Spatial Ecology via Reaction-Diffusion Equations

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For our families and friends

Contents

Preface

The "origin of this species" lies in the pages of the journal *Biometrika* and precedes the birth of either of the authors. There, in his remarkable landmark 1951 paper "Random dispersal in theoretical populations," J.G. Skellam made a number of observations that have profoundly affected the study of spatial ecology. First, he made the connection between random walks as a description of movement at the scale of individual members of some theoretical biological species and the diffusion equation as a description of dispersal of the organism at the scale of the species' population density, and demonstrated the plausibility of the connection in the case of small animals using field data for the spread of the muskrat in central Europe. Secondly, he combined the diffusive description of dispersal with population dynamics, effectively introducing reaction-diffusion equations into theoretical ecology, paralleling Fisher's earlier contribution to genetics. Thirdly, Skellam in particular examined reaction-diffusion models for the population density of a species in a bounded habitat, employing both linear (Malthusian) and logistic population growth rate terms, one- and two-dimensional habitat geometries, and various assumptions regarding the interface between the habitat and the landscape surrounding it. His examinations lead him to conclude that "[just] as the area/volume ratio is an important concept in connection with continuance of metabolic processes in small organisms, so is the perimeter/area concept (or some equivalent relationship) important in connection with the survival of a community of mobile individuals. Though little is known from the study of field data concerning the laws which connect the distribution in space of the density of an annual population with its powers of dispersal, rates of growth and the habitat conditions, it is possible to conjecture the nature of this relationship in simple cases. The treatment shows that if an isolated terrestrial habitat is less than a certain critical size the population cannot survive. If the habitat is slightly greater than this the surface which expresses the density at all points is roughly dome-shaped, and for very large habitats this surface has the form of a plateau."

The most general equation for a population density u mentioned in Skellam's paper has the form

$$\frac{\partial u}{\partial t} = d\nabla^2 u + c_1(x, y)u - c_2(x, y)u^2.$$

Writing in 1951, Skellam observed that "orthodox analytic methods appear in adequate" to treat the equation, even in the special case of a one-dimensional habitat. The succeeding half-century since Skellam's paper has seen phenomenal advances in many areas of mathematics, including partial differential equations, functional analysis, dynamical systems, and singular perturbation theory. That which Skellam conjectured regarding reaction-diffusion models (and indeed much more) is now rigorously understood mathematically and has been employed to provide new ecological insight into the interactions of populations and communities of populations in bounded terrestrial (and, for that matter, marine) habitats. Heretofore, the combined story of the mathematical development of reaction-diffusion theory and its application to the study of populations and communities of populations in bounded habitats has not been told in book form, and

telling said story is the purpose of this work. Such is certainly not to suggest in anyway that this is the first book on the mathematical development of reaction-diffusion theory or its applications to ecology, just the first combining a (mostly) self-contained development of the theory with the particular application at hand. There are two other principal uses of reaction-diffusion theory in ecology, namely in the study of ecological invasions (dating from the work of Fisher in the 1930s) and in the study of pattern formation (dating from the work of Turing in the 1950s). It is fair to say that both these other applications have been more widely treated than has the focus of this work. (We discuss this issue further at an appropriate point in Chapter 1 and list some specific references.)

The book is structured as follows. In Chapter 1, we are primarily concerned with introducing our subject matter so as to provide a suitable context–both ecologically and mathematically–for understanding the material that follows. To this end, we begin with an overview of ecological modeling in general followed by an examination of spatial models. So doing enables us then to focus on reaction-diffusion models in more particular terms– how they may be derived, what sorts of ecological questions they may answer, and how we intend to use them to examine species' populations and communities of such populations on isolated bounded habitats. We follow our discussion of reaction-diffusion models as models with a (hopefully) self-contained compilation of the mathematical results that are needed for the analyses of subsequent chapters. For the most part, these results are well-known, so we mainly refer the reader interested in their proofs to appropriate sources. However, our analyses will draw on the theories of partial differential equations, functional and nonlinear analysis, and dynamical systems, and there is quite simply no single source available which contains all the results we draw upon. Consequently, we believe that the inclusion of this material is not merely warranted, but rather essential to the self-containment and readability of the remainder of the book. In Chapter 2, we consider linear reaction-diffusion models for a single species in an isolated bounded habitat and argue that the notion of principal eigenvalue for a linear elliptic operator is the means for measuring average population growth of a species over a bounded habitat which Skellam anticipated in his phrase "perimeter/area concept (or some equivalent relationship)." As with all subsequent chapters, our approach here is a blend of ecological examples, perspective, and applications with model development and analysis. The results in Chapter 2 enable us to turn in Chapter 3 to density dependent reaction-diffusion models for a single species in a bounded habitat. The predictions of such models *viz-a-viz* persistence versus extinction of the species in question may be described rather precisely by employing the notion of a positive (or negative) principal eigenvalue. Frequently, a prediction of persistence corresponds to the existence of a globally attracting positive equilibrium to the model. When we turn to the corresponding models for interacting populations in Chapter 4, the notion of a principal eigenvalue as a measure of average population growth retains its importance. However, the predictive outcomes of such models are not usually so tidily described as in the case of single species models. Frequently, a prediction of persistence cannot be expected to correspond to a componentwise positive globally attracting equilibrium. Instead, one needs to employ the more general notion of a globally attracting set of configurations of positive species' densities. Such configurations include a globally attracting equilibrium as a special case. This notion has come to be called permanence, and Chapter 4 is devoted to the development and application of this concept, followed in Chapter 5 with discussion of notions of persistence beyond permanence. The material in Chapters 4 and 5 is then applied in Chapter 6 to models for two competing species in an isolated bounded habitat and finally in Chapter 7 to nonmonotone models such as models for predation and food chain models.

