Part III

Spatiotemporal dynamics and pattern formation: deterministic approach
Chapter 8

Spatial aspect: diffusion as a paradigm

The analysis of the previous few chapters has largely ignored the spatial aspects of the population dynamics. The underlying assumptions are twofold. First, the results of the nonspatial analysis apply to the case of spatially homogeneous, “well-mixed” populations, which usually implies that the corresponding habitat is sufficiently small. Alternatively, the impact of spatial dimension(s) can possibly be ignored in a somewhat more exotic case when the individuals of a given species remain fixed in space at any time and in any generation.

Although these assumptions are not totally unrealistic, obviously, they do not always hold in the real world. Populations of ecological species do not remain fixed in space; their distribution changes continuously due to the impact of environmental factors, such as wind in case of airborne species, and/or due to self-motion of individuals. The properties of the motion can be significantly different depending on the particular ecological situation, i.e., on the behavioral traits characteristic for a given species and also on the temporal and spatial scale of the phenomenon under study. In this book we are not concerned with seasonal migrations, which are typical for many fish and bird species. Although migrations of that type may take place on a scale of thousands of kilometers, it often happens that a given flock of birds every spring returns to exactly the same pond or field, thus leaving the properties of the species spatial distribution essentially unchanged. Instead, we are interested in the dynamics that goes on a much smaller spatial scale but yet may eventually change the area of species dwelling as well as the features of the population spatial distribution inside.

A widely accepted and the most theoretically developed paradigm of the small-scale individual motion is the process called random walks. Having first appeared in physics, it was further developed and adapted for biological applications by Skellam (1951) and Okubo (1980).¹ One of its essential features is that the corresponding motion is isotropic in space (i.e., at each step the direction of motion is chosen randomly), and that has been a point of controversy and criticism. Indeed, it seems difficult to imagine that a mammal or

a bird or even an insect moves in a completely random manner, unless it has suffered from some sort of brain damage. However, this problem is easily resolved by choosing the adequate spatial scale of the random walk. The clue is that the random walk is just an abstract technique, which means that we are not at all obliged to associate each step in this highly theoretical procedure with the real “steps” or motion of a living being. Consider, for instance, the motion of a flying insect. Each of its short flights is apparently highly motivated and is by no means random, whether it is concerned with searching for food or avoiding predators or looking for a mating partner or something else. However, the “decision” for each new flight or “walk” is inevitably affected by a great variety of factors. Therefore, the direction of the next flight may be only slightly correlated with the direction of the previous one. Moreover, this loss of information is likely to accumulate with each new flight, so that after a certain number of steps the correlation will be lost completely (assuming, of course, that the external environmental conditions do not bring any preferred direction of motion). Now, what we should do is to coarse the scale of consideration combining several nonrandom flights together in order to produce one “step” for the random walk procedure. These heuristic speculations appear to be in a very good agreement with some data of field observations (cf. Root and P.M., 1984) showing that the motion of insects can indeed be very closely approximated as random on an appropriate spatial scale. Obviously, this way “to randomize” the motion is not restricted to insects and should be applicable to virtually any other species. Moreover, it has been shown recently that even the motion of humans can be very well approximated with random walks if considered on a relevant spatiotemporal scale (Brockmann et al., 2006).

The accepted assumption of isotropy of the individual motion does not fully define the random walk motion yet. Another very important property of the process is how the mean square displacement $<x^2>$ depends on time. A rather general case is given by the power law, $<x^2> \sim t^\sigma$, and then the three different cases are (i) Brownian motion with $\sigma = 1/2$, (ii) “subdiffusion” with $\sigma < 1/2$, and (iii) “superdiffusion” with $\sigma > 1/2$ (e.g., see Klafter et al., 1996). Although both case (ii) and especially case (iii) have been attracting considerable attention recently, cf. “Lévy flights,” in this book we assume that the individual random walks are of standard Brownian type, which eventually results in the diffusion equation.

There are a few somewhat different theoretical routes to arrive at the “macroscopic” description of the population dynamics (e.g., by the diffusion equation) from the “microscopic” level of individual motion. A detailed description can be found elsewhere; for instance, an interested reader is advised to look into the classical works by Okubo (1980, 1986). Here we employ another way to derive the diffusion-type equations of population dynamics based on the “control volume” approach and making use of the available relation between the population flux and the corresponding population density.

Let us consider a population community consisting of $n$ species. At any moment of time $t$, the state of this community is described by the population
densities \( U_1, U_2, \ldots, U_n \). Let us consider a small “control volume” \( V \) with the boundary given by a surface \( S \); correspondingly, the population size of the \( i \)th species inside the volume \( V \) is given as

\[
N_i = \int_V U_i(\mathbf{R}, T) d\mathbf{R} ,
\]  

(8.1)

where \( d\mathbf{R} \) is an infinitesimally small element of volume.

\( N_i \) may change for two reasons, i.e., either due to the events of birth and death and/or due to the population fluxes through the boundary \( S \). Let us begin with the latter. A cornerstone of the approach is then the equation for the fluxes. The simplest case is given by the Fick law when the flux of a given quantity is proportional to its gradient. In a general case, however, the flux of species \( i \) may depend not only on its distribution but also on the spatial distribution of other species due to the phenomenon called crossdiffusion:

\[
J_i = -\sum_{j=1}^{n} D_{ij} \nabla U_j , \quad i = 1, \ldots, n ,
\]  

(8.2)

where \( D_{ij} \) are nonnegative (cross)diffusion coefficients.\(^2\)

The rate of increase/decrease in the size of population \( i \) per unit time is then given by integration of (8.2) over the surface enclosing the control volume \( V \), i.e.,

\[
\int_S J_i ds ,
\]  

(8.3)

where \( ds \) is an oriented element of surface \( S \) directed outwards.

Equation (8.3) gives the rate of population change due to the motion of the individuals. In any real population, however, its individuals not only move but also, if considered on a time scale large enough, reproduce and die. Let the rate of the local population growth of species \( i \) be described by a function \( F_i(U) \) (which also takes into account the interspecific interactions such as predation, competition, etc.), where \( U = (U_1, U_2, \ldots, U_n) \). The total increase in \( N_i \) over a time interval from \( t_1 \) to \( t_2 \) is then given by the following equation:

\[
\Delta N_i = N_i(T_1) - N_i(T_0) = \int_{T_0}^{T_1} \left[ -\int_S J_i ds + \int_V F_i(U) d\mathbf{R} \right] dT .
\]  

(8.4)

Equation (8.4) allows us, in principle, to calculate the population size of a given species at any time \( T \) provided that the initial species distribution is known. Note that it is obtained under rather unrestrictive assumptions and, as such, applies to a wide variety of real systems and situations. The reverse

\(^2\)In a still more general case, \( D_{ij} \) can be functions of time, space, and/or population density.
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side of its generality, however, is that it is very difficult to study its properties other than through straightforward numerical simulations.

In order to arrive at an equation of population dynamics in a more convenient form, we have to make additional assumptions. Namely, we assume that the population densities are smooth functions so that there exists a continuous first derivative with respect to time and the second derivative with respect to space. The second term on the right-hand side of (8.4) can then be written as

\[ -\int_S J_i ds = -\int_V \nabla J_i dR = \int_V \sum_{j=1}^{n} D_{ij} \nabla^2 U_j dR. \]  

(8.5)

Equation (8.4) takes the following form:

\[ \int_V [U_i(R, T_1) - U_i(R, T_0)] dR \]

(8.6)

\[ = \int_{T_0}^{T_1} \left[ \int_V \sum_{j=1}^{n} D_{ij} \nabla^2 U_j dR + \int_V F_i(U) dR \right] dT, \]

or

\[ \int_V \left( [U_i(R, T_1) - U_i(R, T_0)] - \int_{T_0}^{T_1} \left[ \sum_{j=1}^{n} D_{ij} \nabla^2 U_j + F_i(U) \right] dT \right) dR = 0. \]  

(8.7)

Let us note that Equation (8.7) is obtained for an unspecified volume \( V \). Therefore, it can only hold if the expression under the integral equals to zero identically, i.e.,

\[ [U_i(R, T_1) - U_i(R, T_0)] - \int_{T_0}^{T_1} \left[ \sum_{j=1}^{n} D_{ij} \nabla^2 U_j + F_i(U) \right] dT = 0. \]  

(8.8)

Now, considering the limiting case \( T_1 \rightarrow T_0 \), from (8.8) we obtain the diffusion–reaction equation describing the dynamics of the \( i \)th species:

\[ \frac{\partial U_i(R, T)}{\partial T} = \sum_{j=1}^{n} D_{ij} \nabla^2 U_j + F_i(U). \]  

(8.9)

Obviously, exactly the same chain of arguments can be repeated for any species from the given community so that, to describe the dynamics of an \( n \)-species system, we actually have a system of \( n \) equations (8.9), \( i = 1, 2, \ldots, n \).

Note that, in order to attain generality, the terms “volume” and “surface” have been used here in an abstract mathematical sense. Their heuristic
meanings depend on the dimensionality of the specific system; for instance, in the case of a population dwelling on the Earth’s surface (apparently two-dimensional), the “volume” actually means area and its boundary is a curve rather than a “surface.”

The system (8.9) together with some of its extensions will be a focus of our analysis for the rest of this book. Its properties essentially depend on the properties of functions \(F_i\), in particular, on the number of feasible steady states and their stability. For biological reasons, there must always exist at least one steady state, i.e., the extinction state \((0, \ldots, 0)\); however, conditions of existence/nonexistence of nontrivial equilibria as well as conditions of their stability vary very much from system to system. In fact, addressing these issues in terms of a biologically reasonable model is a core issue of mathematical biology, cf. Part II. Remarkably, as we will show in the following few chapters, some important features of the system spatiotemporal dynamics (cf. “biological turbulence”) appear to depend more on the system behavior in the vicinity of a given nontrivial steady state rather than on the global structure of the phase space.

Let us also mention that, while crossdiffusion is a rather common phenomenon in some other natural sciences, e.g., in chemistry, in population dynamics it is rarely observed. Therefore, in order to avoid unnecessary generalizations, in the following we neglect its effect assuming that \(D_{ij} = 0\) for \(i \neq j\).

The properties of the (nonlinear) functions \(F_i\) create a “skeleton” of the system dynamics; however, its spatiotemporal behavior also depends essentially on the initial conditions. Consider the case when, along with the extinction state, there exists a coexistence steady state \(\bar{U} = (\bar{U}_1, \bar{U}_2, \ldots, \bar{U}_n)\). In case the initial species distribution creates some sort of a boundary or front, i.e.,

\[
U_i(X, Y, T) \to 0 \quad \text{for} \quad X \to -\infty, \quad U_i(X, Y, T) \to \bar{U}_i \quad \text{for} \quad X \to \infty \quad (8.10)
\]

(i=1, \ldots, n), then the generic solution of the system (8.9) describes a traveling population front propagating along or against axis \(X\), cf. Kolmogorov et al. (1937) and Fisher (1937).

The traveling wave solutions of the diffusion–reaction system (8.9) are of high ecological relevance. They arise naturally in the problems related to exotic species spread, e.g., as a result of biological invasion (Shigesada and Kawasaki, 1997; Petrovskii and Li, 2006). In terms of pattern formation, they also provide the simplest pattern arising as a result of the system’s self-organization. However, since they have been studied intensively in relation to other applications (such as, for instance, combustion and flame propagation; see Zeldovich et al., 1985; Volpert et al., 1994), as well as in a somewhat more abstract mathematical aspect (Aronson and Weinberger, 1975, 1978; Fife, 1979), here we will not focus on traveling population fronts per se. What makes them relevant to the scope of this book is that the propagation of a traveling front may trigger the formation of complex spatiotemporal patterns.
That will be considered in detail in Chapter 10. In the next chapter, however, we start with a more classical mechanism of pattern formation that is not related to front propagation.
Chapter 9

Instabilities and dissipative structures

In the previous chapter, we mentioned a traveling population front as the simplest theoretical example of a spatiotemporal structure. A front separates space into two regions with different properties, e.g., quantified by high and low values of population density, respectively. In each of these regions the population is distributed homogeneously. Therefore, the system actually exhibits spatial heterogeneity (in terms of large gradients of the population density) only within a narrow crossover region.

Front-like structures or patterns play an important role in ecology, but they of course do not exhaust all possible scenarios of spatiotemporal population dynamics. Spatial distribution of ecological populations is usually very inhomogeneous, so that patches of high population density in some way alternate with patches of low population density. Correspondingly, of high ecological relevance are scenarios of pattern formation where self-organized spatial heterogeneity would not be confined to only a certain domain, which is much smaller in size than the area actually available for the population dynamics, but could be observed, in principle, throughout the whole space.

In this chapter, we revisit some classical mechanisms of pattern formation in diffusion–reaction models of population dynamics, namely, those resulting from the Turing instability and the differential flow instability. The seminal work by Turing (1952) was eventually followed by a huge number of other publications reporting examples of the Turing instability in various systems along with some generalizations of the original mechanism. In particular, this topic is covered by many reviews, textbooks, and research monographs in mathematical biology, e.g., see Okubo (1980), Meinhardt (1982), Murray (1989), Ben-Jacob et al. (2000), Britton (2003), and Solé and Bascompte (2006). Thus, since the main goal of this book is to give an account about recent advances in this field, we address the Turing instability only briefly (yet with enough details to make our description comprehensible); an interested reader can find more information in the sources cited above.

Although the original study done by Turing was concerned with interacting chemical species, Segel and Jackson (1972) showed that the same ideas could potentially be applied to population dynamics as well. Correspondingly, here we also recall some more recent studies that used the Turing ideas in order to explain patterns in population communities – in particular, the patterns ob-
served in semiarid vegetation systems; cf. Section 3.2.4. Finally, we conclude
the chapter with a discussion (Section 3.2.5) of the Turing scenario relevance
to ecological pattern formation in a general case.

9.1 Turing patterns

The basic idea of Turing’s work (Turing, 1952) is very simple, although
highly nontrivial: A steady state that is locally asymptotically stable in a
nonspatial system can become unstable in the corresponding diffusive sys-
tem. Relevant mathematical analysis shows that, on the onset of instability,
the system first becomes unstable with respect to a spatially heterogeneous
perturbation with a certain wavenumber. That leads to the formation of a
regular spatial structure.

Remarkably, although the Turing instability manifests itself in a most won-
derful way in nonlinear systems, where it results in the so-called “dissipative
patterns” (Glansdorff and Prigogine, 1971), in a purely mathematical per-
spective, it is actually a property of a linear system. Indeed, let us consider
the following system of linear diffusion–reaction equations:

\begin{align*}
\frac{\partial U(R,T)}{\partial T} &= D_1 \nabla^2 U + a_{11} U + a_{12} V , \quad (9.1) \\
\frac{\partial V(R,T)}{\partial T} &= D_2 \nabla^2 V + a_{21} U + a_{22} V , \quad (9.2)
\end{align*}

where $U$ and $V$ are the state variables for the two interacting agents at the
position $R = (X, Y)$ and time $T$.

Apparently, $(0, 0)$ is a steady state of the system (9.1)–(9.2) without space,
i.e., without diffusion terms. This state is stable under the following condi-
tions:

\begin{align*}
a_{11} + a_{22} &< 0 , \quad (9.3) \\
a_{11}a_{22} - a_{12}a_{21} &> 0 \quad (9.4)
\end{align*}

(which reflects the simple fact that both eigenvalues must have negative real
parts), and it is unstable if at least one of these conditions is violated. In
terms of the “full” system (9.1)–(9.2), conditions (9.3)–(9.4) mean that the
spatially homogeneous steady state $u(r) \equiv 0$, $v(r) \equiv 0$ is stable with respect
to spatially homogeneous perturbations.

We consider the case that the steady state $(0, 0)$ is stable so that conditions
(9.3)–(9.4) hold. Now, the question is whether the system remains stable in
the case of an inhomogeneous perturbation.

Mathematical treatment of this issue is slightly different depending on
whether the system (9.1)–(9.2) is considered in a bounded or in an unbounded
domain. In the former case, any solution of the system (9.1)–(9.2) can be expanded into a Fourier series so that

\[ U(R, T) = \sum_{n, m=0}^{\infty} U_{nm}(R, T) = \sum_{n, m=0}^{\infty} \alpha_{nm}(T) \sin kR, \quad (9.5) \]

\[ V(R, T) = \sum_{n, m=0}^{\infty} V_{nm}(R, T) = \sum_{n, m=0}^{\infty} \beta_{nm}(T) \sin kR \quad (9.6) \]

(assuming the zero-function Dirichlet conditions at the domain boundaries), where \(0 < x < L_x\) and \(0 < y < L_y\), \(L_x\) and \(L_y\) giving the size of the system in the directions of \(x\) and \(y\), respectively, \(k = (k_n, k_m)\) and \(k_n = \pi n/L_x, k_m = \pi m/L_y\) are the corresponding wavenumbers.

Since Equations (9.1)–(9.2) are linear, an interplay between different terms in (9.5)–(9.6) is impossible. Therefore, it is not necessary to work with the general solution (9.5)–(9.6) because it will decay with time if and only if \(u_{nm}\) and \(v_{nm}\) decay for any \(n\) and \(m\). (In more rigorous mathematical terms, it is a consequence of the fact that \(\cos (k_n x + k_m y)\) and \(\cos (k_p x + k_q y)\) are linearly independent for any \((n, m) \neq (p, q)\).) Thus, it is sufficient to consider the properties of a single pair \(u_{nm}, v_{nm}\) for unspecified indices \(n\) and \(m\).

