/\max[f'(0)]$ (Gurney and Nisbet 1975).

Nonrandom movement has varied effects on the critical patch size. Organisms may be convectively carried out of the patch by the wind or water currents and, in this case, will require larger patch sizes to compensate for increased migration from the patch. A curious spatial patterning can occur if there is convection and the boundaries of the patch are only slightly hostile. In this case, multiple peaks of population density are possible (Murray and Sperb 1983). Alternatively, dispersal rates may be a function of organism density. In particular, if organisms move via biased random motion (Eq. 5), then the critical patch size is determined by the density-independent (Brownian) component of movement and specified by Eq. 16 (Gurney and Nisbet 1975). The density-dependent movement is irrelevant to the critical patch size, since near the critical patch size the population density in the patch is very low. While density-dependent movement does not affect population sustainability in small patches, it does have a strong effect on the density in large patches. In particular, density-dependent movement will tend to regulate population sizes in large patches by increasing dispersal as crowding intensifies. A more substantial modification in the critical patch size is produced by alternate growth dynamics. In particular, for Allee growth dynamics (i.e., negative growth rate at low densities), a patch must exceed a larger critical patch size in order to ensure that the minimum population density necessary for a positive growth rate is attained (Bradford and Philip 1970*a, b,* Okubo 1980, Murray 1989).

In general, the many different versions of critical patch size theory share three key points: (1) factors that increase movement out of a patch (drift or repulsion) lead to larger critical patch sizes, while (2) factors that decrease movement out of the patch (attraction or density-dependent dispersal) lead to smaller critical patch sizes. Finally, (3) density-dependent growth rates regulate the population size within the patch but do not affect the critical patch size unless the population has Allee growth dynamics.

Dispersal-mediated Coexistence

Identifying mechanisms that promote the coexistence of competitors is one of the major challenges before ecologists. Naturalists have often noted that the interplay of dispersal and the spatial dimension somehow seem to facilitate coexistence, but the details of how this happens can be complex and varied. In the context of reaction–diffusion models, if the environment is uniform and dispersal is via simple diffusion, then dispersal has no effect on coexistence (Pao 1981, Brown 1984). When these assumptions are altered, dispersal can enhance coexistence. For example, Mimura et al. (1991) showed if two competing species live on two islands connected by a small corridor then the two species may coexist by segregating onto different islands. In the absence of dispersal, either could outcompete the other depending on initial densities (cf. Levin 1974).

However, multiple patches are not necessary for dispersal-mediated coexistence to occur. Indeed, dispers-

al-mediated coexistence will occur in a homogeneous, one-patch environment when the two species exhibit dispersal behaviors that are more complex than simple diffusion. In particular, aggregation behaviors have been modelled that promote coexistence primarily by the aggregation of conspecifics, which causes intraspecific competition to greatly exceed interspecific competition (Britton 1989). Similarly, aggregation promotes dispersal-mediated coexistence in heterogeneous environments when competitors have differential dispersal rates, growth rates, or response to increased density of conspecifics (Shigesada et al. 1979, Shigesada and Roughgarden 1982, Bertsch et al. 1984, 1985). On the other hand, simple avoidance behaviors will also foster coexistence. In particular, the cross-diffusion model,

$$\frac{\partial u}{\partial t} = D_u \frac{\partial^2 u}{\partial x^2}[\alpha_{uu} uu + \alpha_{vu} uv] + f_u(u)$$

$$\frac{\partial v}{\partial t} = D_v \frac{\partial^2 u}{\partial x^2}[\alpha_{vv} vv + \alpha_{uv} uv] + f_v(v),$$

describes organisms that avoid each other by increasing their dispersal rate in response to both conspecifics and nonconspecifics. The model predicts competitive coexistence of two species if intraspecific avoidance has a greater impact on population density than interspecific avoidance (Namba 1989).