Series Preface

Theoretical biology is an old subject, tracing back centuries. At times, theoretical developments have represented little more than mathematical exercises, making scant contact with reality. At the other extreme have been those works, such as the writings of Charles Darwin, or the models of Watson and Crick, in which theory and fact are intertwined, mutually nourishing one another in inseparable symbiosis. Indeed, one of the most exciting developments in biology within the last quarter-century has been the integration of mathematical and theoretical reasoning into all branches of biology, from the molecule to the ecosystem. It is such a unified theoretical biology, blending theory and empiricism seamlessly, that has inspired the development of this series.

This series seeks to encourage the advancement of theoretical and quantitative approaches to biology, and to the development of unifying principles of biological organization and function, through the publication of significant monographs, textbooks and synthetic compendia in mathematical and computational biology. The scope of the series is broad, ranging from molecular structure and processes to the dynamics of ecosystems and the biosphere, but it is unified through evolutionary and physical principles, and the interplay of processes across scales of biological organization.

The principal criteria for publication, beyond the intrinsic quality of the work, are substantive biological content and import, and innovative development or application of mathematical or computational methods. Topics will include, but not be limited to, cell and molecular biology, functional morphology and physiology, neurobiology and higher function, immunology and epidemiology, and the ecological and evolutionary dynamics of interacting populations. The most successful contributions, however, will not be so easily categorized, crossing boundaries and providing integrative perspectives that unify diverse approaches; the study of infectious diseases, for example, ranges from the molecule to the ecosystem, involving mechanistic investigations at the level of the cell and the immune system, evolutionary perspectives as viewed through sequence analysis and population genetics, and demographic and epidemiological aspects at the level of the ecological community.

The objective of the series is the integration of mathematical and computational methods into biological work; hence the volumes published should be of interest both to fundamental biologists and to computational and mathematical scientists, as well as to the broad spectrum of interdisciplinary researchers that comprise the continuum connecting these diverse disciplines.

Simon Levin

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Introduction

1.1 Introductory Remarks

A fundamental goal of theoretical ecology is to understand how the interactions of individual organisms with each other and with the environment determine the distribution of populations and the structure of communities. Empirical evidence suggests that the spatial scale and structure of environments can influence population interactions (Gause, 1935; Huffaker, 1958) and the composition of communities (MacArthur and Wilson, 1967). In recent decades the role of spatial effects in maintaining biodiversity has received a great deal of attention in the literature on conservation; see for example Soulè (1986) or Kareiva et al. (1993). One of the most common ways that human activities alter environments is by fragmenting habitats and creating edges. Some habitat fragments may be designated as nature reserves, but they are fragments nonetheless.

One way to try to understand how spatial effects such as habitat fragmentation influence populations and communities is by using mathematical models; see Tilman and Kareiva (1997), Tilman (1994), Molofsky (1994), Holmes et al. (1994), Goldwasser et al. (1994). In this book we will examine how one class of spatial population models, namely reaction-diffusion equations, can be formulated and analyzed. Our focus will be primarily on models for populations or communities which occupy an isolated habitat fragment. There are several other types of spatial population models, including cellular automata, interacting particle systems, metapopulation models, the ideal free distribution, and dispersal models based on integral kernels. Each type of model is based on some set of hypotheses about the scale and structure of the spatial environment and the way that organisms disperse through it. We describe some of these types of models a bit later in our discussion of model formulation; see also Tilman and Kareiva (1997). Some of the ideas used in analyzing reaction-diffusion systems also can be applied to these other types of spatial models. We also describe a few of the connections between different types of models and some unifying principles in their analysis.

Reaction-diffusion models provide a way to translate local assumptions or data about the movement, mortality, and reproduction of individuals into global conclusions about the persistence or extinction of populations and the coexistence of interacting species. They can be derived mechanistically via rescaling from models of individual movement which are based on random walks; see Turchin (1998) or Durrett and Levin (1994). Reaction-diffusion models are spatially explicit and typically incorporate quantities such as dispersal rates, local growth rates, and carrying capacities as parameters which may vary with location or time.

Thus, they provide a good framework for studying questions about the ways that habitat geometry and the size or variation in vital parameters influence population dynamics.

The theoretical advances in nonlinear analysis and the theory of dynamical systems which have occurred in the last thirty years make it possible to give a reasonably complete analysis of many reaction-diffusion models. Those advances include developments in bifurcation theory (Rabinowitz 1971, 1973; Crandall and Rabinowitz 1971, 1973), the formulation of reaction-diffusion models as dynamical systems (Henry 1981), the creation of mathematical theories of persistence or permanence in dynamical systems (Hofbauer and Sigmund 1988, Hutson and Schmitt 1992), and the systematic incorporation of ideas based on monotonicity into the theory of dynamical systems (Hirsch 1982, 1985, 1988a,b, 1989, 1990, 1991; Hess 1991; Smith 1995). One of the goals of this book is to show how modern analytical approaches can be used to gain insight into the behavior of reaction-diffusion models.