Having substituted \(u_{nm}\) and \(v_{nm}\) into (9.1)–(9.2), we obtain:

\[ \frac{d\alpha_{nm}}{dT} = \left(a_{11} - D_1 k^2\right) \alpha_{nm} + a_{12} \beta_{nm}, \quad (9.7) \]

\[ \frac{d\beta_{nm}}{dT} = a_{21} \alpha_{nm} + \left(a_{22} - D_2 k^2\right) \beta_{nm}, \quad (9.8) \]

where \(k^2 = k_n^2 + k_m^2\).

A general solution of (9.7)–(9.8) has the form \(C_1 \exp(\lambda_1 t) + C_2 \exp(\lambda_2 t)\), where the constants \(C_1\) and \(C_2\) are determined by the initial conditions and the exponents \(\lambda_{1,2}\) are the eigenvalues of the following matrix:

\[ A = \begin{pmatrix} a_{11} - D_1 k^2 & a_{12} \\ a_{21} & a_{22} - D_2 k^2 \end{pmatrix}. \quad (9.9) \]

Correspondingly, \(\lambda_{1,2}\) arise as the solution of the following equation:

\[ \lambda^2 - (\hat{a}_{11} + \hat{a}_{22}) \lambda + (\hat{a}_{11}\hat{a}_{22} - a_{12} a_{21}) = 0, \quad (9.10) \]

where the notations \(\hat{a}_{11}, \hat{a}_{22}\) are introduced for convenience so that

\[ \hat{a}_{11} = a_{11} - D_1 k^2, \quad \hat{a}_{22} = a_{22} - D_2 k^2. \quad (9.11) \]

In the case that the system (9.1)–(9.2) is considered in an unbounded domain, \(-\infty < x, y < \infty\), Fourier series are no longer applicable; however, the
solution can be expanded into a Fourier integral:

\[
U(R, T) = \frac{1}{2\pi} \int \tilde{U}(T, k)e^{ikR}dk,
\]

\[
V(R, T) = \frac{1}{2\pi} \int \tilde{V}(T, k)e^{ikR}dk,
\]

where \(\tilde{U}(T, k)\) and \(\tilde{V}(T, k)\) are the Fourier transforms. Having substituted (9.12) into (9.1)–(9.2), we eventually arrive at the same Equation (9.10) for the linearized system’s eigenvalues.

An inhomogeneous perturbation to the homogeneous steady state, cf. (9.5)–(9.6), decays with time if and only if all eigenvalues have negative real parts. Thus, the conditions of stability of the state \(u(r) \equiv 0, v(r) \equiv 0\) with respect to a perturbation with a given wavenumber \(k\) are given by

\[
\hat{a}_{11} + \hat{a}_{22} < 0 \quad (9.13)
\]

and

\[
\hat{a}_{11}\hat{a}_{22} - \hat{a}_{12}\hat{a}_{21} > 0 . \quad (9.14)
\]

A few important conclusions can be made simply from comparison between (9.3)–(9.4) and (9.13)–(9.14), without making any calculations. Indeed, it is readily seen that:

- If condition (9.3) holds then condition (9.13) holds as well. Thus, a change in stability, if any, can only be associated with (9.14).
- In case both \(a_{11}\) and \(a_{22}\) are negative, (9.14) follows from (9.4). Thus, a change in stability is possible only if \(a_{11}\) and \(a_{22}\) are of different sign, so that \(a_{11}a_{22} < 0\).
- Therefore, \(a_{12}\) and \(a_{21}\) must be of different signs, \(a_{12}a_{21} < 0\), otherwise condition (9.4) is violated, which contradicts our assumption that \((0, 0)\) is locally stable.

Interestingly, the above conclusions do not yet tell much about the spatial aspect of the system, which is quantified by the diffusion coefficients. However, the impact of space becomes explicit when we consider inequality (9.14) in more details. From (9.14) and (9.11), we obtain:

\[
D_1D_2k^4 - (D_1a_{22} + D_2a_{11})k^2 + (a_{11}a_{22} - a_{12}a_{21}) \equiv Q(k^2) > 0 .
\]

Inequality (9.15) is a necessary condition of stability. Correspondingly, the locally stable homogeneous steady state becomes unstable with respect to a
perturbation with a given wavenumber $k$ if (9.15) is violated, i.e., if $Q(k^2) < 0$ (see Figure 9.1). By virtue of (9.4), it can only be possible if

$$D_1a_{22} + D_2a_{11} > 0.$$  \hspace{1cm} (9.16)

From (9.16), one can derive an important relation between the diffusion coefficients. Indeed, let us recall that $a_{11}$ and $a_{22}$ are of different signs. Assume that $a_{11} > 0$ and $a_{22} < 0$; then

$$a_{11} + a_{22} = a_{11} - |a_{22}| < 0$$

so that

$$a_{11} < |a_{22}| \quad \text{and} \quad \frac{a_{11}}{|a_{22}|} < 1.$$  \hspace{1cm} (9.17)

Therefore, from (9.16) we obtain:

$$\frac{D_1}{D_2} < \frac{a_{11}}{|a_{22}|} < 1.$$  \hspace{1cm} (9.18)

**FIGURE 9.1:** A sketch of the polynomial $Q(k^2)$ for different values of $\epsilon = D_1/D_2$: curve 1 for $\epsilon = 0.65$, curve 2 for $\epsilon = 0.48$, curve 3 for $\epsilon = 0.3$. Coefficients $a_{ij}$ are calculated as in the Segel-Jackson system (9.35)–(9.36) with $\eta = 0.85$ and $a = 0.8$; correspondingly, $a_{11} = 16$, $a_{12} = -17$, $a_{21} = 20$, and $a_{22} = -20$. 
It is readily seen that, in the opposite case $a_{11} < 0$ and $a_{22} > 0$, inequality (9.16) appears to be equivalent to $D_1/D_2 > 1$. Thus, we arrive at the following conclusion:

- Destabilization of the locally stable homogeneous steady state and the subsequent pattern formation are only possible if the diffusion coefficients are not equal.

Provided the inequality (9.16) holds, a sufficient condition for the instability onset is reached when $Q < 0$ in its minimum,

$$Q(k_m^2) < 0,$$

where

$$k_m^2 = \frac{D_1a_{22} + D_2a_{11}}{2D_1D_2}$$

(assuming that $D_1D_2 \neq 0$) and the corresponding spatial period is then given as

$$l_m = 2\pi \left(\frac{2D_1D_2}{D_1a_{22} + D_2a_{11}}\right)^{1/2}.$$

Taking into account (9.15), from (9.19) and (9.20) we obtain:

$$(a_{11}a_{22} - a_{12}a_{21}) - \frac{(D_1a_{22} + D_2a_{11})^2}{4D_1D_2} < 0.$$ (9.22)

Since both terms on the left-hand side are positive, cf. (9.4) and (9.16), we can take a square root so that (9.22) takes the following form:

$$D_1a_{22} + D_2a_{11} > 2(D_1D_2)^{1/2}(a_{11}a_{22} - a_{12}a_{21})^{1/2}.$$ (9.23)

Note that if (9.23) holds, the condition (9.16) holds automatically.

Inequality (9.23) considered together with (9.3)–(9.4) defines the domain in the parameter space of the system (9.1)–(9.2), where the Turing instability can be observed.

It should be mentioned that the system (9.1)–(9.2) by itself does not have any clear biological meaning. Normally, a solution of a diffusion–reaction equation would have the meaning of concentration or density of a given agent, e.g., the population density. However, in the system (9.1)–(9.2), the domain $U \geq 0$, $V \geq 0$ is not an invariant manifold and the system trajectories can cross the axes $U = 0$ and $V = 0$. This means that the population density can become negative (or vice versa, a population can appear from thin air), which is, of course, biological nonsense. What is important, however, is that any realistic system can be reduced to (9.1)–(9.2) in case we consider its behavior in vicinity of a homogeneous steady state. In this case, $U$ and $V$ denote
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not the population densities but (small) transient perturbations to the steady state values $\bar{U}$ and $\bar{V}$, respectively.

Indeed, let us consider a two-species system in a general form:

$$\frac{\partial U}{\partial T} = D_1 \nabla^2 U + \phi(U, V),$$  \hspace{1cm} (9.24)

$$\frac{\partial V}{\partial T} = D_2 \nabla^2 V + \psi(U, V),$$  \hspace{1cm} (9.25)

where different ecological situations can be taken into account by choosing an appropriate form of the functions $\phi$ and $\psi$; cf. Chapter 2.

Now, assume that for given nonlinear feedbacks $\phi(U, V)$ and $\psi(U, V)$ there exists a “positive” coexistence state $(\bar{U}, \bar{V})$, where $\bar{U} > 0$ and $\bar{V} > 0$. By means of introducing $\hat{U} = U - \bar{U}$ and $\hat{V} = V - \bar{V}$, where $\hat{U}$ and $\hat{V}$ are small, Equations (9.24)–(9.25) can be linearized so that we arrive at the system (9.1)–(9.2) where

$$a_{11} = \frac{\partial \phi}{\partial U}, \quad a_{12} = \frac{\partial \phi}{\partial V}, \quad a_{21} = \frac{\partial \psi}{\partial U}, \quad a_{22} = \frac{\partial \psi}{\partial V},$$  \hspace{1cm} (9.26)

and the hats are omitted.

Considering (9.26) together with the necessary conditions (9.3)–(9.4) (see also the conclusions itemized earlier in this section), one can immediately identify some cases where the Turing instability is impossible. In order to give an instructive example, let us consider a Lotka–Volterra system of two competing species:

$$\frac{\partial U}{\partial T} = D_1 \nabla^2 U + r_1 U \left(1 - \frac{\alpha_{11} U}{r_1}\right) - \alpha_{12} U V,$$  \hspace{1cm} (9.27)

$$\frac{\partial V}{\partial T} = D_2 \nabla^2 V + r_2 V \left(1 - \frac{\alpha_{22} V}{r_2}\right) - \alpha_{21} U V.$$  \hspace{1cm} (9.28)

The null-isoclines are given by the following equations:

$$r_1 - \alpha_{11} \bar{U} - \alpha_{12} \bar{V} = 0,$$  \hspace{1cm} (9.29)

$$r_2 - \alpha_{21} \bar{U} - \alpha_{22} \bar{V} = 0.$$  \hspace{1cm} (9.30)

Obviously, under some restrictions on the coefficients (for details, see Shigesada and Kawasaki, 1997), there exists a coexistence steady state $(\bar{U}, \bar{V})$. Calculating $a_{ij}$ according to (9.26) and taking into account (9.29)–(9.30), we then obtain

$$a_{11} = -\alpha_{11} \bar{U}, \quad a_{12} = -\alpha_{12} \bar{U},$$

$$a_{21} = -\alpha_{21} \bar{V}, \quad a_{22} = -\alpha_{22} \bar{V}.$$  

Since all $\alpha_{ij}$ are positive (or, at least, nonnegative), all $a_{ij}$ appear to be negative (nonpositive) and the necessary conditions for the Turing instability
are violated. Thus, we conclude that in a system of two competing species
the Turing instability cannot occur.

On the contrary, in a prey–predator system, the Turing instability may be-
come possible. To reveal possible constraints on the choice of parametrization,
let us first consider the following generic equations:

\[ \frac{\partial U}{\partial T} = D_1 \nabla^2 U + f(U)U - r(U)UV , \quad (9.31) \]
\[ \frac{\partial V}{\partial T} = D_2 \nabla^2 V + \kappa r(U)UV - g(V)V , \quad (9.32) \]

where we use slightly different notations compared to Chapter 2.

Correspondingly, the matrix entries are given as

\[ a_{11} = f'(\bar{U})\bar{U} - r'(\bar{U})\bar{U}V , \quad a_{12} = -r(\bar{U})\bar{U} , \]
\[ a_{21} = [r'(\bar{U})\bar{U} + r(\bar{U})]\kappa V , \quad a_{22} = -g'(\bar{V})\bar{V} , \]

where prime denotes a derivative with respect to the argument.

One important observation can be made already in this general case: The
Turing instability cannot occur if the predator mortality is density indepen-
dent. Indeed, if \( g(V) \equiv \text{const} \), then \( a_{22} = 0 \). Correspondingly, \( a_{11} \) must be
negative in order to ensure the local stability of the steady state, cf. (9.3),
which means that condition (9.16) cannot be fulfilled. Thus, the formation of
dissipative patterns in a prey–predator system is impossible unless the preda-
tor mortality is density dependant.\(^1\) In particular, it means that the Turing
instability is not possible in the classical Lotka–Volterra system.

More observations can be made in more specific situations. For instance,
let us assume that \( f(U) \) and \( g(V) \) are a decreasing and an increasing function,
respectively. (In biological terms, it may mean that the populations are not
affected by the Allee effect.) Then it is readily seen that the Turing instability
can only take place if density dependence in predation is taken into account.
Indeed, consider that predation is described by a bilinear term, which means
that \( r(U) = \text{const} \) and \( r'(U) = 0 \). Then we immediately obtain that both
\( a_{11} < 0 \) and \( a_{22} < 0 \), which makes the Turing instability impossible.

We want to emphasize here that relations (9.3)–(9.4) are only necessary
conditions for the Turing instability and they alone cannot guarantee that
the instability actually takes place. The full set of conditions also includes
relation (9.23), and then one must prove that for a given choice of functional
responses the corresponding domain in the parameter space is not empty. A
biologically reasonable parametrization usually turns conditions (9.3)–(9.4)
and (9.23) into a complicated system of inequalities that can only be solved
numerically. There are not many cases that can be treated analytically; the
one considered below was originally studied by Segel and Jackson (1972).

\(^1\)The situation is different if predation is ratio dependent, e.g., see Alonso et al. (2002).
In order to make the problem analytically solvable, we have to keep it as simple as possible (and yet biologically sensible). Therefore, we consider a prey–predator system where predation is described by a bilinear term. Correspondingly, to fulfill the necessary conditions for the Turing instability, we have to introduce the Allee effect into the prey growth. This is, however, in agreement with numerous biological data showing that the Allee effect is a common phenomenon in population dynamics (Courchamp et al., 1999). Also, we take into account density dependence in the predator mortality, which may account for either the intra-specific competition or the impact of a top predator (in the latter case it is called a “closure term,” cf. Steele and Henderson, 1992a). Having chosen a polynomial parametrization for $f(U)$ and $g(V)$, we arrive at the following system:

$$
\frac{\partial U}{\partial T} = D_1 \nabla^2 U + (A_0 + A_1 U - A_2 U^2)U - r_0 UV , \quad (9.33)
$$

$$
\frac{\partial V}{\partial T} = D_2 \nabla^2 V + \kappa r_0 UV - (B_0 + B_1 V)V , \quad (9.34)
$$

where $A_0$, $A_1$, $A_2$, $B_0$, and $B_1$ are nonnegative parameters.

Since here we are more interested in giving an example of a system prone to the Turing instability rather than in making a biologically meaningful prediction, in order to avoid tedious calculations we assume that $A_2 = B_0 = 0$. Introducing, for convenience, dimensionless variables as $t = A_0 T$, $x = (D_2/A_0)^{-1/2} X$, $y = (D_2/A_0)^{-1/2} Y$, $u = (r_0 \kappa / A_0) U$, and $v = (B_1/A_0) V$, from (9.33)–(9.34) we obtain:

$$
\frac{\partial u}{\partial t} = \epsilon \nabla^2 u + (1 + au)u - \eta uv , \quad (9.35)
$$

$$
\frac{\partial v}{\partial t} = \nabla^2 v + uv - v^2 , \quad (9.36)
$$

where $a = A_1/r_0 \kappa$, $\eta = r_0 / B_1$, and $\epsilon = D_1 / D_2$.

The null-isoclines of the system are given by

$$
1 + au - \eta v = 0 , \quad u - v = 0 , \quad (9.37)
$$

so that the steady-state density is

$$
\bar{u} = \bar{v} = \Omega , \quad (9.38)
$$

where $\Omega = (\eta - a)^{-1}$. Obviously, the steady state must be situated in the first quadrant; thus, we obtain the following constraint on the parameter values:

$$
\eta > a . \quad (9.39)
$$

For the matrix of the linearized system we have

$$
\begin{align*}
a_{11} &= a \Omega , & a_{12} &= - \eta \Omega , & a_{21} &= \Omega , & a_{22} &= - \Omega . \quad (9.40)
\end{align*}
$$
FIGURE 9.2: Parameter plane $(a, \eta)$ for the model (9.35)–(9.36) in the cases (a) $\epsilon = 0.1$ and (b) $\epsilon = 0.3$; other parameters are the same as in Figure 9.1. Turing instability can be observed for the parameter values inside the curvilinear triangle(s).
Takong (9.3) into account, we therefore obtain that

\[ a < 1 . \]  

(9.41)

Since \( a_{11} > 0 \) and \( a_{22} < 0 \), the condition (9.18) is applicable so that we obtain

\[ \epsilon < a . \]  

(9.42)

Finally, from (9.23) we arrive at

\[ a - \epsilon > 2\sqrt{\epsilon(\eta - a)} , \]  

(9.43)

from which we obtain that

\[ \eta < \frac{(a - \epsilon)^2}{4\epsilon} + a . \]  

(9.44)

The relations (9.39), (9.41), (9.42) and (9.44) taken together define the required domain in the parameter space; see Figure 9.2. It is readily seen that the diffusivity ratio is a controlling factor of crucial importance. While for \( \epsilon \ll 1 \) the parameter domain corresponding to the Turing instability can be of considerable size, for \( \epsilon \approx 1 \) it shrinks to a single point. This situation appears to be typical and does not depend much on the specific parametrization of the functional responses.

An interesting question is what the actual scenario of pattern formation may look like. The shape of the stationary structures achieved in the large-time limit is determined by the specific form of the problem nonlinearity (being described by a stationary solution of Equations (9.35)–(9.36), i.e., for \( \partial u/\partial t = \partial v/\partial t = 0 \) and therefore is unlikely to be affected by the choice of the initial conditions. However, the transient stage of the system dynamics obviously can be different depending on the presence/absence of “partial” perturbations with different \( k \).