Given the intuition of naturalists regarding niche partitioning and coexistence, it is probably not surprising that many different reaction–diffusion formations indicate mechanisms for coexistence when heterogeneous environments are considered. For example, if regions vary immensely in quality and the "better competitor" squanders more individuals through dispersal than does an inferior competitor, then the combination of dispersal and heterogeneity can ensure a coexistence that would otherwise be impossible (Pacala and Roughgarden 1982). Taken to the extreme, this example could be viewed as a variation on the "critical patch size" formulation, with the question of interest being the critical patch size for coexistence. In particular, if a competitively inferior species is less sensitive to patch size via a lower ratio of dispersal to growth rate, then it may coexist with superior competitors at intermediate patch sizes and, in fact, can dominate at small patch sizes (Cantrell and Lazer 1984; C. Cosner, *personal communication*). It is important to note that PDE examinations of dispersal-mediated coexistence focus on very different mechanisms than do cellular automata or metapopulation models (Molofsky 1994, Tilman 1994). In particular, PDE models tend to identify coexistence scenarios that depend on either differential sensitivities of competing species to heterogeneous environments or nonrandom movement of competitors. In contrast, most field ecologists would probably argue that dispersal is important to coexistence because it allows inferior competitors to colonize recently disturbed vacant space, a scenario best embodied in metapopulation theory (Tilman 1994).

Pattern Formation in Homogeneous Environments

An aspect of reaction–diffusion theory that has excited numerous applied mathematicians is the realization that simply adding diffusion to certain types of multispecies interactions will cause striking spatial patterns to emerge even in homogeneous environments (see Murray 1989). In an ecological setting, the key idea is that the interaction of dispersal and certain types of population kinetics can amplify perturbations into predictable spatial patterns. These dispersal-driven patterns are currently gaining attention as a result readily generated in cellular automata models (Hassell et al. 1991, Comins et al. 1992). Dispersal-driven patterns are equally readily generated in PDE models. An ecological appreciation of these patterns, known in the PDE literature as diffusion-driven instabilities, originated from studies of predator–prey dynamics that might generate the patchy plankton distributions observed in a seemingly homogeneous environment (Segel and Jackson 1972, Levin and Segel 1976). Subsequently, a large amount of research (Conway 1984, Edelstein-Keshet 1986, Murray 1989) has established the general conditions under which spatial patterns are generated in predator–prey systems of the form:

$$\frac{\partial u}{\partial t} = f_u(u) - \alpha v g(u) + D_u\left(\frac{\partial^2 u}{\partial x^2} + \frac{\partial^2 u}{\partial y^2}\right),$$

$$\frac{\partial v}{\partial t} = \beta v g(u) - f_v(v) + D_v\left(\frac{\partial^2 v}{\partial x^2} + \frac{\partial^2 v}{\partial y^2}\right), \qquad (17)$$

where u is the prey density and v the predator density, f_u is the population growth function for prey in the absence of predators, f_v is the population decline function for predators in the absence of prey, $g(u)$ is the functional response that describes how rates of predation vary with prey density, and the coefficients α and β scale prey losses or predator gains by some constant proportion that reflects conversion of prey into predators, instances of unsuccessful predation attempts, and so forth. The formation of diffusion-driven patterns can be understood in terms of an activator–inhibitor system, in which increases in prey induce (activate) the production of more prey and more predators, whereas increases in predators reduce (inhibit) further predator and prey production. In the absence of dispersal, the prey and predator arrive at a stable equilibrium so that any increase in prey is consumed by the predator, and any increase in predator is reduced