There are many contexts in which reaction-diffusion systems arise as models, many phenomena that they support, and many ways to approach their analysis. Existing books on reaction-diffusion models reflect that diversity to some extent but do not exhaust it. There are three major phenomena supported by reaction-diffusion models which are of interest in ecology: the propagation of wavefronts, the formation of patterns in homogeneous space, and the existence of a minimal domain size that will support positive species density profiles. In this book we will focus our attention on topics related to the third of those three phenomena. Specifically, we will discuss in detail the ways in which the size and structure of habitats influence the persistence, coexistence, or extinction of populations. Some other treatments of reaction-diffusion models overlap with ours to some extent, but none combines a specific focus on issues of persistence in ecological models with the viewpoint of modern nonlinear analysis and the theory of dynamical systems. The books by Fife (1979) and Smoller (1982) are standard references for the general theory of reaction-diffusion systems. Both give detailed treatments of wave-propagation, but neither includes recent mathematical developments. Waves and pattern formation are treated systematically by Grindrod (1996) and Murray (1993). Murray (1993) discusses the construction of models in considerable detail, but in the broader context of mathematical biology rather than the specific context of ecology. Okubo (1980) and Turchin (1998) address the issues of formulating reaction-diffusion models in ecology and calibrating them with empirical data, but do not discuss analytic methods based on modern nonlinear analysis. Hess (1991) uses modern methods to treat certain reaction-diffusion models from ecology, but the focus of his book is mainly on the mathematics and he considers only single equations and Lotka-Volterra systems for two interacting species. The book by Hess (1991) is distinguished from other treatments of reaction-diffusion theory by being set completely in the context of time-periodic equations and systems. The books by Henry (1981) and Smith (1995) give treatments of reaction-diffusion models as dynamical systems, but are primarily mathematical in their approach and use specific models from ecology or other applied areas mainly as examples to illustrate the mathematical theory. Smith (1995) and Hess (1991) use ideas from the theory of monotone dynamical systems extensively. An older approach based on monotonicity and related ideas is the method of monotone iteration. That method and other methods based on sub- and supersolutions are discussed by Leung (1989) and Pao (1992) in great detail. However, Leung (1989) and Pao (1992) treat reaction-diffusion models in general without a strong focus on ecology, and they do not discuss ideas and methods that do not involve sub- and supersolutions in much depth. One such idea, the notion of permanence/uniform persistence, is discussed by Hofbauer and Sigmund (1988, 1998) and in the survey paper by Hutson and Schmitt (1992). We will use that idea fairly extensively but our treatment differs from those

given by Hofbauer and Sigmund (1988, 1998) and Hutson and Schmitt (1992) because we examine the specific applications of permanence/uniform persistence to reaction-diffusion systems in more depth, and we also use other analytic methods. Finally, there are some books on spatial ecology which include discussions of reaction-diffusion models as well as other approaches. Those include the volumes on spatial ecology by Tilman and Kareiva (1997) and on biological invasions by Kawasaki and Shigesada (1997). However, those books do not go very far with the mathematical analysis of reaction-diffusion models on bounded spatial domains.

We hope that the present volume will be interesting and useful to readers whose backgrounds range from theoretical ecology to pure mathematics, but different readers may want to read it in different ways. We have tried to structure the book to make that possible. Specifically, we have tried to begin each chapter with a relatively nontechnical discussion of the ecological issues and mathematical ideas, and we have deferred the most complicated mathematical analyses to Appendices which are attached to the ends of chapters. Most chapters include a mixture of mathematical theorems and ecological examples and applications. Readers interested primarily in mathematical analysis may want to skip the examples, and the readers interested primarily in ecology may want to skip the proofs. We hope that at least some readers will be sufficiently interested in both the mathematics and the ecology to read both.

To read this book effectively a reader should have some background in both mathematics and ecology. The minimal background needed to make sense of the book is a knowledge of ordinary and partial differential equations at the undergraduate level and some experience with mathematical models in ecology. A standard introductory course in ordinary differential equations, a course in partial differential equations from a book such as Strauss (1992), and some familiarity with the ecological models discussed by Yodzis (1989) or a similar text on theoretical ecology would suffice. Alternatively, most of the essential prerequisites with the exception of a few points about partial differential equations can be gleaned from the discussions in Murray (1993). Readers with the sort of background described above should be able to understand the statements of theorems and to follow the discussion of the ecological examples and applications.

To follow the derivation of the mathematical results or to understand why the examples and applications are of interest in ecology requires some additional background. To be able to follow the mathematical analysis, a reader should have some knowledge of the theory of functions of a real variable, for example as discussed by Royden (1968) or Rudin (1966, 1976), and some familiarity with the modern theory of elliptic and parabolic partial differential equations, as discussed by Gilbarg and Trudinger (1977) and Friedman (1976), and dynamical systems as discussed by Hale and Koçak (1991). To understand the ecological issues behind the models, a reader should have some familiarity with the ideas discussed by Tilman and Kareiva (1997), Soulè (1986), Soulè and Terborgh (1989), and/or Kareiva et al. (1993). The survey articles by Tilman (1994), Holmes et al. (1994), Molofsky (1994), and Goldwasser et al. (1994) are also useful in that regard. For somewhat broader treatments of ecology and mathematical biology respectively, Roughgarden et al. (1989) and Levin (1994) are good sources.

1.2 Nonspatial Models for a Single Species

The first serious attempt to model population dynamics is often credited to Malthus (1798). Malthus hypothesized that human populations can be expected to increase geometrically

with time but the amount of arable land available to support them can only be expected to increase at most arithmetically, and drew grim conclusions from that hypothesis. In modern terminology the Malthusian model for population growth would be called a density independent model or a linear growth model. In nonspatial models we can describe populations in terms of either the total population or the population density since the total population will just be the density times the area of the region the population inhabits. We will typically think of the models as describing population densities since that viewpoint still makes sense in the context of spatial models. Let $P(t)$ denote the density of some population at time t. A density independent population model for $P(t)$ in continuous time would have the form

$$\frac{dP}{dt} = r(t)P(t); \tag{1.1}$$

in discrete time the form would be

$$P(t+1) = R(t)P(t). \tag{1.2}$$

These sorts of models are linear in the terminology of differential or difference equations, which is why they are also called linear growth models. In the discrete time case we must have $R(t) \geq 0$ for the model to make sense. If r is constant in (1.1) we have $P(t) = e^{rt}P(0)$; if $R(t)$ is constant in (1.2) we have $P(t) = R^t P(0)$. In either case, the models predict exponential growth or decay for the population. To translate between the models in such a way that the predicted population growth rate remains the same we would use $R = e^r$ or $r = \ln R$.