In order to give an instructive example, we consider the dynamics of the system (9.35)–(9.36) in the one-dimensional case and for the following initial conditions:

\[ u(x,0) = \bar{u} , \]  

(9.45)

\[ v(x,0) = \bar{v} \text{ for } 0 < x < x_1 \text{ and } x_2 < x < L , \]  

(9.46)

\[ v(x,0) = \bar{v} + v_0 \sin \left( \frac{2\pi(x - x_1)}{l_0} \right) \text{ for } x_1 < x < x_2 , \]  

(9.47)

where the parameters have obvious meanings.

It can be expected that the system evolves differently depending on whether \( l_0 \) is “critical” (i.e., lies inside the critical range where the instability takes place) or not, i.e., whether \( Q(2\pi/l_0) \) is negative or positive (see Figure 9.1).
FIGURE 9.3: Pattern formation in the Segel–Jackson system (9.35)–(9.36) calculated numerically for the diffusivity ratio $\epsilon = 0.75$. Panels from top to bottom correspond to $t = 0$, $t = 5$, $t = 10$, and $t = 20$, respectively. Other parameters are $a = 0.95$ and $\eta = 0.96$, so that $l_m \approx 1.8$. The spatial period of the initial perturbation of the steady state is $l_0 = 5$.

Note that the function (9.46) is not monochromatic; due to its piecewise nature, its Fourier expansion apparently contains an infinite spectrum of wavenumbers. Thus, one can expect that, for parameter values allowing for the Turing instability, the patterns begin to emerge in the vicinity of points $x = x_1$ and $x = x_2$. As for the periodic initial perturbation in the middle of the domain, it will be “amplified” for critical values of $l_0$ and will decay otherwise. This heuristic expectation is confirmed by numerical simulations; see Figures 9.3 and 9.4. It is readily seen that the pattern first emerges in a certain subdomain, which appears to be different for $l_0$ being critical or
noncritical, and eventually spreads over the whole area.

FIGURE 9.4: Pattern formation in the Segel–Jackson system (9.35)-(9.36). Panels from top to bottom correspond to $t = 0$, $t = 5$, $t = 10$, and $t = 20$, respectively. All parameters are the same as in Figure 9.3, except for the wavelength of the initial perturbation, which is now $l_0 = 2$.

It should also be mentioned that the Turing patterns are rather sensitive to the boundary conditions. While the initial conditions are likely to affect only the transient stage of pattern formation, the boundary conditions may affect both the transient stage and the large-time asymptotics. In particular, in a spatially bounded system, a stationary pattern can be formed only in case the boundary conditions are consistent with the intrinsic properties of
the pattern and with the size and shape of the domain. A simple example of such consistency for the one-dimensional version of system (9.1)–(9.2) is given by the zero-flux Neumann conditions, provided \( L/l^* \) is an integer, where \( L \) is the length of the domain and \( l^* \) is the intrinsic spatial period of the emerging pattern. The situation when the boundary conditions are not consistent with the intrinsic properties of the patterns is usually called the “boundary forcing.” In this case, the system is more likely to evolve into a spatiotemporal pattern (e.g., periodical both in time and space) rather than into a stationary one; for more details and references, see Dillon et al. (1994) and Shoji and Iwasa (2003), and also Meinhardt (1982).

How can pattern formation due to the Turing instability take place in reality? Let us consider a certain natural system (e.g., a population community) that is in a spatially homogeneous steady stable equilibrium. Such an equilibrium normally appears as a result of balance between various deterministic “forces.” However, the dynamics of any real system is actually a result of the interplay between deterministic and stochastic factors, and this steady state is continuously disturbed by spatially heterogeneous stochastic perturbations and fluctuations. Unless the magnitude of these perturbations is very large, which rarely happens, and the parameters are outside the Turing instability range, the perturbations decay with time and the system remains in a close vicinity of the steady state; therefore, no patterns emerge.

Now, consider the case when a certain parameter is gradually changing with time; for population communities it can be, for instance, either a result of adaptation to climate change or a consequence of natural selection. If it happens that the parameter value enters the domain in the parameter space of the given system where the system becomes unstable to heterogeneous perturbation, the corresponding spatial modes will be amplified, and that results in pattern formation.

### 9.1.1 Turing patterns in a multispecies system

In the above section, we were concerned with a dissipative instability in a system of two interactive species. A question that immediately arises is whether a similar scenario can be observed for a system with more than two species. The Turing instability is widely regarded to be responsible for morphogenesis (Turing, 1952; Meinhardt, 1982; Murray, 1989) and is also considered as a probable explanation for pattern formation in some ecological communities (Segel and Jackson, 1972; Levin and Segel, 1976; Alonso et al., 2002); however, natural systems are normally multispecific.

A closer look at the analysis done in the previous section shows that, from a mathematical point of view, a search for instability conditions is essentially an eigenvalue problem. Indeed, let us consider the following general model of an \( N \)-species community:

\[
\frac{\partial U_i}{\partial t} = D_i \nabla^2 U_i + f_i(U_1, \ldots, U_N), \quad i = 1, \ldots, N ,
\]
where notations have the same meanings as above.

It is readily seen that the corresponding linearized system in the vicinity of a spatially homogeneous steady state \((\bar{U}_1, \ldots, \bar{U}_N)\) has the form
\[
\frac{du_i(t)}{dt} = \sum_{j=1}^{N} a_{ij} u_j(t), \quad i = 1, \ldots, N, \quad (9.49)
\]
in case of a spatially homogeneous perturbation, and
\[
\frac{d\tilde{u}_i(t;k)}{dt} = \sum_{j=1}^{N} (a_{ij} - k^2 D_i \delta_{ij}) \tilde{u}_j(t;k), \quad i = 1, \ldots, N, \quad (9.50)
\]
in case of a spatially inhomogeneous perturbation with the wavenumber \(k\).
Here \(a_{ij} = \partial f_i / \partial U_j\) (where the derivatives are taken in the steady state), \(\delta_{ij}\) is the Kronecker symbol, and \(u_i(t)\) and \(\tilde{u}_i(t;k)\) are the amplitudes of the perturbation and its Fourier transform, respectively. Obviously, for \(k = 0\), system (9.50) coincides with (9.49).

According to preconditions of the Turing instability, the steady state \((\bar{U}_1, \ldots, \bar{U}_N)\) is assumed to be stable with respect to spatially homogeneous perturbations. Therefore, the Turing instability takes place when all eigenvalues of the system (9.49) have negative real parts but at least one eigenvalue of the system (9.50) has, at least for some \(k\), a positive real part. Therefore, identification of the Turing instability is equivalent to identification of the corresponding eigenvalue properties for matrix \(A(k)\) with \(k = 0\) and \(k > 0\).

For the sake of clarity, and in order to avoid tedious calculations, here we consider in some detail only a special case of a three-species system basing mainly on the paper by Qian and Murray (2003). A more general study of this problem, which addresses the case of a system with an arbitrary number of interacting species, can be found in Satnoianu et al. (2000).

Let us consider the following general model of a three-species community:
\[
\frac{\partial U}{\partial T} = D_1 \nabla^2 U + \phi(U, V, W), \quad (9.51)
\]
\[
\frac{\partial V}{\partial T} = D_2 \nabla^2 V + \psi(U, V, W), \quad (9.52)
\]
\[
\frac{\partial W}{\partial T} = D_3 \nabla^2 W + \rho(U, V, W). \quad (9.53)
\]

The matrix of the linearized system is
\[
A(k) = \begin{pmatrix}
a_{11} - D_1 k^2 & a_{12} & a_{13} \\
a_{21} & a_{22} - D_2 k^2 & a_{23} \\
a_{31} & a_{32} & a_{33} - D_3 k^2
\end{pmatrix}. \quad (9.54)
\]
The eigenvalues are the solutions of the characteristic equation, i.e.,

\[ \lambda^3 + p_2 \lambda^2 + p_1 \lambda + p_0 = 0 \]  \hspace{1cm} (9.55)

where

\[ p_2 = -\text{tr} A(k), \quad p_0 = -\det A(k) \]  \hspace{1cm} (9.56)

\[ p_1 = M_{11}(k) + M_{22}(k) + M_{33}(k) \],

and \( M_{ij} \) are cofactors.

According to the Routh–Hurwitz criterium (see Section 4.4.3), all the eigenvalues have negative real parts if and only if the following conditions hold:

(a) \( p_2 > 0 \),

(b) \( p_0 > 0 \),

(c) \( q = p_0 - p_1 p_2 < 0 \).  \hspace{1cm} (9.57)

Since the steady state is locally stable, the properties (a)–(c) hold for \( k = 0 \). What we need to do now is to understand how the values of \( p_0, p_2, \text{ and } q \) change with an increase in \( k \), in particular, whether it may happen that for some \( k \) one or more of the conditions (a)–(c) above get violated. The latter situation would mean instability with respect to a perturbation with a given wavenumber \( k \).

By direct calculation, we obtain that

\[ q = \text{tr} A \cdot (M_{11} + M_{22} + M_{33}). \]  \hspace{1cm} (9.58)

From (9.56), (9.57), and (9.58), we readily see that necessary conditions of the steady state stability are \( \text{tr} A < 0 \) and \( \det A < 0 \). Since

\[ \text{tr} A(k) = \text{tr} A(0) - \sum_{i=1}^{3} D_i k^2 \]  \hspace{1cm} (9.59)

and \( \text{tr} A(0) < 0 \), it is clear that condition (9.57a) cannot be violated for any \( k > 0 \).

In order to reveal how \( \det A \) changes with an increase in \( k \), we may notice that its value is affected by inhomogeneity only through the matrix diagonal elements. Therefore, we can consider \( \det A \) as a function of three variables, \( a_{11}, a_{22}, \text{ and } a_{33} \), and apply a standard differential-based technique:

\[ d (\det A) = \sum_{i=1}^{3} \frac{\partial (\det A)}{\partial a_{ii}} d(a_{ii}) \]  \hspace{1cm} (9.60)

\[ = M_{11} d(a_{11}) + M_{22} d(a_{22}) + M_{33} d(a_{33}) , \]

where \( d(a_{ii}) < 0, i = 1, 2, 3 \). Recalling that \( \det A(0) < 0 \), in case all \( M_{ii} > 0 \) \( (i = 1, 2, 3) \) we obtain \( d(\det A) < 0 \), which means that an increase in \( k \) cannot break condition (9.57b).
Similarly to (9.57b), for the condition (9.57c) we obtain

\[
\frac{dq}{da_{ii}} = \sum_{i=1}^{3} \frac{\partial q}{\partial a_{ii}} d(a_{ii}) = \left[ M_{22} + M_{33} + (\text{tr}A)^2 - a_{11}\text{tr}A \right] d(a_{11}) + \left[ M_{11} + M_{33} + (\text{tr}A)^2 - a_{22}\text{tr}A \right] d(a_{22}) + \left[ M_{11} + M_{22} + (\text{tr}A)^2 - a_{33}\text{tr}A \right] d(a_{33}).
\]

Assuming all \( M_{ii} > 0 \) in order to satisfy condition (9.57b), and since \( \text{tr}A < 0 \), we obtain that condition \( a_{ii} < 0 \) (\( i = 1, 2, 3 \)) is sufficient to make \( dq < 0 \). Recalling that (9.57c) holds for \( k = 0 \), it means that it cannot be violated for any \( k > 0 \).

From (9.60) and (9.61), we arrive, respectively, at the following conditions of the steady-state “absolute” stability (i.e., stability for any \( k \)):

(i) All diagonal cofactors of matrix \( A(k) \) must be positive;

(ii) All diagonal elements of matrix \( A(k) \) must be negative.

The two above conditions taken together are sufficient to ensure absolute stability of a given steady state. It means that instability for some \( k > 0 \) can only be observed if at least one of them is violated. Thus, we arrive at the following necessary condition of Turing instability (Qian and Murray, 2003):

- The largest diagonal element of matrix \( A(k) \) must be positive and/or the smallest diagonal cofactor of matrix \( A(k) \) must be negative.

We want to emphasize that this is a necessary condition and it does not, by itself only, guarantee onset of the Turing instability. In order to obtain a sufficient condition of the Turing instability, we have to additionally introduce certain constraints on the values of the species diffusivity.

Although in the case of a three-species system it is hardly possible to obtain explicit analytical relations similar to (9.16) and (9.18), an instructive example can be built easily. By virtue of the Routh–Hurwitz criterium, instability takes place if and only if one of the conditions (9.57a–9.57c) is broken. Let us consider (9.57b). After some standard although rather tedious calculations, we obtain:

\[
p_0(k) = D_1 D_2 D_3 k^6 - (D_1 D_2 a_{33} + D_2 D_3 a_{11} + D_1 D_3 a_{22}) k^4 + (D_1 M_{11} + D_2 M_{22} + D_3 M_{33}) k^2 - \det A.
\]

Under conditions (i)–(ii) all terms on the right-hand side are positive and so \( p_0(k) > 0 \) for any \( k \).

Now, let us assume, without any loss of generality, that the smallest cofactor is \( M_{33} \), so that \( M_{33} < 0 \). The third term on the right-hand side of (9.62) then becomes negative if \( D_1 \) and \( D_2 \) are sufficiently small. Let \( D_1/D_3 \ll 1 \) and
\[ \frac{D_2}{D_3} \ll 1; \text{ then } p_0(k) < 0 \text{ for some } k > k_0, \text{ where} \]
\[ k_0 \approx \left( \frac{\det A}{D_3 M_{33}} \right)^{1/2}. \tag{9.63} \]
(9.63) 
(Note that, for \( k \to \infty \), \( p_0(k) \) is always positive due to the contribution of the higher-order terms in (9.62), however small [but positive] the coefficients \( D_1 \) and \( D_2 \) can be.)

In a similar manner, let us assume that all diagonal cofactors are positive and the largest diagonal element is \( a_{11} \), so that \( a_{11} > 0 \). Considering the case that \( D_2 \sim D_3 \) but \( D_1/D_2 \ll 1 \) and \( D_1/D_3 \ll 1 \), from (9.62) we obtain:
\[ p_0(k) \approx -(D_2 D_3 a_{11}) k^4 + (D_2 M_{22} + D_3 M_{33}) k^2 - \det A. \tag{9.64} \]
Clearly, the right-hand side of (9.64) becomes negative for sufficiently large \( k \).

The above condition of the Turing instability has been obtained in terms of diagonal elements of the community matrix and the corresponding cofactors. We should recall, however, that \( M_{ii} \) obviously depend on \( k \). Therefore, now we have to find out under what conditions the cofactors actually remain positive for any \( k \) and under what conditions they may become negative.

Consider cofactor \( M_{11} \):
\[ M_{11} = \begin{vmatrix} a_{22} - D_2 k^2 & a_{23} \\ a_{32} & a_{33} - D_3 k^2 \end{vmatrix} = (a_{22} - D_2 k^2) (a_{33} - D_3 k^2) - a_{23} a_{32}. \]

Assuming all \( a_{ii} \) are negative, \( a_{23} a_{32} < 0 \) is a sufficient condition for \( M_{11}(k) > M_{11}(0) > 0 \ \forall k \). Similarly, it is readily seen that \( a_{13} a_{31} < 0 \) and \( a_{12} a_{21} < 0 \) are sufficient conditions for \( M_{22}(k) \) and \( M_{33}(k) \) being positive for any \( k \).

Note that the latter conditions have a clear biological interpretation. If, for any two species \( i \) and \( j \), the two corresponding elements \( a_{ij} \) and \( a_{ji} \) of the community matrix are of different sign, it means that the interspecific interaction is asymmetrical. Growth of one of these species appears to be damaging for the other one; if one of the species wins, the other looses; etc. Immediate biological examples are given by prey–predator and host-parasite interactions. In a more general perspective, it is sometimes said that the species interacting in this way make an “activator–inhibitor” pair. Thus, condition (i) of absolute stability can be reformulated in a more general way as follows:

(i) For each pair of species, their interaction must be of the activator–inhibitor type.

In the case that the three species described by system (9.51)–(9.53) correspond to three trophic levels, e.g., prey, predator, and top predator, the above condition means that there must be a certain “looping” in the species interaction so that the upper level can be negatively affected by the lower one by means of some direct interactions.
Correspondingly, a necessary condition of Turing instability is that at least one of the following inequalities should hold:

\[ a_{12}a_{21} > 0 , \quad a_{13}a_{31} > 0 , \quad a_{23}a_{32} > 0 . \]  

(9.65)

The fact that the coefficients \(a_{ij}\) and \(a_{ji}\) have the same signs means that the species \(i\) and \(j\) interact in a “symmetric” way. The simplest example of such an interaction is given by competition; cf. Chapter 2. Comparing this conclusion to the conditions of Turing instability in a two-species system, we now arrive at a curious result: In a system of two competing species, Turing’s instability is impossible, but it may become possible when the two species are affected by the same predator. Therefore, one might expect that the conditions of Turing instability are somewhat less restrictive in a multispecific community than they are in the two species case.

9.2 Differential flow instability

Pattern formation resulting from the Turing instability in a system of at least two interacting diffusive species is a phenomenon of great theoretical and practical interest. It is currently regarded as a generic process in morphogenesis, and it is also potentially important for ecological applications making a theoretical background for studying the heterogeneous distribution of ecological populations. However, it also has some inherent constraints; in particular, in a two-species system, the difference in species diffusivity must be sufficiently large. Apparently, this is not always the case in nature, and that gives a reason to look for some alternative scenarios of pattern formation.