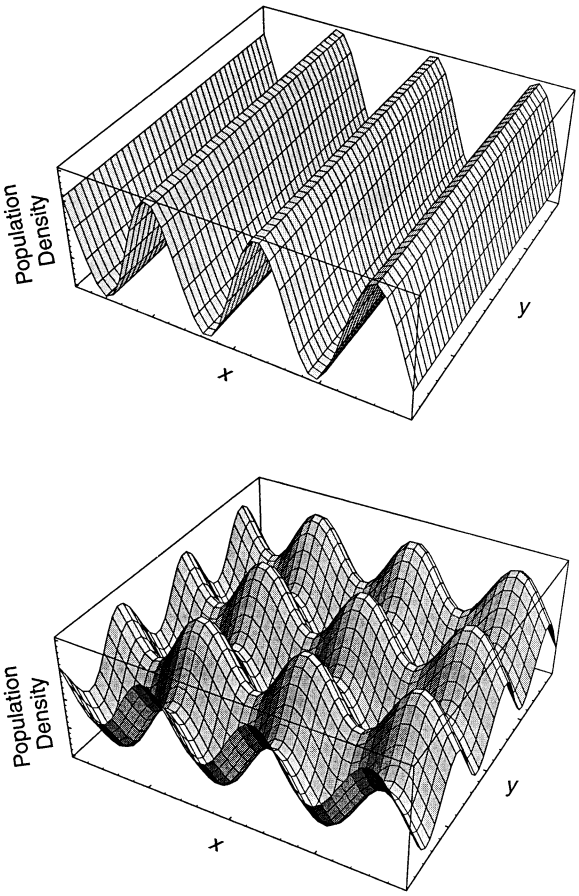

FIG. 4. An aerial view illustrating the type of patterns that can be formed by diffusion-driven instabilities. These are typical patterns for a predator–prey model in which both the predator and prey are moving via a diffusion process. These patterns are generated purely by the dynamics of the interactions between the predator and prey and not by any underlying heterogeneity in the environment. The specific pattern formed, that is, strips or a checkerboard pattern, depends on the size and shape of the patch in which predator and prey live and on the specifics of the population growth rates and diffusion rates.

by self-limitation. When diffusion is added and the diffusion rate of the predator is sufficiently greater than the diffusion of the prey, then the stabilizing influence of the predator may be dissipated by diffusion, yielding regular peaks and troughs of prey and predator densities (Fig. 4).

For a wide variety of biologically plausible growth functions, f_u and f_v, and predator functional responses, $g(u)$, this predator–prey system easily produces these periodic spatial patterns. The key ingredients for pattern formation are (1) the predator disperses faster than the prey, (2) at low densities, an increase in prey density tends to increase the net rate of prey population growth, and (3) an increase in predator densities decreases both prey and predator population growth. Criterion (3) is almost always satisfied for predator–prey systems, whereas criterion (2) requires more specifically either that prey population growth is by itself autocatalytic or prey are consumed according to a saturating functional response such that increases in prey density imply reduced per capita predation risk. Thus, two particular cases under which pattern is readily formed are (a) if prey have Allee growth dynamics (Mimura and Murray 1978, Conway 1984) and (b) if predators experience density-dependent mortality and exhibit a Type II functional response (Levin 1977, Okubo 1980).

The striking aspect of this result is that these spatially periodic patterns are formed despite Brownian random motion of both predator and prey in a homogeneous environment. More realistic predator movement, as in predator aggregation toward prey (preytaxis), can either promote or prevent the formation of spatial patterns; the effect depends on the strength of preytaxis response (Kareiva and Odell 1987, Wollkind et al. 1991). Strong preytaxis tends to homogenize prey spatial distributions because unless the predator diffusion rate is very high, the predator is able to aggregate sufficiently to control the prey population. At the same time, some degree of predator increase in response to prey, such as preytaxis, is necessary for spatial patterning. Particularly in the absence of a sufficient numerical response of predators to prey, preytaxis will promote spatial patterning by providing a mechanism for predator increase in response to prey.

In contrast to predator–prey systems in which diffusion-driven patterns are readily formed, these patterns are not formed in two-species competitive systems, such as in Eq. 12 (Jorne and Carmi 1977, Hastings 1978) unless the two competitors avoid each other (Mimura and Kawasaki 1980). Multispecies competition systems will produce patterns if indirect interactions occur such that the increase of one species indirectly increases the growth of another (Mimura 1984, Mimura and Fife 1986) but, in general, simple multispecies competition systems do not produce patterns (Rosen 1975, Chow and Tamm 1976). Similarly, spatial patterning does not occur when a single species moves via Brownian random motion throughout a homogeneous environment. However, with some types of aggregation behavior, single species can form spatial patterns with a characteristic wavelength, in much the same way as a characteristic wavelength arises via diffusion-driven instabilities (Fig. 4). These patterns are produced by nonlocal aggregation, which assumes that individuals can respond by moving towards conspecifics that are some distance away (Cohen and Murray 1981). In contrast, local aggregation (e.g., the biased

random motion model), in which individuals aggregate by moving up density gradients of neighboring conspecifics (Alt 1985, Okubo 1986, Turchin 1989*a*, *b*), will not produce these patterns. Single-species spatial patterning will also arise if crowding is locally advantageous, but at the same time, organisms are negatively impacted by high densities in surrounding sites (Britton 1989, Furter and Grinfeld 1989). Recently, theoreticians have broadened their examination of dispersal-mediated pattern formation in ecology to include: age-structured single-species dynamics (Hastings 1992), plant–herbivore interactions (Lewis 1993), and temporally and spatially varying environments (Kawasaki and Teramoto 1979, McLaughlin and Roughgarden 1991, Timm and Okubo 1992). The general impression obtained from this theory is that numerous ecological interactions should promote pattern formation; however it will be difficult to show experimentally that such phenomena play a major role in the distributional patterns for plants and animals in natural settings.