The second major contribution to population modeling was the introduction of population self-regulation in the logistic equation of Verhulst (1838). The key element introduced by Verhulst was the notion of density dependence, that is, the idea that the density of a population should affect its growth rate. Specifically, the logistic equation arises from the assumption that as population density increases the effects of crowding and resource depletion cause the birth rate to decrease and the death rate to increase. To derive the logistic model we hypothesize that the birthrate for our population is given by $b(t) - a(t, P)$ and the death rate by $d(t) + e(t, P)$ where b, a, d, and e are nonnegative and a and e are increasing in P. The simplest forms for a and e are $a = a_0(t)P$ and $e = e_0(t)P$ with $a_0, e_0 \geq 0$. The net rate of growth for a population at density P is then given by

$$\begin{aligned}
\frac{dP}{dt} &= ([b(t) - a_0(t)P] - [d(t) + e_0(t)P])P \\
&= ([b(t) - d(t)] - [a_0(t) + e_0(t)]P)P \\
&= (r(t) - c(t)P)P,
\end{aligned} \tag{1.3}$$

where $r(t) = b(t) - d(t)$ may change sign but $c(t) = a_0(t) + e_0(t)$ is always nonnegative. We will almost always assume $c(t) \geq c_0 > 0$. If r and c are constant we can introduce the new variable $K = r/c$ and write (1.3) as

$$\frac{dP}{dt} = r\left(1 - \left[\frac{P}{K}\right]\right)P. \tag{1.4}$$

Equation (1.4) is the standard form used in the biology literature for the logistic equation. The parameter r is often called the intrinsic population growth rate, while K is called

the carrying capacity. If $r(t) > 0$ then equation (1.3) can be written in the form (1.4) with K positive. However, if K is a positive constant then letting $r = r(t)$ in (1.4) with $r(t)$ negative some of the time leads to a version of (1.3) with $c(t) < 0$ sometimes, which contradicts the underlying assumptions of the model. We will use the form (1.4) for the logistic equation in cases where the coefficients are constant, but since we will often want to consider situations where the intrinsic population growth rate r changes sign (perhaps with respect to time, or in spatial models with respect to location) we will usually use the form (1.3). Note that by letting $p = P/K$ and $\tau = rt$ we can rescale (1.4) to the form $dp/d\tau = p(1 - p)$. We sometimes assume that (1.4) has been rescaled in this way. A derivation along the lines shown above is given by Enright (1976). The specific forms $a = a_0(t)P$, $e = e_0(t)P$ are certainly not the only possibilities. In fact, the assumptions that increases in population density lead to decreases in the birth rate and increases in the death rate may not always be valid. Allee (1931) observed that many animals engage in social behavior such as cooperative hunting or group defense which can cause their birth rate to increase or their death rate to decrease with population density, at least at some densities. Also, the rate of predation may decrease with prey density in some cases, as discussed by Ludwig et al. (1978). In the presence of such effects, which are typically known as Allee effects, the model (1.3) will take a more general form

$$\frac{dP}{dt} = g(t, P)P \tag{1.5}$$

where g may be increasing for some values of P and decreasing for others. A simple case of a model with an Allee effect is

$$\frac{dP}{dt} = r(P - \alpha)(K - P)P \tag{1.6}$$

where $r > 0$ and $0 < \alpha < K$. The model (1.6) implies that P will decrease if $0 < P < \alpha$ or $P > K$ but increase if $\alpha < P < K$.

The behavior of (1.4) is quite simple. Positive solutions approach the equilibrium K monotonically as $t \to \infty$ at a rate that depends on r, so that the equilibrium $P = 0$ is unstable and $P = K$ is stable. The behavior of (1.6) is slightly more complicated. Solutions which start with $0 < P < \alpha$ will approach 0 as $t \to \infty$; solutions starting with $P > \alpha$ will approach K monotonically as $t \to \infty$. Thus, the equilibrium $P = \alpha$ is unstable but $P = 0$ and $P = K$ are stable.

There are various ways that a logistic equation can be formulated in discrete time. The solution to (1.4) can be written as $P(t) = e^{rt}P/(1 + [(e^{rt} - 1)/K]P)$. If we evaluate $P(t)$ at time $t = 1$ we get $P(1) = e^r P(0)/(1 + [(e^r - 1)/K]P(0))$; by iterating we obtain the discrete time model

$$P(t + 1) = e^r P(t)/(1 + [(e^r - 1)/K]P(t)). \tag{1.7}$$

The model (1.7) is a version of the Beverton-Holt model for populations in discrete time (see Murray (1993), Cosner (1996)). A different formulation can be obtained by integrating the equation $dP/dt = r[1 - (P(0)/K)]P(t)$; that yields $P(1) = \exp(r[1 - (P(0)/K)])P(0)$ and induces an iteration

$$P(t + 1) = \exp(r[1 - (P(t)/K)])P(t). \tag{1.8}$$

This is a version of the Ricker model (see Murray (1993), Cosner (1996)). The difference in the assumptions behind (1.7) and (1.8) is that in (1.7) intraspecific competition is assumed to occur throughout the time interval (t, t_1) while in (1.8) the competitive effect is only based on conditions at time t. The behaviors of the models (1.7) and (1.8) are quite different. Model (1.7) behaves much like the logistic model (1.4) in continuous time. Solutions that are initially positive converge to the equilibrium $P = K$ monotonically (see Cosner (1996)). On the other hand, (1.8) may have various types of dynamics, including chaos, depending on the parameters (see Murray (1993)). In most of what follows we will study continuous time models which combine local population dynamics with dispersal through space, and we will describe dispersal via diffusion. Some of the ideas and results we will discuss can be extended to models in discrete time, but the examples (1.7), (1.8) show that models in discrete time may or may not behave in ways that are similar to their continuous time analogues, so some care is required in going from continuous to discrete time.