It seems obvious that self-organized spatial patterning can only become possible when there is a relevant mechanism of species transport “integrating” the local dynamics at different positions into a single spatiotemporal system, which otherwise would be an ensemble of disconnected points or sites.\(^2\) One mechanism of transport is diffusion, and that may result in diffusive instability. Note that diffusion must not necessarily be of the Brownian type; a possibility of pattern formation due to a diffusive instability in a more complicated system with fractional diffusion has been reported recently; see Gafiychuk and Datsko (2006). Another physically different mechanism is advection. Therefore, a question arises whether the existence of an advective flow in a system of interacting species may result in the instability of an otherwise stable homogeneous state.

\(^2\)Here we are not interested in pattern formation in a disconnected system that may arise, for instance, as a result of specific initial conditions or inhomogeneous forcing.
Let us consider the following advection–reaction system:

\[
\frac{\partial U}{\partial T} = \phi(U, V) + w_u \nabla U , \quad (9.66)
\]

\[
\frac{\partial V}{\partial T} = \psi(U, V) + w_v \nabla V , \quad (9.67)
\]

where \( U \) and \( V \) are the population densities and \( w_u \) and \( w_v \) are constant velocities of advective transport of species \( U \) and \( V \), respectively.

In a general case, both velocities can be nonzero. However, by introducing the speed of relative motion,

\[
w = w_u - w_v,
\]

and considering the system dynamics in a framework moving with speed \( w_v \), Equations (9.66)–(9.67) turn into

\[
\frac{\partial U}{\partial T} = \phi(U, V) + w \nabla U , \quad (9.68)
\]

\[
\frac{\partial V}{\partial T} = \psi(U, V) \quad (9.69)
\]

without any loss of generality. A nontrivial impact of advection may be expected if \( w \neq 0 \), in which case the system (9.68)–(9.69) is called a system with differential flow (Rovinsky and Menzinger, 1992).

Now, we assume that the corresponding nonspatial system, i.e., system (9.68)–(9.69) without the advection terms, possesses a stable steady state \( (\bar{U}, \bar{V}) \) so that \( \phi(\bar{U}, \bar{V}) = \psi(\bar{U}, \bar{V}) = 0 \). Obviously, in the spatial system it corresponds to the homogeneous steady state \( U \equiv \bar{U}, \ V \equiv \bar{V} \).

Since we are interested in stability, we focus on the system dynamics in the vicinity of \( U \equiv \bar{U}, \ V \equiv \bar{V} \). Therefore, introducing new variables as \( \hat{U} = U - \bar{U} \) and \( \hat{V} = V - \bar{V} \), which we assume to be small, from (9.68)–(9.69) we arrive at the following linearized system:

\[
\frac{\partial \hat{U}}{\partial T} = a_{11} \hat{U} + a_{12} \hat{V} + w \nabla \hat{U} , \quad (9.70)
\]

\[
\frac{\partial \hat{V}}{\partial T} = a_{21} \hat{U} + a_{22} \hat{V} , \quad (9.71)
\]

omitting hats for simplicity. Local stability of the steady state \( (\bar{U}, \bar{V}) \) implies that \( \text{tr} \ A = a_{11} + a_{22} < 0 \) and \( \text{det} \ A = a_{11}a_{22} - a_{12}a_{21} > 0 \).

Further analysis is similar to the one that we have previously applied to diffusive instability. For the sake of brevity, we restrict our consideration to the case of an unbounded space. Solution of the system (9.70)–(9.71) can be expanded into a Fourier integral; cf. Equation (9.12). Since the system (9.70)–(9.71) is linear, in order to study its stability it is sufficient to study the solution properties for a given \( k \):

\[
U_k(R, T) = \hat{U}(T, k)e^{ikR} , \quad V_k(R, T) = \hat{V}(T, k)e^{ikR} . \quad (9.72)
\]
Apparently, local stability of the system (9.70)–(9.71) means that the mode with $k = 0$ is stable.

Substituting (9.72) into (9.70)–(9.71), we obtain:

$$\frac{d\tilde{U}_k(T)}{dT} = (a_{11} - i kw) \tilde{U}_k + a_{12} \tilde{V}_k, \quad (9.73)$$

$$\frac{\tilde{V}_k(T)}{dT} = a_{21} \tilde{U}_k + a_{22} \tilde{V}_k, \quad (9.74)$$

where $i^2 = -1$. Correspondingly, the eigenvalues $\lambda_{1,2}$ are the solutions of the following equation:

$$\begin{vmatrix}
  a_{11} + i kw - \lambda & a_{12} \\
  a_{21} & a_{22} - \lambda 
\end{vmatrix} = 0,
\quad (9.75)$$

that is,

$$(a_{11} + i kw - \lambda)(a_{22} - \lambda) - a_{12} a_{21} = 0,\quad (9.76)$$

so that

$$\lambda_{1,2}(k) = \frac{1}{2} \left( \text{tr} A + i kw \pm \sqrt{\Gamma} \right), \quad (9.77)$$

where

$$\Gamma = \Delta - (kw)^2 + 2i kw (a_{11} - a_{22}), \quad (9.78)$$

where $\Delta = (\text{tr} A)^2 - 4 \det A$.

For the equilibrium state to become unstable, there must exist an eigenvalue with a positive real part. After standard although rather tedious calculations, from (9.78) we obtain the following expression for the largest real part:

$$\text{Re} \lambda(k) = \frac{\text{tr} A}{2} + \frac{1}{2\sqrt{2}} \cdot \left( \left[ (\Delta - (kw)^2)^2 + 4(kw)^2(a_{11} - a_{22})^2 \right]^{1/2} + \Delta - (kw)^2 \right)^{1/2}. \quad (9.79)$$

Recall that $\text{tr} A < 0$ because the steady state is assumed to be locally stable. However, the second term in (9.79) is apparently positive. The question then is, how actually large can it be for different $k$?

For $k = 0$ (i.e., for a spatially homogeneous perturbation), Equation (9.79) turns into

$$\text{Re} \lambda(0) = \frac{1}{2} \left[ \text{tr} A + \left( \frac{\Delta + |\Delta|}{2} \right)^{1/2} \right], \quad (9.80)$$
FIGURE 9.5: The real part of the eigenvalues vs wavenumber $k$ of an inhomogeneous perturbation of a locally stable homogeneous equilibrium state in a system with a differential advective flow, cf. (9.70)–(9.71); curve 1 for $w = 10$, curve 2 for $w = 1$, curve 3 for $w = 0.1$. Other parameters are $a_{11} = 16$, $a_{12} = -17$, $a_{21} = 20$, and $a_{22} = -20$.

which is negative by the assumption. However, in the short-wave limit $kw \to \infty$, (9.79) takes the following form:

$$\text{Re} \lambda(\infty) = \frac{1}{2} (\text{tr}A + |a_{11} - a_{22}|).$$

(9.81)

The left-hand side of (9.79), considered as a function of $kw$, may have a shallow minimum for intermediate values of its argument, but it will be increasing for large $kw$. The outcome then depends on the relation between the matrix elements $a_{11}$ and $a_{22}$. Three different cases are possible:

(a) $a_{11} < 0$, $a_{22} > 0$,  
(b) $a_{11} > 0$, $a_{22} < 0$,  
(c) $a_{11} < 0$, $a_{22} < 0$.

(9.82)

It is readily seen that, in case both $a_{11}$ and $a_{22}$ are negative, $\text{Re} \lambda(\infty)$ is negative as well and no instability can take place. However, in the cases (9.82a) and (9.82b), we obtain $\text{Re} \lambda(\infty) = a_{22}$ and $\text{Re} \lambda(\infty) = a_{11}$, respectively, which means that the spatially homogeneous steady state becomes unstable for sufficiently large $k$. Thus, we arrive at the following necessary condition of differential flow instability:

- A change in stability is possible only if $a_{11}$ and $a_{22}$ are of different signs so that $a_{11}a_{22} < 0$. 


Note that it coincides with one of the conditions of diffusive instability; cf. the lines below Equations (9.13)–(9.14).

Re $\lambda(k)$ as given by Equation (9.79) is shown in Figure 9.5 for the case $kw = kw$. The system becomes unstable for $k > k_0$, where $\text{Re} \lambda(k_0) = 0$, the expression for the critical wavenumber $k_0$ being obtained from (9.79):

$$k_0 = -\frac{\text{tr}A}{w} \left( -\frac{\det A}{a_{11}a_{22}} \right)^{1/2}.$$  \hspace{1cm} (9.83)

In full agreement with the above analysis, the larger the speed $w$ of the differential flow, the smaller is $k_0$.

It should also be mentioned that the system response to perturbations with different $k$ is apparently anisotropic. For the same value of $k$, the largest value of $\text{Re} \lambda$ is reached when $k$ is parallel to $w$, while in the direction perpendicular to $w$ the spatially homogeneous steady state remains stable for any $k$. Consequently, Equation (9.83) gives the minimum possible critical value of $k$.

Thus, similar to the case of diffusive instability, the impact of a differential flow between two interacting species can make the spatially homogeneous steady state unstable to heterogeneous perturbations. The steady state remains stable with respect to long-wave (small $k$) spatial perturbations but will be destabilized by short-wave perturbations when the wavenumber exceeds a certain critical value.

There is one important distinction, though. In the diffusive-driven instability, the emerging positive eigenvalue is real; however, in the differential flow instability, the eigenvalue with a positive real part is apparently complex. Correspondingly, while the Turing patterns are standing, the existence of a non-zero imaginary part results in a traveling wave-type structure: It is readily seen from (9.72) that an emerging periodical pattern with an overcritical wavenumber $k$ will be traveling with a constant velocity

$$s = -\frac{k}{k^2} \text{Im} \lambda(k),$$ \hspace{1cm} (9.84)

where

$$\text{Im} \lambda(k) = \frac{kw}{2} + \frac{\text{sign}(a_{11} - a_{22})}{2\sqrt{2}}.$$ \hspace{1cm} (9.85)

$$\cdot \left( \left[ (\Delta - (kw)^2)^2 + 4(kw)^2(a_{11} - a_{22})^2 \right]^{1/2} - \Delta + (kw)^2 \right)^{1/2}.$$

Now let us recall that the whole analysis has been done in the framework moving with speed $w_c$; cf. the lines above Equations (9.68)–(9.69). It means that, in the original framework, the emerging periodical structure will be traveling with velocity $w_c + w$; the instability conditions apparently remain the same regardless of the choice of framework.
FIGURE 9.6: The largest real part of the eigenvalues vs wavenumber $k$ in a system with a differential flow and diffusion in the case that diffusivity is equal for both species. Here curve 1 for $w = 10$, curve 2 for $w = 2$, curve 3 for $w = 1$, curve 4 for $w = 0.1$; other parameters are the same as in Figure 9.5.

Note that the speed (9.85) of the traveling pattern is obviously different from $w$, its value depending both on the speed $w$ of the “external” flow and on the intrinsic system’s kinetics quantified by the matrix $(a_{ij})$. This is a clear manifestation of the self-organized system’s dynamics. The instability itself and the subsequent pattern formation are induced by the impact of the differential flow, but it cannot be reduced to it.

A drawback of the model system (9.66)–(9.67), or (9.68)–(9.69), is that the homogeneous steady state appears to be unstable with respect to modes with an infinitely large wavenumber, i.e., for any $k > k_0$ (see Figure 9.5 and Equation (9.83)). Since a perturbation of an arbitrary shape contains, in a general case, the whole spectrum of $k$, this means that even a very slow differential flow would destabilize the system. This does not seem realistic. Indeed, one can expect that perturbations from a short-wave part of the spectrum should be damped down by diffusion, which is always present in any physical, chemical, or biological system, even if its impact can be sometimes quite small compared to the impact of advection.

Therefore, although system (9.68)–(9.69) was very useful to demonstrate the principal possibility of steady-state destabilization by a differential flow,
**FIGURE 9.7:** The largest real part of the eigenvalues vs wavenumber $k$ arising as a result of the combined effect of differential flow and differential diffusivity: (a) for $w = 2$ and (b) for $w = 0.1$. Here curve 1 for $\epsilon = D_1/D_2 = 1$, curve 2 for $\epsilon = 0.5$ and curve 3 for $\epsilon = 0.2$; other parameters as in Figure 9.5.
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A more reasonable model should include diffusion for both species:

\[
\frac{\partial U}{\partial T} = a_{11} U + a_{12} V + w \nabla U + D_1 \nabla^2 U ,
\]

(9.86)

\[
\frac{\partial V}{\partial T} = a_{21} U + a_{22} V + D_2 \nabla^2 V .
\]

(9.87)

A complete model should also account for cross-diffusion, cf. Malchow (2000a); here we assume that its impact can be neglected.

Without advection, the system (9.86)–(9.87) was shown to be prone to diffusive instability (see Section 9.1) while without diffusion it is subject to differential flow instability. The question now arises what may be an outcome of the interplay between the two types of instability?

Focusing on the behavior of a kth mode, cf. (9.72), it is readily seen that Equations (9.73) to (9.79) remain essentially the same up to the change \( a_{11} \to \tilde{a}_{11} = a_{11} - D_1 k^2 \) and \( a_{22} \to \tilde{a}_{22} = a_{22} - D_2 k^2 \). The equation for \( \text{Re} \lambda(k) \) now appears to be too cumbersome to be treated analytically; in particular, an explicit expression for the critical wavenumber is not available. However, it is straightforward to plot it as a function of \( k \). Recalling that the system’s stability limit is lowest with respect to perturbations with \( k \) being parallel to \( w \), we restrict our further insights to the case \( kw = kw \).

We begin with the case \( D_1 = D_2 \) when no diffusive instability is possible. Figure 9.6 shows the maximum real part of the eigenvalues as a function of \( k \). Apparently, the impact of diffusion changes the situation considerably. Contrary to the purely advective system, cf. Figure 9.5, now the homogeneous steady state can become unstable only with respect to modes with the wavenumbers from a certain intermediate range, \( k_{\text{min}} < k < k_{\text{max}} \). Moreover, for small values of \( w \) (see curves 3 and 4), \( \text{Re} \lambda \) appears to be negative for any \( k \). Consequently, instability onset is only possible if the speed of the differential flow is large enough.

In case \( D_1 \neq D_2 \), the impact of differential diffusivity enlarges the parameter range where instability occurs. Figure 9.7a shows \( \text{Re} \lambda(k) \) for different diffusivity ratios \( \epsilon = D_1 / D_2 \) in a situation when the differential advective flow alone would be strong enough to destabilize the system; cf. curve 1. However, differential diffusivity makes the effect more prominent by means of increasing the relevant range of \( k \).

In case the flow is not strong enough, instability onset is still possible due to the impact of differential diffusivity. Curve 1 in Figure 9.7b shows \( \text{Re} \lambda(k) \) in the case when the differential flow instability is not possible. As soon as the diffusivity ratio becomes sufficiently large, stability of the homogeneous steady state gets broken; there emerges a range of \( k \) where the eigenvalues’ real part is positive (curve 3).

Thus, in this section we have shown that, in a system of two interacting species with diffusion and a differential advective flow, a locally stable homogeneous steady state may become unstable with respect to heterogeneous
perturbation with a wavenumber form a certain intermediate range. As a result of instability onset, the system becomes capable of developing periodic spatial patterns of the traveling wave type.

A question may yet remain regarding the ecological significance of this generic mechanism. A relevant pair of species is given by a prey–predator system; however, the situation when the predator and prey populations move with respect to each other may seem rather exotic (but see Malchow and Shigesada, 1994). This seeming difficulty is immediately resolved if we realize that the interacting agents must not necessarily be biological species but can be treated, for instance, as a resource–consumer system. This apparently broadens the range of potential ecological applications of the above scenario; one appropriate example will be considered below.

9.3 Ecological example: semiarid vegetation patterns

In the previous sections of this chapter we revisited some classical theoretical approaches describing pattern formation in a system of two or more interacting species. A common feature of these approaches is that pattern formation becomes possible as a result of system instability with respect to a heterogeneous perturbation with a certain wavelength – or, more generally, wavelengths or wavenumbers from a certain range. For that reason, the above mechanisms, although apparently different in specific details, are often known under the same name of a “Turing-type” scenario. As a result of a Turing-type instability, the emerging patterns appear to be periodical. That brings a certain problem when one tries to link the Turing-type scenario(s) to the spatiotemporal patterns in ecosystems because the spatial distribution of ecological populations is usually highly irregular.

There is, however, at least one example of spatial patterning in ecosystems where the structures exhibit a remarkable degree of regularity. Vegetation in arid and semiarid regions of the world is often distributed in space not randomly but forms distinct regular patterns, normally in the form of stripes, where areas covered with bushes and trees alternate with areas/patches of grass or even bare ground; see Figure 9.8. Due to the apparent visual similarity of the structure to the pattern on the skin of a tiger, this vegetation pattern is often referred to as the “tiger bush.” The phenomenon was originally reported by MacFadyen (1950a,b) and for some time it was regarded as an exotic feature of a particular land. The reason is that the variation of the population density across the stripes is not always easily observed on the spot, although in most cases it can be readily seen from the air. Thus, it was not before significant development of airborne methods of field observations, such as aerial photography, that it became clear that vegetation stripes in semiarid
regions is a usual rather than an exotic phenomenon (Beard, 1967; White, 1969, 1970; Bernd, 1978; Greig-Smith, 1979); for more references also see Lefever and Lejeune (1997).

**FIGURE 9.8:** An example of tiger bush vegetation pattern in Niger (with permission from Lejeune et al., 1999). The dark gray patches correspond to high vegetation density, e.g., bushes and trees, and the white patches correspond to low vegetation density (grass or bare ground). The wavelength of the pattern is estimated to be about 70 m.

Apparent curiosity of this phenomenon stimulated several studies aimed at identifying the mechanism of the vegetation pattern formation and to explain its main properties, such as the typical wavelength of the pattern in the direction across the stripes. Attempts were made to relate it to the spatial distribution of precipitation (Greig-Smith, 1979), to soil heterogeneity (Beard, 1967), and to the impact of anthropogenic activity (Wickens and Collier, 1971). These approaches, however, do not seem to catch the essence of the phenomenon simply because tiger bush is a much more general phenomenon. Indeed, it is often observed in areas where none of the above factors apply.