Discussion

It should be clear that PDEs can portray a great variety of ecological processes and interactions. Less clear is whether or not PDEs generate useful insights or predictions of relevance to a field ecologist. We think that certain spatial questions are ideally studied in the context of a PDE framework, whereas other spatial questions merit alternative mathematical formulation. Implicit to the formulation of a PDE model is the assumption that the rates of birth, death, and movement can take on a continuous range of values in both space and time; when this continuity assumption is not met, alternative mathematical models are more appropriate. For example, situations in which organisms reproduce continually and move between discrete patches may be better modelled by a system of coupled ordinary differential equations (see Levin 1974, Tilman 1994). Seasonal birth and death can be modelled by difference equations with spatial dispersal incorporated either by using an integral formulation (Kot and Schaffer 1986) or by spatially coupling the difference equations (Hastings 1992). If space, time, and population levels are best represented as discrete variables, then cellular automata models are the ideal tool (Wolfram 1984, Molofsky 1994). Additionally, a key feature in many dispersal-mediated coexistence scenarios is random disturbance, which is a process not conveniently represented in the PDE framework. For this reason, questions pertaining to biodiversity and the coexistence of species are probably best examined with the sorts of models outlined by Tilman (1994) or Molofsky (1994). However, PDEs are useful for examining the interaction between habitat geometry and competitive coexistence. In particular, PDE models emphasize that environments that are fragmented into a variety of habitat patch sizes will also promote coexistence via the differential ability of competitors to survive and compete in small vs. large patches.

On the other hand, for questions relating to invasions or to spatial patterning, PDEs are ideal. For instance, by applying a wide variety of PDE models to the process of invasion, we see that rates of invasion consistently are proportional to rates of population growth at low density and dispersal distances per generation. The robustness of this result provides compelling guidance to a field worker interested in ecological invasions; if he or she wants to predict the dynamics of an invasion, data on low density population growth and the frequency distribution of dispersal distances are essential. Likewise, PDEs are ideal for investigating spatial patterning because they provide a mathematical tool that naturally depicts a continuous, homogeneous space as the "null model." Any patterning that develops is thus clearly due to the interplay of population interactions and dispersal, and not the environment itself. The practical-minded ecologist might wonder why anyone would attempt to explain spatial patterning without environmental heterogeneity when all environments are so obviously heterogeneous. This is the same as asking why anyone would want to explain population fluctuations in terms of species interactions when the environment so obviously fluctuates in time. The point is not to claim that diffusive instabilities explain whatever patterns we see in nature, but to recognize that heterogeneous spatial environments are not necessarily needed to produce striking spatial irregularities in population densities. The other major ecological application of PDEs involves studies regarding population dynamics and sustainability within habitat patches of varying size or shape. Many of the results concerning single-species population dynamics in habitat patches are self-evident without any models. However, PDEs make predictions about population densities and diversity as a function of habitat geometry in the absence of stochastic extinctions (Cantrell and Cosner 1993). It is certainly useful to determine and explain what patterns of species distribution might arise due to habitat geometry without stochastic extinction, even though the colonization/extinction balance of island biogeographic theory has become the dominant explanation for "island size" effects.

Much work needs to be done before PDEs realize their full potential as a tool for illuminating ecological processes. First, more attention should be paid to investigating transient dynamics, how rapidly are asymptotic rates of invasion obtained, and departures from simple idealized portraits of habitat shape or dispersal behavior. Second, careful comparison of the many different mathematical tools available for rep-

resenting spatial processes is needed; for example, it would be very useful to identify similarities in the criteria for pattern formation in PDEs, cellular automata, and integrodifferential equation models. Finally, approaches for coupling the analytical power of PDEs with the realism of spatially explicit individual-based models are worth developing. Specifically, PDEs provide a concise understanding of the connections between individual behavior and population-level spatial effects and thus can guide the analysis and construction of individual-based computer simulations in conservation biology and landscape ecology. In general, PDEs are much more practical than most empiricists realize as a tool for exploring the interplay of dispersal and population dynamics.

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