In many populations individuals are subject to different levels of mortality and have different rates of reproduction at different ages or stages in their lives. Models which account for these effects typically classify the population by developmental stage, age, or size and specify the rates at which individuals move from one stage to another, what fraction survive each stage of their life history, and the rates at which individuals at each of the stages produce offspring. The type of models which have been used most frequently to describe age or stage structured populations are discrete time matrix models of the sort introduced by Leslie (1948) and treated in detail by Caswell (1989). These models divide a population into n classes, with the population in each class denoted by P_i. Usually the class P_0 represents eggs, seeds, or recently born juveniles. The total population is then given by $\sum_{i=0}^{n} P_i$. The models typically specify the fraction S_i of individuals in class i that survive and enter class $i+1$ at each time step, the fraction S_{n+1} that survive and remain in class n, and the number of offspring R_i of class $i = 0$ produced in each time step by an individual of class i. The models then take the form

$$\vec{P}(t) = M\vec{P}(t) \tag{1.9}$$

where $\vec{P} = (P_0, \ldots, P_n)$ and M is the matrix

$$M = \begin{pmatrix} R_0 & R_1 & R_2 & \ldots & R_n \\ S_1 & 0 & 0 & \ldots & 0 \\ 0 & S_2 & \ddots & \ddots & \ldots \\ \vdots & \ddots & \ddots & 0 & 0 \\ 0 & \ldots & 0 & S_n & S_{n+1} \end{pmatrix}.$$

Models of the form (1.9) are discussed at length by Caswell (1989). In general the entries in the matrix M may depend on $\vec{P}$ in various ways. A key property of matrices of the form shown for M with constant positive entries is that M^n has all its entries positive. It follows from the theory of nonnegative matrices that M has a positive eigenvalue λ_1 whose corresponding eigenvector $\vec{v}$ is componentwise positive. (This is a consequence of the Perron-Frobenius theorem. See Caswell (1989), Berman and Plemmons (1979), or the discussion of positivity in Chapter 2.) The eigenvalue λ_1 is called the principal eigenvalue

of M, and it turns out that λ_1 is larger than the real part of any other eigenvalue of M. If $\lambda_1 > 1$ then the population will increase roughly exponentially; specifically, if $\vec{v}$ is the componentwise positive eigenvector of unit length corresponding to λ_1 we will have $P(t) \approx \lambda_1^t (\vec{P}(0) \cdot \vec{v}) \vec{v}$ for t large. (See Caswell (1989).) Similarly, if $\lambda_1 < 1$ then the population will decline roughly exponentially. Thus, λ_1 plays the same role as R plays in (1.2). If we viewed λ_1 as giving an overall growth rate for the entire population $\sum_{i=0}^{n} P_i$, which is reasonable in view of the asymptotic behavior of (1.9), we would use $r = \ln \lambda_1$ in the corresponding continuous model. In this case $r > 0$ if and only if $\lambda_1 > 1$. Because they break down the life history of an organism into simpler steps, models of the form (1.9) are useful in deriving population growth rates from empirical data on survivorship and fecundity; again, see Caswell (1989). The principal eigenvalue of M in effect averages population growth rates over the age or stage classes of a structured population. The use of eigenvalues to obtain something like an average growth rate for a structured population will be a recurring theme in this book. However, the populations we consider will usually be structured by spatial distribution rather than age, and the eigenvalues will generally correspond to differential operators rather than matrices. If the entries in the matrix M depend on $\vec{P}$ then the model (1.9) can display the same types of behavior as (1.7) and (1.8). See Caswell (1989) or Cosner (1996) for additional discussion of density dependent models of the form (1.9).

It is also possible to formulate age structured population models in continuous time. The simplest formulation of such models describes a population in terms of $P(a, t)$ where a is a continuous variable representing age, so that the number of individuals in the population at time t whose ages are between a_1 and a_2 is given by $\int_{a_1}^{a_2} P(a, t) da$. The basic form of a linear (or density independent) model for a population with a continuous age structure consists of an equation describing how individuals age and experience mortality, and another equation describing the rate at which new individuals are born. The equation describing how individuals age is the McKendrick-Von Foerster equation

$$\frac{\partial P}{\partial t} + \frac{\partial P}{\partial a} = -d(a) P \qquad (1.10)$$

where $d(a)$ is a age-specific death rate. The equation describing births is the birth law

$$P(0, t) = \int_0^\infty b(a) P(a, t) da \qquad (1.11)$$

where $b(a)$ is an age dependent birth rate. Density dependent models arise if d or b depends on P. Age structured models based on generalizations of (1.10) and (1.11) are discussed in detail by Webb (1985).