The generality of vegetation stripes in semiarid regions gives us a reason to assume that the underlying mechanism is intrinsic for this ecosystem type, which means that it arises from some of its inherent processes or interactions. A common feature for arid and semiarid ecosystems is that water is the main
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limiting factor, which may lead to a hypothesis that pattern formation might be a result of the interaction between vegetation (consumer) and water (resource). The principal source of water is rainfall; but what is the mechanism of its redistribution on the land surface? To apply a Turing-type scenario, the two interacting agents, i.e., plants and water, must exhibit different mobility. While mobility/diffusivity of a plant population originates in seed dispersal, in the case of water the relevant factors are not immediately clear.

An answer to the above question can be found in a more careful analysis of field observations. It was concluded by several authors that vegetation stripes are more likely to be found on a slope rather than on flat ground. On a sloping surface, water flows downhill. Consequently, we arrive at the following model of a system with a differential advective flow:

\[
\begin{align*}
\frac{\partial U(X,Y,T)}{\partial T} &= R - \Upsilon U - C(U, V) + W \frac{\partial U}{\partial X} + D_1 \nabla^2 U , \\
\frac{\partial V(X,Y,T)}{\partial T} &= \kappa C(U, V) - M(V) + D_2 \nabla^2 V ,
\end{align*}
\]

where \(U\) is the amount of water per unit surface area, \(V\) is the vegetation biomass, \(R\) is the rate of water supply due to rains, \(W\) is the speed of the water flow, \(\Upsilon\) is the rate of water loss due to evaporation, function \(C\) describes water consumption by plants, \(\kappa\) is the consumption efficiency, \(M\) is mortality, and \(D_2\) is the plant diffusivity due to seed dispersal. The diffusive term in (9.89) takes into account the impact of the surface roughness on the water flow; the higher the roughness, the larger is \(D_1\).

We mention here that parameter \(R\) is not a momentary rate of water supply (due to the rainfall) but is a value averaged over a certain time interval covering at least one rain. Correspondingly, \(U\) also gives an average value of surface water density; otherwise, \(U\) would simply be zero for most of the time.

Apparently, the system (9.88)–(9.89) belongs to the type considered in the previous section and thus is capable of exhibiting pattern formation due to a Turing-type instability. The specific properties of the model depend on the choice of functions \(C\) and \(M\). As an illustrative example, cf. Klausmeier (1999), we assume that \(C = e U V^2\) and \(M(V) = M_0 V\), where \(e\) and \(M_0\) are coefficients. Introducing dimensionless variables as

\[
\begin{align*}
t &= \Upsilon T , \quad x = \left( \frac{\Upsilon}{D_2} \right)^{1/2} X , \quad y = \left( \frac{\Upsilon}{D_2} \right)^{1/2} Y , \\
u &= \left( \frac{e}{\Upsilon} \right)^{1/2} \kappa U , \quad v = \left( \frac{e}{\Upsilon} \right)^{1/2} V ,
\end{align*}
\]

from (9.88)–(9.89) we obtain

\[
\begin{align*}
\frac{\partial u(x,y,t)}{\partial t} &= r - u - u^2 + w \frac{\partial u}{\partial x} + e \nabla^2 u , \\
\frac{\partial v(x,y,t)}{\partial t} &= u v^2 - m v + \nabla^2 v ,
\end{align*}
\]

(9.90) (9.91)
where \( r = ke^{1/2}Y^{-3/2}R \), \( w = W(YD_2)^{-1/2} \), \( m = M_0/Y \), and \( \epsilon = D_1/D_2 \).

It should be mentioned here that the system (9.90)–(9.91) is mathematically identical to a chemical “substrate-autocatalyst” system, which has been studied extensively, e.g., see Rasmussen et al. (1996); Davidson (1998). Here we only recall as many of its properties as is necessary for the purposes of this section; more details can be obtained from the cited papers.

It is readily seen that the nonspatial system (9.90)–(9.91) may possess either one or three steady states. Specifically, there is a semi-trivial “no vegetation” state \((r, 0)\) and two nontrivial coexistence states \((u^-, v^-)\) and \((u^+, v^+), \) where

\[
v_{\pm} = \frac{1}{2m} \left( r \pm \sqrt{r^2 - 4m} \right), \quad u_{\pm} = \frac{m}{v_{\pm}}. \tag{9.92}\]

The semi-trivial steady state is feasible for any parameter value and is always linearly stable. The coexistence states exist only if \( m < r/2; \) for \( m \geq r/2, \) they merge and disappear in a saddle-node bifurcation. From the two coexistence states, the one with lower vegetation biomass, i.e., \((u^-, v^-)\), is always a saddle. As for \((u^+, v^+), \) it can be either a node or a focus and can be either stable or unstable. In particular, it is straightforward to see that, whatever the value of \( r, \) steady state \((u^+, v^+)\) is stable for sufficiently small \( m. \)

Moreover, Klausmeier (1999) showed that the steady state \((u^+, v^+)\) is feasible and stable for biologically reasonable parameter values. Therefore, what we have to do now is to reveal whether the corresponding spatially homogeneous steady state can be destabilized by a heterogeneous perturbation in a relevant parameter range and whether the properties of the emerging pattern are congenial with those observed in semiarid ecosystems.

Having applied the standard linearization technique, in the vicinity of \((u^+, v^+)\) the system (9.90)–(9.91) takes the following form:

\[
\frac{\partial u}{\partial t} = \left( -\frac{r v_+}{m} \right) u - 2mv + w \frac{\partial u}{\partial x} + \epsilon \nabla^2 u, \tag{9.93}\]
\[
\frac{\partial v}{\partial t} = \left( r v_+ - 1 \right) u + mv + \nabla^2 v. \tag{9.94}\]

Obviously, system (9.93)–(9.94) is exactly of the type that was considered in the previous section and thus all the results apply. In particular, instability onset is definitely to occur for sufficiently high speed of the flow and/or for sufficiently small diffusivity ratio.

Let us consider the case when the impact of diffusion can be neglected, which applies to plants with a short-range seed dispersal growing on a relatively steep slope with even ground surface. Then, the diffusion terms in...
Equations (9.93)–(9.94) are missing and a simple estimate of the typical spatial wavelength is given by (9.83), which now turns into

$$k_0 = \frac{1}{w} \left( \frac{rv_+}{m} - m \right) \left( \frac{rv_+ - 2m}{rv_+} \right)^{1/2}. \tag{9.95}$$

In order to calculate the corresponding wavelength of the pattern, we need to know the value of the ecological parameters. This is a subtle issue because the accuracy of ecological measurements is usually rather low. Moreover, parameter values can be of different orders of magnitude depending on whether the vegetation stripes consists of grass or bushes and trees, so that these two cases should be considered separately.

In the case of stripes of grass, using some typical parameter values according to Mauchamp et al. (1994) and Klausmeier (1999), one can obtain that $r = 1.9$ and $m = 0.45$. Consequently, it gives $v_+ \approx 4$ and $k_0 \approx 15.4 w^{-1}$, so that the wavelength of the pattern is

$$l_0 = \frac{2\pi}{k_0} \approx 0.4 w, \tag{9.96}$$

which, in dimensional units, gives

$$L_0 \approx 0.4 \left( \frac{W}{\Upsilon} \right). \tag{9.97}$$

For semiarid regions, the evaporation rate $\Upsilon$ is estimated to be 4 year$^{-1}$ (Klausmeier, 1999). On the other hand, $W$ apparently depends on the properties of a given landscape and can vary significantly. Using a tentative value $W = 200$ m-year$^{-1}$ we arrive at $L_0 \approx 20$ m, which agrees very well with the typical measured values of grass stripe widths being between 1 and 40 m.

Similarly, for stripes of bushes and trees, one can obtain $r = 0.15$ and $m = 0.05$, so that $v_+ \approx 2.6$ and the wavelength of the pattern is $l_0 = 0.95w$ or

$$L_0 \approx 0.95 \left( \frac{W}{\Upsilon} \right). \tag{9.98}$$

For the same values of $\Upsilon$ and $W$ as above, it gives $L_0 \approx 50$ m.

We emphasize that, since the chosen value of $W$ is purely hypothetical, the above figures by themselves should not be taken too seriously. What is encouraging, however, is that this approach predicts, at least qualitatively, a correct relation between the width of the grassy and bushy stripes. Indeed, field data show that a typical stripe width in case of trees/bushes is a few times larger than that of grass.

Moreover, let us recall that the pattern formation scenario due to differential flow instability predicts that the emerging patterns are of the traveling wave type rather than stationary. Remarkably, field observations show that the
vegetation stripes tend to move up the slope with approximately constant speed. Using Equation (9.84) in order to calculate the value of the traveling wave speed, we obtain that the stripes should move in the direction opposite to the water flow (i.e., up the slope) with the speed $v = 2.7 \text{ m-year}^{-1}$ for grass and with $v = 0.65 \text{ m-year}^{-1}$ for trees/bushes. Once again, although these values appear to be somewhat larger than those obtained in the field (which are 0.9 and 0.2, respectively), they give the correct predictions regarding the speed ratio.

In the case that diffusion cannot be neglected, an explicit relation for a typical wavenumber is not available and thus Equations (9.97) and (9.98) do not immediately apply. However, since the impact of diffusion tends to broaden the critical range of wavenumbers rather than shifting it (cf. Figure 9.7), and also taking into account the usual low accuracy of ecological data, these estimates are likely to remain valid, at least in the case when the diffusion coefficients are not very large.

### 9.3.1 Pattern formation due to nonlocal interactions

The above application of a conceptual advection–diffusion–reaction model to pattern formation in semiarid vegetation, also known as “tiger bush,” leads to apparently encouraging results giving a reasonable estimate for the width of the vegetation band and for the speed with which the pattern gradually travels up the slope. Moreover, it gives a correct prediction of how these values change between the cases when the vegetation stripes are formed by grass and trees/bushes, respectively.

However, a closer look at published data of field observations reveals that, although the model relating the patterns to a differential flow instability agrees very well with some of them, there are some aspects that remain beyond its catch:

- A few studies reported that, in some cases, the stripes can be oriented not across the slope (i.e., parallel the contour lines) but along the slope (MacFadyen, 1950b; White, 1969). While the stripes of the first type tend to travel uphill, the stripes of the second type are stationary.

- On a flat surface, the vegetation pattern normally consists of patches rather than stripes. The first naive ideas were to relate this patchiness either to the impact of a topographic “noise” or to an inherent heterogeneity of the soil. However, a later study (see Couteron and Lejeune, 2001) proved that the patchy pattern has a clear periodical structure, which makes those explanations irrelevant.

A question then arises whether an alternative approach could be possible where all these features would appear as a result of the same dynamical mechanism. It should be noted here that a diffusion–reaction system (the system from the previous section without water flow, i.e., when applied to flat
grounds) is still intrinsically capable of generating a spatiotemporal patchy structure; however, it takes place for parameter values inconsistent with those of a semiarid vegetation community (Klausmeier, 1999).

In order to outline what kind of a model it can be, let us have a somewhat deeper look at vegetation functioning. In an arid or semiarid region, the main controlling factor is the availability of water. An individual plant collects water from a certain area defined by its root system, so its success is apparently subject to competition with the neighboring plants. On the other hand, interaction with neighbors can have not only negative but also a positive effects: Obviously, the presence of other plants nearby results in a slower evaporation rate. A common sense supported also by biological data tells us that, in a situation where a single tree is likely to dry out and die, a group of trees has higher chances to survive.

Having based on these and other similar arguments, Lefever and Lejeune (1997) proposed the following model:

$$\frac{\partial U(R, T)}{\partial T} = \Lambda_1[U(R, T)] \cdot \Lambda_2[U(R, T)] - \Lambda_3[U(R, T)] ; \quad (9.99)$$

see also Lejeune et al. (1999); Lefever and Lejeune (2000); Couteron and Lejeune (2001). Here factors $\Lambda_1[U]$ and $\Lambda_2[U]$ account for all collective effects enhancing and hampering reproduction, respectively (where the term “reproduction” includes all processes and stages between the production of seeds and appearance of new seedlings), and the last term stands for the plants’ natural mortality.\footnote{Similar ideas, but in a somewhat more general context, have recently been further developed by Genieys et al. (2006).}

For the sake of simplicity, we assume that the natural mortality is a local process so that $\Lambda_3[U] \sim U$. On the contrary, along the lines of the above description, most the processes involved in reproduction have a clear spatial aspect and are essentially nonlocal. Therefore, $\Lambda_1[U]$ and $\Lambda_2[U]$ are functionals rather than functions, which we parameterize as follows:

$$\frac{\partial U(R, T)}{\partial T} = \left( \alpha \int_{-\infty}^{\infty} w_1(|R| - 1)^2 |U(R, T)| [1 + AU(R, T)] dR \right) \cdot \left( 1 - \frac{1}{K} \int_{-\infty}^{\infty} w_2(|R| - 1)^2 |U(R, T)| dR \right) \quad (9.100)$$

$$- MU ,$$

where $\alpha$ is the (linear) per capita growth rate, $M$ is the mortality rate, $K$ is the population carrying capacity in the limiting case $M \to 0$, and $A$ is a coefficient quantifying intensity of cooperative behavior. Due to their meanings, all the parameters are nonnegative. The dynamics takes place in a two-dimensional
space so that $\mathbf{R} = (X, Y)$. The kernels $w_i(|\mathbf{R} - \tilde{\mathbf{R}}|)$ describe how the intensity of the plant-plant interaction changes along with the interplant distance $|\mathbf{R} - \tilde{\mathbf{R}}|$. The kernels must be normalized, i.e.,

$$
\int_{-\infty}^{\infty} w_i(|\mathbf{R} - \tilde{\mathbf{R}}|) \, d\tilde{\mathbf{R}} = 1, \quad i = 1, 2,
$$

(9.101)

where integration is taken over the whole space.\footnote{In equations (9.100), (9.101), and further on, the integrals are, actually, multiple integrals due to the dimensionality of space.}

At first glance, it may seem that, unlike diffusion–reaction equations, (9.100) does not describe any motion. Indeed, interplant competition occurs due to the interplay between the root systems, which are static. Positive cooperative effect occurs due to mutual shadowing, which is static as well. It is not exactly true, however. The point is that, besides these processes, (9.100) describes seed dissemination. The rate \(\partial U(\mathbf{R}, T)/\partial T\) of the population growth at a given position $\mathbf{R}$ depends on the amount of seeds that land at this position. This is readily seen if we neglect competition, cooperation, and mortality (by setting $A = M = 0$ and $K \to \infty$) and keep only multiplication; Equation (9.100) then takes the form

$$
\frac{\partial U(\mathbf{R}, T)}{\partial T} = \alpha \int_{-\infty}^{\infty} w_1(|\mathbf{R} - \tilde{\mathbf{R}}|) U(\tilde{\mathbf{R}}, T) \, d\tilde{\mathbf{R}},
$$

(9.102)

where $\alpha$ is a product of the average number of seeds per adult plant and the survival rate, and the kernel $w_1$ gives the probability density of seed distribution (hence the normalization condition (9.101)). Equation (9.100) describes a diffusion-like processes; moreover, it can be shown that, on a certain spatial scale, it is actually reduced to a diffusion equation (cf. Petrovskii and Li, 2006).

Apparently, $w_i(|\mathbf{R} - \tilde{\mathbf{R}}|)$ should be a decreasing function of $|\mathbf{R} - \tilde{\mathbf{R}}|$ tending to zero for $|\mathbf{R} - \tilde{\mathbf{R}}| \to \infty$. The faster the decay in $w_i(|\mathbf{R} - \tilde{\mathbf{R}}|)$, the less prominent is the impact of nonlocality. In case $w_i(|\mathbf{R} - \tilde{\mathbf{R}}|) \sim \delta(\mathbf{R} - \tilde{\mathbf{R}})$, all processes are strictly local and (9.100) turns to a usual equation of nonspatial population dynamics:

$$
\frac{dU}{dt} = \alpha U(1 + AU) \left(1 - \frac{U}{K}\right) - MU.
$$

(9.103)

Due to condition (9.101), the steady states of nonspatial Equation (9.103) correspond to the spatially homogeneous steady states of Equation (9.100); therefore, it is convenient to start with the nonspatial case.

In order to study (9.103), we first introduce dimensionless variables as $t = \alpha T$ and $u = U/K$, so that (9.103) turns into

$$
\frac{du}{dt} = u(1 + au)(1 - u) - mu,
$$

(9.104)
where $\mu = M/\alpha$ and $a = AK$. Correspondingly, the steady states are given by

$$\bar{u} = u_0 = 0$$

and

$$\bar{u} = u_{\pm} = \frac{1}{2a} \left[ (a - 1) \pm \sqrt{(a - 1)^2 + 4a(1 - \mu)} \right].$$

(9.105)

While the no-vegetation state $u = 0$ exists for any parameter values, feasibility of the nontrivial states $u_+$ and $u_-$ depends on the relation between $a$ and $\mu$. For $0 < a \leq 1$, the only positive state is $u_+$, and it only exists if $0 < \mu < 1$. For $a > 1$, the parameter range of $u_+$ existence extends to $0 < \mu < \mu_{\text{max}} = (a + 1)^2/(4a)$. In the latter case, for $1 < \mu < \mu_{\text{max}}$, there also exists the other steady state $u_-$. It is readily seen, however, that the state $u = u_-$ is locally unstable and thus is not biologically relevant. The no-vegetation state $u = 0$ is stable for $1 < \mu < \mu_{\text{max}}$ and unstable for $0 < \mu < 1$. In its turn, the state $u = u_+$ is stable for all parameter values when it is feasible.