Our main goal is to understand spatial effects, so we will usually assume that the population dynamics of a given species at a given place and time are governed by a simple continuous time model of the form (1.5). We will often consider situations where the population dynamics vary with location, and we will typically model dispersal via diffusion. Before we discuss spatial models, however, we describe some models for interacting populations which are formulated in continuous time via systems of equations analogous to (1.5).

The population models described above are all deterministic, and all of them can be interpreted as giving descriptions of how populations behave as time goes toward infinity.

It is also possible to construct models based on the assumption that changes in population are stochastic. Typically such models predict that populations will become extinct in finite time, and often the main issue in the analysis of such models is in determining the expected time to extinction. We shall not pursue that modeling approach further. A reference is Mangel and Tier (1993).

1.3 Nonspatial Models For Interacting Species

1.3.1 Mass-Action and Lotka-Volterra Models

The first models for interacting species were introduced in the work of Lotka (1925) and Volterra (1931). Those models have the general form

$$\frac{dP_i}{dt} = \left(a_i + \sum_{j=1}^{n} b_{ij} P_j \right) P_i, \quad i = 1, \dots, n, \tag{1.12}$$

where P_i denotes the population density of the ith species. The coefficients a_i are analogous to the linear growth rate $r(t)$ in the logistic model (1.3). The coefficients b_{ii} represent intraspecies density dependence, in analogy with the term $c(t)P$ in (1.3), so we have $b_{ii} \leq 0$ for all i. The coefficients b_{ij}, $i \neq j$, describe interactions between different species. The nature of the interaction–competition, mutualism, or predator-prey interaction–determines the signs of the coefficients b_{ij}. If species i and j compete then b_{ij}, $b_{ji} < 0$. If species i preys upon species j, then $b_{ij} > 0$ but $b_{ji} < 0$. If species i and j are mutualists, then b_{ij}, $b_{ji} > 0$. (In the case of mutualism Lotka-Volterra models may sometimes predict that populations will become infinite in finite time, so the models are probably less suitable for that situation than for competition or predator-prey interactions.) Usually Lotka-Volterra competition models embody the assumption that $b_{ii} < 0$ for each i, so the density of each species satisfies a logistic equation in the absence of competitors. In the case where species i preys on species j, it is often assumed that $b_{jj} < 0$ (so the prey species satisfies a logistic equation in the absence of predation), but that $b_{ii} = 0$ while $a_i < 0$. Under those assumptions the predator population will decline exponentially in the absence of prey (because $a_i < 0$), but the only mechanism regulating the predator population is the availability of prey (because $b_{ii} = 0$, implying that the growth rate of the predator population does not depend on predator density). If the predator species is territorial or is limited by the availability of resources other than prey, it may be appropriate to take $b_{ii} < 0$. Lotka-Volterra models are treated in some detail by Freedman (1980), Yodzis (1989), and Murray (1993).

The interaction terms in Lotka-Volterra models have the form $b_{ij} P_i P_j$. If species i and species j are competitors then the equations relating P_i and P_j in the absence of other species are

$$\begin{aligned}
\frac{dP_i}{dt} &= (a_i - b_{ij} P_j - b_{ii} P_i) P_i \\
\frac{dP_j}{dt} &= (a_j - b_{ji} P_i - b_{jj} P_j) P_j.
\end{aligned} \tag{1.13}$$

In the context of competition, the interaction terms appear in the same way as the self-regulation terms in the logistic equation. Thus, if b_{ii} is interpreted as measuring the extent to which members of species i deplete resources needed by that species and thus reduce the

net population growth rate for species i, then b_{ij} can be interpreted as measuring the extent to which members of species j deplete the same resources. This interpretation can be used to study the amount of similarity in resource utilization which is compatible with coexistence; see MacArthur (1972) or Yodzis (1989). The interpretation in the context of predator-prey interaction is more complicated. The interaction rate $b_{ij} P_i P_j$ can be interpreted as a mass-action law, analogous to mass-action principles in chemistry. The essential idea is that if individual predators and prey are homogeneously distributed within some region, then the rate at which an individual predator searching randomly for prey will encounter prey individuals should be proportional to the density of prey, but predators will search individually, so that the number of encounters will be proportional to the prey density times the predator density. Another assumption of the Lotka-Volterra model is that the birth rate of predators is proportional to the rate at which they consume prey, which in turn is directly proportional to prey density. Both of these assumptions are probably oversimplifications in some cases.

1.3.2 Beyond Mass-Action: The Functional Response

A problem with the mass-action formulation is that it implies the rate of prey consumption by each predator will become arbitrarily large if the prey density is sufficiently high. In practice the rate at which a predator can consume prey is limited by factors such as the time required to handle each prey item. This observation leads to the notion of a functional response, as discussed by Holling (1959). Another problem is that predators and prey may not be uniformly distributed. If predators search in a group then the rates at which different individual predators encounter prey will not be independent of each other. Finally, predators may spend time interacting with each other while searching for prey or may interfere with each other, so that the rate at which predators encounter prey is affected by predator density. These effects can also be incorporated into predator-prey models via the functional response.

We shall not give an extensive treatment of the derivation of functional response terms, but we shall sketch how functional responses can be derived from considerations of how individuals utilize time and space. We begin with a derivation based on time utilization, following the ideas of Holling (1959) and Beddington et al. (1975). Suppose a predator can spend a small period of time ΔT searching for prey, or consuming captured prey, or interacting with other predators. (The period of time ΔT should be short in the sense that the predator and prey densities remain roughly constant over ΔT.) Let P_1 denote the predator density and P_2 the prey density. Let ΔT_s denote the part of ΔT that the predator spends searching for prey. Let ΔT_1 denote the part of ΔT the predator spends interacting with other predators and let ΔT_2 denote the part of ΔT the predator spends handling prey. We have $\Delta T = \Delta T_s + \Delta T_1 + \Delta T_2$, but ΔT_1 and ΔT_2 depend on the rates at which the predator encounters other predators and prey, and on how long it takes for each interaction. Suppose that during the time it spends searching each individual predator encounters prey and other predators at rates proportional to the prey and predator densities, respectively (i.e. according to mass action laws.) The number of prey encountered in the time interval ΔT will then be given by $e_2 P_2 \Delta T_s$, while the number of predators encountered will be $e_1 P_1 \Delta T_s$, where e_1 and e_2 are rate constants that would depend on factors such as the predator's movement rate while searching or its ability to detect prey or other predators. If h_1 is the length of time required for each interaction between predators and h_2 is the length of time required for each interaction between a predator and a prey item, then $\Delta T_1 = e_1 h_1 P_1 \Delta T_s$ and $\Delta T_2 = e_2 h_2 P_2 \Delta T_s$. Using the relation $\Delta T = \Delta T_s + \Delta T_1 + \Delta T_2$,

we have $\Delta T = (1 + e_1 h_1 P_1 + e_2 h_2 P_2) \Delta T_s$. Also, the predator encounters $e_2 P_2 \Delta T_s$ prey items during the period ΔT, so the overall rate of encounters with prey over the time interval ΔT is given by

$$
\begin{aligned}
e_2 P_2 \Delta T_s / \Delta T &= e_2 P_2 \Delta T_s / (1 + e_1 h_1 P_1 + e_2 h_2 P_2) \Delta T_s \\
&= e_2 P_2 / (1 + e_1 h_1 P_1 + e_2 h_2 P_2).
\end{aligned}
\tag{1.14}
$$

The expression $g(P_1, P_2) = e_2 P_2 / (1 + e_1 h_1 P_1 + e_2 h_2 P_2)$ is a type of functional response, introduced by Beddington et al. (1975) and DeAngelis et al. (1975). Under the assumption that predators do not interact with each other, so that $h_1 = 0$, it reduces to a form derived by Holling (1959), known as the Holling type 2 functional response. If we maintain the assumption that the rate at which new predators are produced is proportional to the per capita rate of prey consumed by each predator, and assume the prey population grows logistically in the absence of predators, the resulting model for the predator-prey interaction is

$$
\begin{aligned}
\frac{dP_1}{dt} &= \frac{a e_1 P_2 P_1}{1 + e_1 h_1 P_1 + e_2 h_2 P_2} - d P_1 \\
\frac{dP_2}{dt} &= r \left(1 - \left[\frac{P_2}{K} \right] \right) P_2 - \frac{e_2 P_1 P_2}{1 + e_1 h_1 P_1 + e_2 h_2 P_2}.
\end{aligned}
\tag{1.15}
$$

(The coefficient a represents the predator's efficiency at converting consumed prey into new predators, while d represents the predator death rate in the absence of prey.) Note that if the prey density is held fixed at the level P_2^* the predator equation takes the form

$$
\frac{dP_1}{dt} = \frac{A P_1}{B + C P_1} - d P_1 = \left[\frac{A}{B + C P_1} - d \right] P_1,
\tag{1.16}
$$

where $A = a e_2 P_2^*$, $B = 1 + e_2 h_2 P_2^*$, and $C = e_1 h_1$. If $A/B > d$ and $C > 0$ the function $[A/(B+C P_1)] - d$ is positive when $P_1 > 0$ is small but negative when P_1 is large. Thus, the model (1.16) behaves like the logistic equation in the sense that it includes self-regulation.

The derivation of the Beddington-DeAngelis (and Holling type 2) functional response in the preceding paragraph from considerations of time utilization retained the assumption that the total rate of encounters between searching predators and items of prey should follow a mass-action law. Other types of encounter rates can arise if predators or prey are not homogeneously distributed. This point is discussed in some detail by Cosner et al. (1999). Here we will just analyze one example of how spatial effects can influence the functional response and then describe the results of other scenarios. Let E denote the total rate of encounters between predators and prey per unit of search time. The rate at which prey are encountered by an individual predator will then be proportional to E/P_1 where P_1 is the predator density. The *per capita* encounter rate E/P_1 reduces to $e_2 P_2$ if $E = e_2 P_1 P_2$, as in the case of mass action. Substituting the form $E/P_1 = e_2 P_2$ into the derivation given in the preceding paragraph yields the Holling type 2 functional response if we assume that predators do not interact with each other. However, the mass action hypothesis $E = e_2 P_1 P_2$ is based on the assumption that predators and prey are homogeneously distributed in space. Suppose instead that the predators do not search for prey independently but form a group in a single location and then search as a group. In that case, increasing the number of predators in the system will not increase the area searched per unit time and thus the number of encounters with prey will not depend on predator density. (This assumes that

adding more predators to the group does not significantly increase the distance at which predators can sense prey or otherwise increase the searching efficiency of the predators.) In that case we would still expect the rate of encounters to depend on prey density, so that $E = e^* P_2$. Since E represents the total encounter rate between all predators and all prey, the per capita rate at which each individual predator encounters prey will be given by $e_2^* P_2 / P_1$. (We are assuming that predators and prey inhabit a finite spatial region so that the numbers of predators and prey are proportional to their densities.) Since we are assuming that all the predators are in a single group, they will not encounter any other predators while searching for prey. Using the *per capita* encounter rate with prey $e_2^* P_2 / P_1$ instead of $e_2 P_2$ in the derivation of (1.14) leads to

$$(e_2^* P_2 / P_1) / (1 + e_2^* h_2 (P_2 / P_1)) = e^* P_2 / (P_1 + e_2^* h P_2). \tag{1.17}$$