Coming back to the spatial model (9.100), our main goal is to check the stability of the homogeneous steady state $u = u_+$ with respect to heterogeneous perturbations and under what conditions the loss of stability, if any, can give rise to periodic spatial patterns. For that purpose, we now consider $u(x, t) = u_+ + \phi(x, t)$, where $\phi$ is small. From (9.100), after some standard transformations, we then obtain in the first order with respect to $\phi$:

$$\frac{\partial \phi(R, t)}{\partial t} = \int \left[ \gamma_1 w_1(|R - \tilde{R}|) - \gamma_2 w_2(|R - \tilde{R}|) \right] \phi(\tilde{R}, t) d\tilde{R} - \mu \phi(R, t),$$

(9.106)

where

$$\gamma_1 = (1 + 2a\bar{u})(1 - \bar{u}) \quad \text{and} \quad \gamma_2 = \bar{u}(1 + a\bar{u}).$$

(9.107)

An arbitrary heterogeneous perturbation can be expanded into a Fourier integral:

$$\phi(R, t) = \frac{1}{2\pi} \int \Phi(t, k) e^{ikR} dR.$$

(9.108)

Having substituted (9.108) to (9.106), we arrive at

$$\int \frac{d\Phi(t, k)}{dt} e^{ikR} dR = \int d\tilde{R} \left[ \gamma_1 w_1(|R - \tilde{R}|) - \gamma_2 w_2(|R - \tilde{R}|) \right] \cdot \int \Phi(t, k) e^{ikR} dR - \mu \int \Phi(t, k) e^{ikR} dR,$$

(9.109)
which is equivalent to
\[ \int \mathcal{H} e^{ikR} dk = 0, \quad (9.110) \]
where

\[ \mathcal{H} = \frac{d\Phi(t,k)}{dt} + \mu \Phi(t,k) \quad (9.111) \]

\[ - \Phi(t,k) \int d\tilde{R} \left[ \gamma_1 w_1(|R - \tilde{R}|) - \gamma_2 w_2(|R - \tilde{R}|) \right] e^{ik(\tilde{R} - R)}. \]

Apparently, in order to satisfy Equation (9.110), \( \mathcal{H} \) should be equal to zero identically. Therefore, we obtain that \( \Phi(t,k) \) should be a solution of the following equation:

\[ \frac{d\Phi(t,k)}{dt} = \omega(k) \Phi(t,k), \quad (9.112) \]
where

\[ \omega(k) = -\mu + \int \left[ \gamma_1 w_1(|S|) - \gamma_2 w_2(|S|) \right] e^{ikS} dS. \quad (9.113) \]

Correspondingly, for any particular \( k \), the solution of (9.112) is given by

\[ \Phi(t,k) = \Phi(0,k) e^{\omega(k)t}, \quad (9.114) \]
so that a small initial perturbation will decay if \( \omega(k) < 0 \) and increase if \( \omega(k) > 0 \). Obviously, in the former case the system is asymptotically stable with respect to an inhomogeneous perturbation with given \( k \); in the latter case it is unstable.

Equations (9.112) to (9.114) were obtained in a general case, i.e., without making any hypotheses about the functional form of \( w_1 \) and \( w_2 \) (except for their sufficiently fast decay to ensure the existence of the integrals). However, further analysis will not be instructive unless we make specific assumptions about the shape of the kernels. In the following, we assume that

\[ w_i(S) = \frac{1}{L_i^2 \pi} \exp \left[ -\left( \frac{S}{L_i} \right)^2 \right], \quad i = 1, 2, \quad (9.115) \]
where parameters \( L_1 \) and \( L_2 \) give, respectively, characteristic distances where the enhancing and hampering cooperative processes are effective.

From (9.113) and (9.115), we obtain

\[ \omega(k) = -\mu + (1 + 2au_+)(1 - u_+) \exp \left( -\frac{k^2 L_1^2}{4} \right) \]
\[ - u_+(1 + au_+) \exp \left( -\frac{k^2 L_2^2}{4} \right). \quad (9.116) \]
FIGURE 9.9: The stability diagram calculated according to (9.116) for (a) different values of $\nu = L_2/L_1$, curve 1 for $\nu = 1$, curve 2 for $\nu = 2$, and curve 3 for $\nu = 5$ (other parameters are $a = 0.8$, $\mu = 0.7$, $L_1 = 1$); and (b) for different values of $\mu$, curve 1 for $\mu = 0.1$, curve 2 for $\mu = 0.7$, and curve 3 for $\mu = 95$ (other parameters $a = 0.8$, $\nu = 5$, $L_1 = 1$).
Therefore, $\omega$ depends on $|k|$ rather than on $k$.

Expression (9.116) is rather difficult to study analytically; however, two things can be seen relatively easily. Both in the short-wave and long-wave limits $\omega(|k|)$ appears to be negative, $\omega(|k| \to \infty) \to -\mu < 0$, and $\omega(|k| \to 0) \to -[(a - 1)^2 + 4a(1 - \mu)]^{1/2} u_+ < 0$, respectively. Analysis of conditions when $\omega$ can be positive for intermediate values of $|k|$ leads to rather cumbersome and bulky equations, which we do not show here for the sake of brevity; details can be found in Lefever and Lejeune (1997). Briefly, function $\omega(|k|)$ appears to have a global maximum at a certain $|k| = k_c$ and $\omega_{\text{max}}$ is positive provided $\nu = L_2/L_1$ is large enough. Indeed, assuming $\nu \gg 1$, for a small positive $|k|$ we obtain

$$\omega(k) \simeq -\mu + (1 + 2au_+)(1 - u_+) \exp \left( -\frac{k^2L_1^2}{4} \right)$$

(9.117)

(taking into account that $\mu = (1 + au_+)(1 - u_+)$ and $0 < u_+ < 1$).

Note that the necessary condition $L_2/L_1 > 1$ of the instability onset is close in its meaning to the necessary condition $D_2/D_1 > 1$ of the Turing instability; cf. (9.16)–(9.18).

A typical behavior of $\omega(|k|)$ is shown in Figure 9.9. While dependence on $\omega_{\text{max}}$ on $L_2/L_1$ is relatively straightforward, its dependence on other parameters is somewhat more complicated. In particular, dependence of $\omega_{\text{max}}$ on $\mu$ is non-monotonic, so that its maximum value (and, correspondingly, the widest range of $k$ where $\omega(|k|) > 0$) is reached for an intermediate value of $\mu$; see Figure 9.9b.

These results prove that the model vegetation system described by Equation (9.100) is capable of generating periodic spatial patterns. It should be mentioned here that the above analysis addresses linear stability and, as such, is only valid when the perturbation of the original homogeneous steady state is small. Therefore, generally speaking, it does not tell much about the properties of the emerging patterns (except for their periodicity with the wavelength from a predicted range). Indeed, numerical simulations (Lefever and Lejeune, 1997; Lejeune et al., 1999) show that there can be two qualitatively different patterns arising in the large-time limit, namely, stripes and patches. Examples are shown in Figure 9.10.

Note that model (9.100) does not account for any spatial anisotropy. Therefore, it predicts that vegetation bands should not be strictly related to a sloping ground. Indeed, although on a flat surface a periodical patchy “spotted” pattern is more commonly observed, the tiger bush pattern has been occasionally observed as well (White, 1970). Remarkably, in the model (9.100), both pattern types arise as an outcome of the same dynamical mechanism. Orientation of the patterns was shown to be determined by the initial conditions (Lefever and Lejeune, 1997; Lejeune et al., 1999).
Now, the next step is to understand how the system properties may change under the presence of spatial anisotropy. For that purpose, we assume that the kernels $w_1$ and $w_2$ are translated or “shifted” by fixed distances $R_1$ and $R_2$, respectively. Equation (9.100) now takes the following form:

$$\frac{\partial U(R, T)}{\partial T} = \left( \alpha \int_{-\infty}^{\infty} w_1(|(R - R_1) - \hat{R}|)U(\hat{R}, T)[1 + AU(\hat{R}, T)]d\hat{R} \right)$$

$$\cdot \left( 1 - \frac{1}{K} \int_{-\infty}^{\infty} w_2(|(R - R_2) - \hat{R}|)U(\hat{R}, T)d\hat{R} \right) - MU.$$  

(9.118)

Generally speaking, in a real-world vegetation system there can be different sources of anisotropy, and they may act differently on different aspects of plants’ reproduction. For instance, competition for water would be affected by anisotropy produced by a sloping surface while anisotropy in seed dissemination is obviously subject to wind direction, which is largely independent on landscape details. For simplicity, here we assume that there is only one preferred direction so that vectors $R_1$ and $R_2$ are parallel to each other. Considering $R_1 = (0, R_1)$ and $R_2 = (0, R_2)$ and using the same parametrization (9.115) for the kernels, after some rather tedious calculations, from (9.119)
one can obtain the following expression for $\omega(k)$:

$$
\omega(k) = -\mu + (1 + 2au_+)(1 - u_+) \cos(R_1k_y) \exp \left(-\frac{k^2L_1^2}{4}\right)
- u_+(1 + au_+) \cos(R_2k_y) \exp \left(-\frac{k^2L_2^2}{4}\right)
+ i \left[(1 + 2au_+)(1 - u_+) \sin(R_1k_y) \exp \left(-\frac{k^2L_1^2}{4}\right)
- u_+(1 + au_+) \sin(R_2k_y) \exp \left(-\frac{k^2L_2^2}{4}\right)\right].
$$  (9.119)

Therefore, the expression for $\omega(k)$ now contains both real and imaginary parts. While stability is still fully determined by the sign of $\text{Re} \, \omega$, the homogeneous steady state being unstable to a perturbation with given $k$ if $\text{Re} \, \omega(k) > 0$, the existence of a nonzero imaginary part opens a possibility of traveling wave patterns.

The principal properties of $\omega(k)$ given by (9.119) are best seen if we consider separately two particular cases, i.e., (i) when anisotropy affects only multiplication/cooperation ($R_1 \neq 0$, $R_2 = 0$), and (ii) when anisotropy affects only competition ($R_1 = 0$, $R_2 \neq 0$). The results are sketched in Figure 9.11. In the former case, the two domains in the $(k_x, k_y)$ plane where $\text{Re} \, \omega(k) > 0$ are centered around the points $(\pm k_x^{(c)}, 0)$; in the latter case, they are situated around the points $(0, \pm k_y^{(c)})$, $k_x^{(c)}$ and $k_y^{(c)}$ being certain characteristic values.

Now, coming back to the semiarid vegetation patterns, we can associate the source of anisotropy with sloping ground so that the $y$-axis is directed along the gradient. This means that, in the cases (i) and (ii) (cf. left and right of Figure 9.11), the instability of the homogeneous state $u = u_+$ favors development of vegetation bands oriented down the slope and perpendicular to the slope, respectively. Thus, both stripe types appear as a result of essentially the same dynamical mechanism. Moreover, since in case (i) $k_y \approx 0$, $\text{Im} \, \omega(k) \approx 0$ as well, which means that the downslope stripes are largely stationary. In case (ii), however, $\text{Im} \, \omega(k) \neq 0$ and the vegetation stripes are traveling rather than stationary, which is in very good agreement with field observations.

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6Subject to additional conditions $|R_ik_y| < \pi/2$ ($i = 1, 2$) in order to avoid model artifacts; cf. Lefever and Lejeune (1997).
Instabilities and dissipative structures

FIGURE 9.11: A sketch of the stability diagram of the model (9.119) in the \((k_x, k_y)\) plane obtained for \(a = 1\) and \(\mu = 0.9\). Anisotropy is assumed to act in the \(y\) direction. Left-hand panel corresponds to the case when the anisotropy affects only multiplication/cooperation, \(R_1 = 0.5\), \(R_2 = 0\), and \(\nu = 5\). Right-hand panel corresponds to the case when the anisotropy affects only competition, \(R_1 = 0\), \(R_2 = 0.5\), and \(\nu = 3.33\). Grey color shows the instability domains where \(\omega > 0\). With permissions from Lefever and Lejeune (1997).

9.4 Concluding remarks

During the last two decades, considerable progress has been made in further developing the Turing ideas and in identification of instabilities in various diffusion–reaction systems; see, for instance, Rovinsky and Menzinger (1992); Kapral and Showalter (1995); Andresen et al. (1999); Satnoianu et al. (2000, 2001). Although a vast majority of the results have been originally obtained for chemical systems, which implies different types of nonlinearities in the equations, there is no doubt that at least some of them are relevant to population dynamics (Malchow, 1993, 1995, 2000a,b). In particular, the pattern formation scenario due to the Turing instability was hypothetically considered as the basic mechanism underlying the commonly observed heterogeneous spatial distribution of marine plankton, the phenomenon also known as “plankton patchiness” (see Levin and Segel, 1976).

It should be mentioned that specific requirements of the “Turing type” instability (i.e., instability of a locally stable equilibrium to a heterogeneous perturbation with wavelengths from a certain finite range) are apparently different for different systems. Particular examples of these requirements are given by the condition that the diffusion coefficient ratio should be sufficiently large (sufficiently small) compared to unity in case of diffusive instability, or
the speed of the differential flow should be large enough in case of advection-induced instability. There is one thing, however, that these seemingly different scenarios have in common: Onset of instability is not possible unless the interacting species or agents possess different "mobility," no matter whether the motion is random or ordered.

Another common feature, which stems from the very "definition" of the Turing-type instability, is that the emerging patterns are periodical in space. Meanwhile, although one real-world example of a spatially periodic ecological pattern was considered in the preceding section, in general, periodicity in the spatial distribution of ecological populations is rarely observed. In particular, the spatial distribution of plankton is remarkably irregular. Therefore, the generality of the Turing scenario as a generic mechanism for ecological pattern formation remains highly controversial.

In this chapter, we focused on pattern formation in purely deterministic systems. Concerning real-world applications, a big issue is how the scenarios and the properties of emerging patterns can be altered by the presence of noise. There is a lot of evidence that noise can make Turing patterns less regular (cf. Malchow et al., 2004). This issue will be considered in detail in Part IV. Meanwhile, the reported impact of noise does not answer the question of whether a deterministic diffusion–reaction system is intrinsically capable of generating irregular patterns. Throughout the next few chapters, we revisit some recently discovered non-Turing mechanisms of pattern formation in deterministic models of population dynamics that result in a remarkably irregular spatial structure, qualitatively (and sometimes quantitatively) similar to those observed in nature.

\footnote{Not to mention the fact that the diffusivity of the phytoplankton (prey) and zooplankton (predator) is essentially the same because it originates in the same turbulent mixing.}
Chapter 10

Patterns in the wake of invasion

Spatial distributions of ecological species are usually distinctly heterogeneous, but the main processes underlying these heterogeneities, as well as scenarios of ecological pattern formation, are often rather poorly understood. A conceptual model treating ecological patterns as the diffusion-induced “dissipative structures” in a system of interacting ecological species, such as a predator and its prey, was considered in the previous chapter. This approach, however, was shown to have severe limitations and, apart from a few specific cases, pattern formation due to a Turing-type diffusive instability can hardly be regarded as a common scenario.

A key to the understanding can probably be found in a historical perspective. Positions of ecological borders change with time (e.g., as a result of continuous climatic changes or, more recently, due to the impact of human activity) and the area inhabited by a given species a few thousands years ago might have been completely different from what it is today. Moreover, many of the species are known to be invaders and came from a distant land or even from another continent. These observations seem to give some reasons for making a hypothesis that the currently observed species distribution has a “historical dimension” and should be considered together with the corresponding processes of species spread, i.e., dispersal and/or migration. Note that, both in the case of moving ecological borders and in the case of invasion, species redistribution is associated with a traveling boundary. Therefore, in more mathematical terms the question can then be formulated as follows: Can it happen that spatiotemporal patterns are “triggered” by the propagation of a traveling population front, considering it as a basic scenario of biological invasion? Indeed, spatiotemporal population oscillations in the wake of invasion have been observed in some ecological data (Caughley, 1970; Jeltsch et al., 1992).

To the best of our knowledge, in the theoretical perspective, pattern formation caused by a propagating front was first discovered by Dunbar (1983), who observed promptly decaying oscillations behind the front. A somewhat more prominent effect was observed by Yachi et al. (1989) in numerical simulations in an ecoepidemic model describing the spread of rabies among a fox population. A few years later, the work was carried forward by Sherratt (1994a) and Sherratt et al. (1995), who showed that patterns arising in the wake can be chaotic. A curious phenomenon of “dynamical stabilization,” which results in the convergence of system’s trajectories to an unstable equilibrium and for-
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formation of a quasi-homogeneous species distribution in the wake of the front, was observed and studied in Petrovskii and Malchow (2000) and Malchow and Petrovskii (2002) using a bifurcation theory along with numerical simulations. Similar phenomena were also found in some chemical systems (Merkin et al., 1996; Merkin and Sadiq, 1996; Rasmussen et al., 1996; Davidson, 1998), which are mathematically similar to the models of population dynamics.

The chapter begins with an insight into a typical scenario of pattern formation in the wake of an invading predator, with special attention paid to the possibility of realistic irregular patterns. After that, we focus on the dynamical stabilization of an unstable equilibrium in the wake of invasion. Then we show that a similar succession of patterns can be observed in a system of competing species, thus indicating that the scenario is generic. Finally, we figure out how these results can be extended to ecoepidemic systems.

10.1 Invasion in a prey–predator system

The spatiotemporal dynamics of a prey–predator system is described by the following equations:

\[
\frac{\partial U}{\partial T} = D \nabla^2 U + P(U) - E(U, V), \quad (10.1)
\]

\[
\frac{\partial V}{\partial T} = D \nabla^2 V + \kappa E(U, V) - MV \quad (10.2)
\]

where all notations have the usual meanings, cf. Chapter 2.

Contrary to the previous chapter, here we deliberately focus on the case when diffusivity is the same for both species in order to exclude the possibility of the Turing instability; therefore, all patterns to be observed should be ascribed to a non-Turing mechanism(s). Note that the assumption of equal diffusivity is not at all ecologically unrealistic. One immediate example is given by a marine ecosystem where diffusion takes place mainly due to turbulent mixing (see Okubo, 1980), which has practically the same impact on prey (e.g., phytoplankton) and predator (zooplankton and/or fish larvae). In terrestrial ecosystems, a predator’s success is often reached more due to an optimal foraging strategy rather than due to a faster motion, and its diffusivity must not necessarily be higher than that of its prey.

For non-Turing pattern formation, effective analytical methods are yet to be developed, and results are usually obtained by means of computer simulations (but see Sherratt, 1994a). For that purpose, we need to decide about the specific functional form of prey growth and predation, i.e., about functions \(P(u)\) and \(E(u, v)\), respectively. Throughout this section, we assume that prey growth is logistic and predation is of Holling type II. Specifically, we use the
following parametrization:
\[ P(U) = -\frac{\alpha}{K} U(U - K), \]
where \( \alpha \) is the intrinsic growth rate, \( K \) is the carrying capacity, and
\[ E(U, V) = A \frac{UV}{U + H}, \]
where \( \gamma \) and \( H \) are the per capita predation rate and the half-saturation prey density, respectively.

We want to emphasize, however, that, actually, the scenarios of pattern formation described below are proved to be robust to the choice of parametrization as long as the main properties of the functions remain qualitatively the same. For instance, instead of the algebraic Michaelis–Menten function (10.4), predator response to prey density could be described by the exponential Ivlev function (Sherratt et al., 1995; Petrovskii et al., 1998) or by a ratio-dependent function (Sherratt et al., 1995) without any significant change in the system dynamics.

We begin with the one-dimensional case so that \( \nabla^2 = \frac{\partial^2}{\partial X^2} \). Introducing, for convenience, dimensionless variables as
\[ t = \alpha T, \quad x = X \left( \frac{\alpha}{D} \right)^{1/2}, \quad u = \frac{U}{K} \quad \text{and} \quad v = \frac{AV}{\alpha K} \]
(see Chapter 2 for more details), Equations (10.1)–(10.2) take the following forms:
\[ \frac{\partial u}{\partial t} = \frac{\partial^2 u}{\partial x^2} + u(1 - u) - \frac{uv}{u + h}, \]
\[ \frac{\partial v}{\partial t} = \frac{\partial^2 v}{\partial x^2} + k \frac{uv}{u + h} - mv, \]
where \( k = \kappa A/\alpha, \quad m = M/\alpha, \quad \text{and} \quad h = H/K \) are dimensionless parameters.

In agreement with ecological reality, we assume that the population system dwells on a finite domain of length \( L \) so that \(-L/2 < x < L/2\).

It seems reasonable to consider the corresponding nonspatial system first:
\[ u_t = u(1 - u) - \frac{uv}{u + h}, \quad v_t = k \frac{uv}{u + h} - mv. \]  

In a general case there is no one-to-one correspondence between the properties of the spatial and nonspatial systems, the dynamics of the spatial system being much richer. Yet it is clear that the nonspatial system provides a certain “skeleton” for understanding more complicated dynamics of its spatial counterpart because of the evident relation between the steady states of Equations (10.8) and the spatially homogeneous stationary solutions of system (10.6)–(10.7).
Basic investigation of the system (10.8) can be done by standard linear stability analysis. It is not difficult to see that there are only three stationary points in the phase plane \((u, v)\), i.e., the extinction state \((0, 0)\), the prey-only state \((1, 0)\), and the coexistence state \((\bar{u}, \bar{v})\) where

\[
\bar{u} = \frac{ph}{1-p}, \quad \bar{v} = (1 - \bar{u})(h + \bar{u})
\]  

(denoting, for convenience, \(p = m/k\)). The type of stationary point depends on the eigenvalues that are the solutions of the equation

\[
\lambda^2 - \lambda \text{tr}A + \text{det}A = 0, \quad (10.10)
\]

where \(A\) is, as usual, the matrix of the linearized system. Obviously, in order to trace changes in the stability of a given steady state, it is sufficient to reveal any change of sign in \(\text{tr}A, \text{det}A,\) and \(\Delta = (\text{tr}A)^2 - 4 \text{det}A\).

It is readily seen that \((0, 0)\) is a saddle for any value of the system parameters \(k, m,\) and \(h\). The stationary point \((1, 0)\) is a saddle for \(h < h_1(p) = 1 - \frac{p}{p}\) (see curve 1 in Figure 10.1), and only in this case is the coexistence state \((\bar{u}, \bar{v})\) situated in the biologically meaningful region \(u \geq 0, v \geq 0\); otherwise, \((1, 0)\) is a stable node.

For \((\bar{u}, \bar{v})\) we obtain

\[
\text{tr}A = \frac{p}{1-p} [(1 - h) - p(1 + h)] , \quad \text{det}A = kp[1 - p(1 + h)], \quad (10.12)
\]

so that the coexistence state changes its stability for

\[
h = h_2(p) = \frac{1 - p}{1 + p}; \quad (10.13)
\]

cf. curve 2 in Figure 10.1. For all parameter values when the state \((\bar{u}, \bar{v})\) is unstable, i.e., below curve 2, it is surrounded by a stable limit cycle that appears through the Hopf bifurcation.

Figure 10.1 shows a sketch of the map in the parameter plane \((p, h)\) for a hypothetical value \(k = 0.1\). Here curves 3 and 4 arise from the equation \(\Delta = 0\) and show where the coexistence steady state changes its type from node to focus (or vice versa). Therefore, domain I above curve 1 corresponds to the case of \((\bar{u}, \bar{v})\) being a saddle-point, where the only attractor in the phase plane \((u, v)\) for these parameter values is the stable node \((1, 0)\). Domain II, between curves 1 and 3, corresponds to \((\bar{u}, \bar{v})\) being a stable node and domain III between curves 2 and 3 to \((\bar{u}, \bar{v})\) being a stable focus. Domains IV (between curves 2 and 4) and V (below curve 4) correspond to \((\bar{u}, \bar{v})\) being an unstable
focus or unstable node, respectively, surrounded by a stable limit cycle that appears via Hopf bifurcation when crossing curve 2.

Note that, although Equations (10.6) and (10.7) contain three parameters, i.e., $k$, $m$, and $h$, $\text{tr}A$ actually depends on the ratio $p = m/k$ but not on $m$ and $k$ separately. As a result, it appears that variations in $k$ lead to only insignificant changes in the structure of the map in the $(p, h)$ plane. Indeed, curves 1 and 2 (defined by $h_1(p)$ and $h_2(p)$, respectively) are “universal” in the sense that their positions do not depend on $k$. As for curves 3 and 4, they show only a slight dependence on $k$ (for values $k \ll 1$, curves 3 and 4 approach curve 2, and for values $k \gg 1$ curve 3 gets close to curve 1 while curve 4 approaches axis $p$).

Having kept the properties of the nonspatial system in mind, now we proceed to the dynamics of the spatial system (10.6)–(10.7). However, firstly, we must complement the equations with boundary and initial conditions. Regarding the initial distribution of species, we consider an invasion of a predator into an area already inhabited by prey at the level of the carrying capacity:

$$u(x, 0) = 1 \text{ for any } x,$$

$$v(x, 0) = v_0 \text{ if } |x| < \frac{d_v}{2} \text{ and } v(x, 0) = 0 \text{ if } |x| > \frac{d_v}{2},$$

where $v_0$ is the predator density inside the initially invaded patch, $d_v$ being the patch diameter.
As for the boundary conditions, generally speaking, they should also be chosen according to the biological situation under study. For instance, a hostile environment outside of the domain would best be taken into account by setting the population densities at the boundary to zero, an “impermeable” boundary would be more adequately described by the no-flux conditions, etc. It should be noted, however, that, in case the time is not very large (so that the spreading predator population does not reach the domain boundary), the type of boundary condition does not really matter. In order to make it consistent with the initial distribution (10.14), the simulation results below are shown for the zero-flux conditions for both species.

Knowledge of the non-spatial dynamics seems to allow us to outline what the dynamics of the spatial system can be. For parameters from domain I we can expect that the predator goes extinct and there is no invasion. For parameters from domain II, we can expect the existence of a propagating front “switching” the system from the prey-only state \((1, 0)\) to the stable coexistence state \((\overline{u}, \overline{v})\). For parameters from domain III, we can probably expect some damped oscillations at the front. For domains IV and V, prediction is more difficult; due to the existence of the stable limit cycle, it seems reasonable to expect some sort of population oscillations.

These semi-intuitive predictions appear to be in a good agreement with results of computer experiments, at least for those parameter values when the coexistence state is stable. In particular, for domains I and II, it is predator extinction and invasion through propagation of monotonous population front, respectively.

For domain III, where the coexistence state is a stable focus, the situation becomes somewhat more interesting because the population front is no longer monotonous; see Figure 10.2. The papers by Dunbar should be mentioned here (Dunbar, 1983, 1984, 1986); he was the first to observe and study this phenomenon. The oscillations can be rather prominent at the front but decay promptly behind the front; on the whole, their amplitude becomes larger the closer the parameters are to curve 2, where the coexistence state \((\overline{u}, \overline{v})\) looses its stability. Note that the oscillations do not change their shape and the pattern simply moves as a whole with a constant speed.

Oscillations at the propagating population front form an interesting pattern; however, its relation to the phenomenon of ecological patchiness may seem somewhat doubtful. The first concern is that the area occupied by these oscillations is rather small is size; thus, this pattern alone can hardly account for the common spatial heterogeneity of ecological populations. Second, the pattern has an apparently regular structure and is essentially stationary in the sense that the shape and relative positions of the peaks do not change with time. These properties are clearly different from those typically observed in nature.

Nevertheless, we want to emphasize that population oscillations at the front are not a mathematical artifact. Note that, due to oscillations' fast decay, the pattern consists of a distinct peak at the boundary separating the area
FIGURE 10.2: Predator density versus space shown at $t = 100$ (curve 1), $t = 200$ (curve 2) and $t = 300$ (curve 3) for parameters $k = 2.0$, $p = 0.4$, $h = 0.6$; the arrow indicates the direction of the predator invasion. The initial distribution is given by (10.14)–(10.15) with $d_v = 200$ and $v_0 = 0.5$. Since the problem is symmetrical with respect to the origin, only half of the domain is shown. The distribution of prey density has qualitatively similar features up to the apparent difference that $u \approx 1$ on the right-hand side of the traveling front; an example is given by the dashed-and-dotted curve 4 showing the prey distribution at $t = 300$.

inhabited by the given species from an empty space and a slightly heterogeneous population distribution behind. This means that the population density reaches its maximum at the boundary rather than inside. Remarkably, spatial patterns with similar properties have been observed for some insect species (Harrison, 1997; Brodmann et al., 1997) and they were attributed to a diffusion–reaction mechanism (cf. Hastings et al., 1997), although somewhat different from considered here.

Up to the minor distinctions mentioned above, for both domains II and III, a spatially homogeneous distribution of the populations arises behind the front with the population densities $u = \bar{u}$ and $v = \bar{v}$. The dynamics of the system becomes significantly different in the domains where the coexistence steady state is unstable, i.e., in domains IV and V below curve 2. In this case, there are population oscillations at the front as well, however, they are now much more distinct; see the top of Figure 10.3. Also, their rate of decay is lower compared to the case when $(\bar{u}, \bar{v})$ is a stable focus. Note that, in spite of the complicated spatial structure, the area occupied by periodic oscillations
adjoining the front is a traveling wave, it moves as a whole with a constant speed and without changing its shape.
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FIGURE 10.4: Phase plane of local population densities at a fixed point $x = 300$ after onset of irregular spatiotemporal oscillations. The trajectory is shown for the time interval between $t = 4000$ and $t = 8000$; parameters are the same as in Figure 10.3, top.

For parameter values when the limit cycle grows in size considerably and approaches the saddle-point $(0, 0)$, the spatial period of oscillations may become so large that the whole succession looks more like an ensemble of moving separated “patches” rather than a single pattern, cf. bottom of Figure 10.3 between $x = 500$ and $x = 900$, although it still retains its regular structure.

Remarkably, the regular population oscillations with decaying amplitude are not the only pattern induced by the propagating front. At the wake of the traveling wave, there also arise oscillations of a different sort; see the left-hand side of the upper panel in Figure 10.3. They are apparently aperiodic both in space and time: Figure 10.4 shows the phase plane of the local population densities obtained at a fixed position after irregular oscillations have developed. A closer look shows that the oscillations are chaotic (Sherratt et al., 1995), the corresponding value of the dominant Lyapunov exponent being estimated as $\lambda_D \sim 0.01$ (Medvinsky et al., 2002). Eventually, after the traveling wave leaves the domain, they occupy the whole area. That regime of spatiotemporal chaos appears to be self-sustained: Large-time computer simulations do not reveal any qualitative changes in the system dynamics after the onset of chaos.

We emphasize that the system (10.6)–(10.7) does not contain any prescribed spatial structure because none of the parameters depend on space. Therefore, although these chaotic spatiotemporal population oscillations are triggered by
FIGURE 10.5: Densities of predator (solid curve) and prey (dashed-and-dotted curve) vs coordinate at $t = 400$ as obtained for parameters $k = 2.0$, $p = 0.3$, and $h = 0.35$ chosen from the region of dynamical stabilization. The initial conditions are given by (10.14)–(10.15) with $a = 200$ and $v_0 = 0.5$. Note that the plateau behind the oscillatory fronts corresponds to the unstable state $(\bar{u}, \bar{v})$.

The traveling front (and therefore may be linked, to some extent, to the initial conditions of special type – but see Chapter 11), they are self-organized in the sense that they are not induced by any sort of “environmental” heterogeneity.

Interestingly, for some parameter values, the onset of chaotic oscillations in the wake of the traveling population wave is preceded by a plateau emerging behind the strongly oscillating front; see Figure 10.5. The population densities inside this area of quasi-homogeneity correspond to those of the coexistence steady state, $u \approx \bar{u}$ and $v \approx \bar{v}$. This is a highly nontrivial result because, for these parameter values, the stationary point $(\bar{u}, \bar{v})$ is unstable in the corresponding nonspatial system. Thus, the “dynamical stabilization” of the unstable equilibrium is an essential spatiotemporal effect and cannot be reduced to the nonspatial dynamics. The plateau gradually grows with time and may reach a considerable length, sometimes nearly as large as the overall length of the domain; see Figure 10.6. Numerical simulations show that, in an unbounded domain, the whole structure would persist over an indefinitely long time, which indicates that the dynamical stabilization of the unstable equilibrium in the wake of invasion is not a transient regime but rather an “intermediate asymptotics” (Barenblatt and Zeldovich, 1971; Barenblatt, 1996). In the next section we provide a more detailed analysis of this phenomenon.
FIGURE 10.6: The density of predator (solid curve) and prey (dashed-and-dotted curve) calculated at $t = 350$ (top) and $t = 1400$ (bottom) for parameters $k = 0.5$, $p = 0.2$, and $h = 0.6$ and the initial conditions the same as above. The region of homogeneity in the middle corresponds to the locally unstable coexistence state.

It should also be mentioned that, in some cases, between the unstable plateau and the chaotic region there can be a region of regular spatiotemporal oscillations periodical in space and time (e.g., see the bottom of Figure 10.6).
Regular oscillations in the wake of invasion were studied in much detail by Sherratt (1994b, 1998); in this book, we will not specifically focus on this pattern for the following reasons. Regular periodical waves can be stable or unstable per se (Kopell and Howard, 1973; Sherratt, 1998); however, there is substantial numerical evidence that they are not persistent in the sense that they are finally displaced by the chaotic oscillations. Moreover, although the regular periodical oscillations can indeed be generated by the initial conditions of special type (e.g., finite), they are rarely observed in a more general case; see Chapter 11.

For parameters from domain V, the dynamics of the system is rather similar to that in domain IV. However, there is one difference: No dynamical stabilization of the unstable node $(\bar{u}, \bar{v})$ has been observed. The region of irregular oscillations begins right behind the front of the predator wave.

Now, our concern is to reveal to what extent the above results (in particular, the typical succession of patterns shown in Figure 10.5) can be extended to the two-dimensional case, i.e., to the system given by Equations (10.6)–(10.7) where $u = u(x, y, t), v = v(x, y, t)$, and $\partial^2/\partial x^2$ is now changed to $\partial^2/\partial x^2 + \partial^2/\partial y^2$.

It should be mentioned here that numerical solution of the two-dimensional system brings considerable difficulty. The matter is that, as it is readily seen from the system dynamics in the one-dimensional case, the problem has a few distinctly different spatial and time scales. In particular, onset of chaos is a slow process and it does not happen until all preceding patterns emerge, such as oscillations at the invasion front and dynamical stabilization in the wake. However, the speed of the invasion front is relatively large and, by the time the chaotic oscillations develop, the front would propagate a significant distance. Correspondingly, the numerical domain must be large enough in order to avoid the impact of the boundary. As a result, a relevant domain size appears to be, in dimensionless units (10.5), on the order of $10^3$. On the other hand, the step-size $\Delta x$ of the numerical grid has to be sufficiently small in order to provide resolution of spatial heterogeneity on a smaller scale and to ensure sufficient approximation. The smallest spatial scale is given by the size of a single hump in the population density, which is typically on the order of 10; therefore, $\Delta x$ should normally be less than 1. That leads to a one-dimensional numerical grid with one thousand nodes, or even larger. In the corresponding two-dimensional system, it results in a grid as large as $10^6$ nodes. Although simulations on a grid as large as that are, in principle, within the capability of modern PCs, a single simulation run (i.e., for a given parameter set) takes many hours, sometimes a few days. That makes a detailed study virtually impossible. Thus, the results shown here should be regarded as illustrative rather than exhaustive.