The corresponding predator-prey model is

$$\frac{dP_1}{dt} = \frac{ae_2^*(P_2/P_1)P_1}{1 + e_2^* h_2(P_2/P_1)} - dP_1 = \left[\frac{ae_2^* P_2}{P_1 + e_2^* h_2 P_2} - d \right] P_1$$

$$\frac{dP_2}{dt} = r\left(1 - \left[\frac{P_2}{K}\right]\right) P_2 - \frac{e_2^* P_1 P_2}{P_1 + e_2^* h_2 P_2}. \tag{1.18}$$

The model (1.18) is said to be ratio-dependent, because the functional response depends on the ratio P_2/P_1. Other types of functional responses arise from other assumptions about the spatial grouping of predators. These include the Hassell-Varley form $e P_2 / (P_1^\gamma + e h P_2)$ where $\gamma \in (0, 1)$, among others; see Cosner et al. (1999). In the ratio-dependent model (1.18) the functional response is not smooth at the origin. For that reason the model can display dynamics which do not occur in predator-prey models of the form

$$\frac{dP_1}{dt} = \left[ag(P_1, P_2) - d\right] P_1$$

$$\frac{dP_2}{dt} = r\left(1 - \left[\frac{P_2}{K}\right]\right) P_2 - g(P_1, P_2) P_1 \tag{1.19}$$

with $g(P_1, P_2)$ smooth and $g(P_1, 0) = 0$. In particular, the ratio-dependent model (1.18) may predict that both predators and prey will become extinct for certain initial densities; see Kuang and Beretta (1998).

There are several other forms of functional response which occur fairly often in predator-prey models. Some of those arise from assumptions about the behavior or perceptions of predators. An example, and the last type of functional response we will discuss in detail, is the Holling type 3 functional response $g(P_2) = e P_2^2 / (1 + f P_2^2)$. The key assumption leading to this form of functional response is that when the prey density becomes low the efficiency of predators in searching for prey is reduced. This could occur in vertebrate predators that have a "search image" which is reinforced by frequent contact with prey, or that use learned skills in searching or in handling prey which deteriorate with lack of practice; i.e. when prey become scarce. It will turn out that the fact that the Holling type 3 functional response tends toward zero quadratically rather than linearly as $P_2 \to 0$ can sometimes be a significant factor in determining the effects of predator-prey interactions.

There are many other forms of functional response terms that have been used in predator-prey models. Some discussion and references are given in Getz (1994) and Cosner et al. (1999). The various specific forms discussed here (Holling type 2 and type 3, Beddington-DeAngelis, Hassell-Varley, etc.) are sometimes classified as prey dependent ($g = g(P_2)$ in

our notation), ratio-dependent ($g = g(P_2/P_1)$) and predator dependent ($g = g(P_1, P_2)$). There has been some controversy about the use of ratio-dependent forms of the functional response; see Abrams and Ginzburg (2000) for discussion and references. In a recent study of various data sets, Skalski and Gilliam (2001) found evidence for some type of predator dependence in many cases. In what follows we will often use Lotka-Volterra models for predator-prey interactions, but we will sometimes use models with Holling type 2 or 3 functional response or with Beddington-DeAngelis functional response, depending on the context. Our main focus will generally be on understanding spatial effects, rather than exhaustively exploring the detailed dynamics corresponding to each type of functional response, and the forms listed above represent most of the relevant qualitative features that occur in standard forms for the functional response. We will not consider the ratio dependent case. That case is interesting and worthy of study, but it presents some extra technical problems, and it turns out that at least some of the scaling arguments which lead to diffusion models can destroy ratio dependence.

1.4   Spatial Models: A General Overview

The simple models we have described so far assume that all individuals experience the same homogeneous environment. In reality, individual organisms are distributed in space and typically interact with the physical environment and other organisms in their spatial neighborhood. The most extreme version of local interaction occurs among plants or sessile animals that are fixed in one location. Even highly mobile organisms encounter only those parts of the environment through which they move. Many physical aspects of the environment such as climate, chemical composition, or physical structure can vary from place to place. In a homogeneous environment any finite number of individuals will necessarily occupy some places and not others. The underlying theoretical distribution of individuals may be uniform, but each realization of a uniform distribution for a finite population will involve some specific and nonuniform placement of individuals. These observations would not be of any great interest in ecology if there were no empirical reasons to believe that spatial effects influence population dynamics or if simple models which assume that each individual interacts with the average environment and the average densities of other organisms adequately accounted for the observed behavior of populations and structure of communities. However, there is considerable evidence that space can affect the dynamics of populations and the structure of communities. An early hint about the importance came in the work of Gause (1935). Gause conducted laboratory experiments with *paramecium* and *didnium* and found that they generally led to extinction of one or both populations, even though the same species appear to coexist in nature. In a later set of experiments Huffaker (1958) found that a predator-prey system consisting of two species of mites could collapse to extinction quickly in small homogeneous environments, but would persist longer in environments that were subdivided by barriers to dispersal. Another type of empirical evidence for the significance of spatial effects comes from observations of natural systems on islands and other sorts of isolated patches of favorable habitat in a hostile landscape. There are many data sets which show larger numbers of species on larger islands and smaller numbers of species on smaller islands. These form the basis for the theory of island biogeography introduced by MacArthur and Wilson (1967); see also Williamson (1981) or Cantrell and Cosner (1994). A different sort of empirical evidence for the importance of space is that simple nonspatial models for resource competition indicate that in competition for a single limiting resource the strongest competitor should exclude