Some snapshots of the system dynamics are shown in Figure 10.7, which is obtained for the same parameters as Figure 10.5. The initial conditions describe a spatially homogeneous distribution of prey at the carrying capacity, $u(x, y, 0) = 1$ for any $(x, y)$, and a finite distribution of predator inside a square.
domain centered around the origin, \( v(x, y, 0) = v_0 = 0.2 \) for \(-10 < x, y < 10\) and \( v(x, y, 0) = 0 \) otherwise. Since the system apparently exhibits symmetry with regards to reflections \( x \rightarrow -x, \ y \rightarrow -y \), the simulations are actually made only in the first quadrant of the system, imposing zero-flux conditions at the artificial boundaries \( x = 0 \) and \( y = 0 \).

\[
\begin{align*}
v(x, y, 0) &= 0.2 \quad \text{for} \quad -10 < x, y < 10 \\
v(x, y, 0) &= 0 \quad \text{otherwise.} 
\end{align*}
\]

FIGURE 10.7: (See color insert.) Spatial distribution of the predator density in the two-dimensional case for (a) \( t = 125 \), (b) \( t = 275 \), (c) \( t = 1000 \), and (d) \( t = 3500 \). Parameters are the same as in Figure 10.5. Prey density shows qualitatively similar behavior up to the apparent difference that in front of the leading edge prey is at its carrying capacity.

It appears that the dynamics of the two-dimensional system follows more or less the same scenario as the corresponding one-dimensional system. At an early stage (see Figure 10.7a), a traveling population front is formed with decaying population oscillations in the wake. It is followed by dynamical stabilization of the unstable coexistence state so that the population densi-
ties converge to $\bar{u}$ and $\bar{v}$, respectively, and a (quasi-)homogeneous plateau is formed; cf. the area near the left-hand bottom corner of Figure 10.7a. As the leading front propagates further, the unstable plateau grows in size considerably (see Figure 10.7b); however, eventually it gives way to a periodical wavetrain. At the wake of the wavetrain, chaotic spatiotemporal oscillations start developing gradually (see the left-hand bottom corner of Figure 10.7c) and eventually occupy the whole domain. Chaotic oscillations are self-sustained and persistent, and, after the onset of chaos all over the domain, there are no other qualitative changes in the system dynamics.

Qualitatively, the emerging chaotic patterns (see Figure 10.7d) look surprisingly similar to those usually observed in spatial distributions of real ecological populations (for instance, in plankton distribution in the ocean; e.g., see Figure 11.13 in the next chapter). Therefore, a hypothesis can be made that it may be the prey–predator interaction that shapes, at least to a certain extent, the “geometry” of the ecological patchiness and the properties of the system’s spatiotemporal dynamics in general. Indeed, prey–predator interactions are among the most common ones in ecosystems. Moreover, there is a widely accepted point of view that it is the prey–predator interaction(s) that are integrating separate species into a community by means of creating the mass flows along the trophic chain or web.

However, a visual resemblance between the patterns is not sufficient yet. In order to demonstrate that the patterns observed above are relevant to population dynamics in nature, at least, one has to show that pattern formation takes place on an appropriate spatial scale (recall that all the results have been obtained in dimensionless units). Also, the generality of the proposed scenario when patterns are generated by a propagating population front is disputable. Although a quick estimate of a typical patch size can be done relatively easy (based on the definition (10.5) of the dimensionless variables), we prefer to postpone detailed consideration of this issue until Chapter 11, where some more general scenarios of pattern formation in a conceptual prey–predator system will be revealed.

### 10.2 Dynamical stabilization of an unstable equilibrium

It is well known that a steady state that is stable in a nonspatial system may become unstable in the corresponding spatial system. Then, after the homogeneity is broken due to linear Turing instability (see Chapter 9), the nonlinear interactions between the components drive the system into the formation of standing spatial patterns (Nicolis and Prigogine, 1977). This is an irreversible process, i.e., the broken homogeneity is never restored unless the parameters of the system are changed considerably in order to violate the
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 instability conditions.

Yet a kind of inverse process – in a somewhat more general sense – may occur. Namely, for some parameter values an “anti-Turing” phenomenon takes place: A locally unstable equilibrium of the system (10.8) can be made dynamically stable in the full diffusion–reaction system (10.6)–(10.7). In this case, for certain time- and length scales, formation of spatial patterns is suppressed and homogeneity is restored.

In the previous section we showed that, when the coexistence steady state \((\bar{u}, \bar{v})\) turns unstable and is surrounded by a stable limit cycle, the diffusive front may still switch the system to the state \(u \equiv \bar{u}, \ v \equiv \bar{v}\). A typical wave profile is presented in Figure 10.5. One can see that, after prominent oscillations at the front of the wave, there comes the region where the concentrations \(u(x,t)\) and \(v(x,t)\) nearly reach their stationary (but unstable!) values \(\bar{u}, \ \bar{v}\). Note that this curious phenomenon is not exotic in the sense that it can be observed for a wide range of parameter values (cf. Figures 10.5 and 10.6), and it takes place both in one and two spatial dimensions; see Figure 10.7. The unstable plateau exists for remarkably long time before it is finally displaced by the irregular spatiotemporal oscillations. Moreover, as it is readily seen from comparison between the top and bottom of Figure 10.6 (cf. also Figures 10.7a and 10.7b), the length of the plateau can grow with time.

A question remains as to the intrinsic properties of the dynamical system (10.1)–(10.2), or its particular case (10.6)–(10.7), that make stabilization of the locally unstable coexistence state possible. A related and somewhat more practical question is how to distinguish between the parameters when dynamical stabilization can and cannot occur. Below we address these issues by using two different approaches.

10.2.1 A bifurcation approach

The idea of the approach is rooted in the observation that the oscillating front preceding the formation of the unstable plateau propagates as a stationary traveling wave. It means that, by the corresponding change of variables, the original system of partial differential equations, cf. (10.6)–(10.7), can be reduced to a system of ordinary differential equations. A required traveling front solution connecting the prey-only state \((1, 0)\) in front of the front to the coexistence state \((\bar{u}, \bar{v})\) behind the front can then be regarded as a heteroclinic connection between the corresponding steady state in the phase space of the system; e.g. see Dunbar (1984).

Since there are parameter values where dynamical stabilization is observed and there are ones where it is not, it means that this heteroclinic connection appears as a result of a global bifurcation. The problem of distinguishing between the parameters can then be addressed by using methods of bifurcation theory (Kuznetsov, 1995). However, global bifurcations are difficult to study, and effective mathematical tools are not fully developed yet. Instead, we endeavor to relate the emerging heteroclinic trajectory to a local bifurcation.
in one of the two steady states. In particular, we are going to show that there is a parameter range where the type/stability of the coexistence state \((\bar{u}, \bar{v})\) makes the existence of the heteroclinic connection impossible while outside of that range it cannot be ruled out.

Let us consider a stationary traveling wave propagating with a certain speed \(c_v\) and “switching” the system from the state \(u = 1, v = 0\) to the state \(u = \bar{u}, v = \bar{v}\). A relevant solution of the system (10.6)–(10.7) has the form \(u(x, t) = u(\xi), v(x, t) = v(\xi)\), where \(\xi = x - c_v t\). Then, system (10.6)–(10.7) is reduced to

\[
\frac{d^2 u}{d\xi^2} + c_v \frac{du}{d\xi} + u(1-u) - \frac{u}{u+h} = 0 ,
\]

\[
\frac{d^2 v}{d\xi^2} + c_v \frac{dv}{d\xi} + k \frac{u}{u+h} v - mv = 0 ,
\]

which, having introduced auxiliary variables \(p\) and \(q\), turns into a system of four ordinary differential equations:

\[
\frac{dp}{d\xi} = -c_v p + u(1-u) - \frac{u}{u+h} ,
\]

\[
\frac{du}{d\xi} = -p ,
\]

\[
\frac{dq}{d\xi} = -c_v q + k \frac{u}{u+h} - mv ,
\]

\[
\frac{dv}{d\xi} = -q ,
\]

where the dot above a letter means the ordinary derivative with respect to \(\xi\). Obviously, each homogeneous stationary state \((\bar{u}, \bar{v})\) of system (10.6)–(10.7) corresponds to a stationary point \((0, \bar{u}, 0, \bar{v})\) in a four-dimensional phase space \((p, u, q, v)\) of system (10.18)–(10.21).

Propagation of the front means that, while \(\xi\) changes from \(+\infty\) to \(-\infty\), the corresponding trajectory in the phase space leaves the state \((0, 1, 0, 0)\) and eventually enters (in the limit \(\xi \to -\infty\)) the state \((0, \bar{u}, 0, \bar{v})\). It means that there must exist at least one trajectory entering \((0, \bar{u}, 0, \bar{v})\). In its turn, it means that, for the system (10.18)–(10.21) linearized in vicinity of \((0, \bar{u}, 0, \bar{v})\), there must exist at least one eigenvalue with Re\(\lambda > 0\). (Note that, since \(\xi\) is changing from \(+\infty\) to \(-\infty\) along the traveling wave profile, the stability of \((0, \bar{u}, 0, \bar{v})\) is associated with eigenvalues with positive real parts.)

It is readily seen that the eigenvalues \(\lambda_{1-4}\) of the system (10.18)–(10.21) linearized in the vicinity of a given equilibrium point are solutions of the following equation:

\[
z^2 + (\text{tr} A) z + \det A = 0 , \quad z = \lambda(c_v + \lambda) ,
\]

where \(A\) is the matrix of the linearized nonspatial system (10.8). In particular, for the steady state \((0, \bar{u}, 0, \bar{v})\) the eigenvalues are given by the following
expression:

\[ \lambda_{1-4} = -0.5 \left( c_v \pm \sqrt{(c_v^2 - 2\text{tr} \bar{A}) \pm i 2\sqrt{|\Delta|}} \right) , \]  

(10.23)

where \( \Delta = (\text{tr} \bar{A})^2 - 4 \det \bar{A} \) and \( \bar{A} \) is the corresponding matrix. Since dynamical stabilization has been observed in numerical simulations only for \((\bar{u}, \bar{v})\) being an unstable focus, here we restrict our consideration to the case \( \Delta < 0 \).

From (10.23) we obtain

\[ \text{Re} \lambda = -0.5 (c_v \pm \omega) , \]  

(10.24)

where

\[ \omega = \sqrt{(c_v^2 - 2\text{tr} \bar{A})^2 + 4|\Delta|} \cos \left( 0.5 \arctan \frac{2\sqrt{|\Delta|}}{c_v^2 - 2\text{tr} \bar{A}} \right) . \]  

(10.25)

Now, if \( |\omega| < c_v \), all four eigenvalues have negative real parts. In this case, there can be no trajectory approaching the equilibrium point \((0, \bar{u}, 0, \bar{v})\) in the \( \xi \to -\infty \) limit. However, for \( |\omega| > c_v \), there are two eigenvalues with positive and two eigenvalues with negative real parts, and in this case such a trajectory can exist. Therefore, the heteroclinic connection and the corresponding change in the dynamical behavior of the system may emerge at the following critical relation:

\[ |\omega| = c_v . \]  

(10.26)

We emphasize that relation (10.26) is obtained without any reference to the specific parametrization used in the systems (10.6)–(10.7) or (10.18)–(10.21). Therefore, one can expect that it remains valid for a more general class given by Equations (10.1)–(10.2), at least in the case that functions \( E(u, v) \) and \( P(u) \) have properties qualitatively similar to the Michaelis–Menten kinetics and the logistic growth, respectively. For any given parametrization of \( E(u, v) \) and \( P(u) \), relation \( |\omega| > c_v \) gives a necessary condition of dynamical stabilization in the wake of the propagating front.

Taking into account (10.25), after some standard although tedious calculations, relation (10.26) takes a somewhat more specific form:

\[ \frac{4 \det \bar{A} - (\text{tr} \bar{A})^2}{\text{tr} \bar{A}} = 2c_v^2 . \]  

(10.27)

Note that an exact expression for the speed of the predator wave \( c_v \) is not available. However, there are many indications that the speed of the wave usually coincide with its minimum possible value (Murray, 1989), even if it is not always the case (Hosono, 1998).

The minimum speed value can be found by considering the solution properties in the vicinity of the steady state \((0, 1, 0, 0)\). Since \( u \) and \( v \) are population
densities, they must be nonnegative. This means that the solution cannot be winding around \((0, 1, 0, 0)\). In its turn, it means that the eigenvalues of the linearized system may not be complex.

From equations (10.22) we obtain

\[ \lambda = -0.5 \left[ c_v \pm \left( c_v^2 + 4z \right)^{1/2} \right] \tag{10.28} \]

where

\[ z = -0.5 \left[ \text{tr} A_1 \pm \left( \left( \text{tr} A_1 \right)^2 - 4 \det A_1 \right)^{1/2} \right] \tag{10.29} \]

and \( A_1 \) is the matrix of system \((10.8)\) linearized in the vicinity of \((1, 0)\). Since \((1, 0)\) is a saddle-point when the equilibrium \((\bar{u}, \bar{v})\) is situated inside the biologically meaningful region \(u \geq 0, v \geq 0\), it holds \( \det A_1 < 0 \) and, therefore, \( z_1 < 0 < z_2 \). Thus, all the solutions of (10.28) are real if and only if

\[ c_v^2 + 4z_1 \geq 0 \tag{10.30} \]

where \( z_1 \) corresponds to plus in Equation (10.29).

From (10.30), we obtain the lower bound for the spectrum of the possible speed values:

\[ c_v \geq c_v^{\text{min}} = \left[ 2 \left( \text{tr} A_1 + \left( \left( \text{tr} A_1 \right)^2 - 4 \det A_1 \right)^{1/2} \right) \right]^{1/2} \tag{10.31} \]

(supposing that the wave propagates along axis \(x\), i.e., \( c_v > 0 \)).

Having assumed \( c_v = c_v^{\text{min}} \), from (10.27) taken together with (10.31) and (10.12), we obtain an algebraic equation giving the critical relation between the problem parameters. Although the equation is rather bulky and cumbersome (we do not show it here for the sake of brevity), and its explicit analytical solution is hardly possible, it can be easily solved numerically. For each value of \( p \) and \( k \), it then gives the critical value of \( h \). For a specific case \( k = 0.1 \), the results are shown in Figure 10.8 by curve 5. For the domain between curves 2 and 5, one can expect dynamical stabilization of the unstable equilibrium \((\bar{u}, \bar{v})\) in the wake of the propagating oscillating front, i.e., formation of a quasi-homogeneous unstable plateau with \( u \approx \bar{u} \) and \( v \approx \bar{v} \), which separates the invading front from the region of irregular spatiotemporal oscillations.

Note that the critical curve is situated in the parameter domain where \((\bar{u}, \bar{v})\) is an unstable focus. This is in agreement with the observation made from numerical experiments that dynamical stabilization in the system (10.6)–(10.7) is not observed if \((\bar{u}, \bar{v})\) is an unstable node.

It should be recalled here that the critical relation obtained in this way is not a criterion but rather a necessary condition; cf. the lines preceding Equation (10.26). A thorough mathematical study would have to prove that the trajectory entering \((0, \bar{u}, 0, \bar{v})\) actually started at \((0, 1, 0, 0)\). Moreover, strictly speaking, it is not an exact necessary condition either because of the
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FIGURE 10.8: Analysis of dynamical stabilization – a map in the parameter plane of the system (10.6)–(10.7). Meanings of curves 1 to 4 are the same as in Figure 10.1. Curve 5 gives the critical relation (10.27) so that dynamical stabilization is only possible for parameters between curves 5 and 2. Curves 1 to 5 are obtained for \( k = 0.1 \). Curves 6 and 7 show the critical relation (10.46) obtained for \( k = 0.1 \) and \( k = 1.2 \), respectively.

above-made assumption \( c_v = c_v^{\text{min}} \). Surprisingly, for values of \( p \) not too close to 1 (see the next section for details), the obtained critical relation is in very good agreement with numerical experiments, so that curve 5 in Figure 10.8 indeed coincides with a boundary of the parameter domain where dynamical stabilization is observed (Petrovskii and Malchow, 2000).

The position of the parameter domain in the \((p, h)\) plane where dynamical stabilization takes place helps to put this phenomenon into a more general context of the global bifurcation structure of the system. Let us recall that a traveling wave with similar properties (decaying oscillations at the front with the population densities converging to the homogeneous state \( u(x, t) \equiv \bar{u}, \ v(x, t) \equiv \bar{v} \); e.g. see Figure 10.2) is also observed when \( (\bar{u}, \bar{v}) \) is stable, i.e., for parameters from above curve 2 in Figure 10.8. Existence of the corresponding heteroclinic trajectory connecting \((0, 1, 0, 0)\) to \((0, \bar{u}, 0, \bar{v})\) was proved rigorously by Dunbar (1984). Since the dynamical stabilization domain is the one adjoining curve 2 from below, it becomes obvious that the traveling front with dynamical stabilization in the wake corresponds to the same trajectory. The coexistence state looses stability through the Hopf bifurcation when crossing curve 2 from above; however, the stability loss does not destroy the heteroclinic connection.
10.2.2 Comparison of wave speeds

The analysis done in the previous section, although providing a condition of dynamical stabilization, does not allow us to obtain any estimate regarding the plateau length. In this section, we endeavor to address the phenomenon of dynamical stabilization in a more heuristic way. We will show that some additional restrictions on parameter values, as well as an equation describing the plateau growth, may appear quite naturally by comparing the speed of relevant diffusive fronts.

The idea of the approach is as follows. We have already shown that stabilization of a locally unstable steady state occurs in the wake of a stationary traveling diffusive front propagating with a certain (constant) speed $c$. Furthermore, if $(\bar{u}, \bar{v})$ is unstable, the propagation of the diffusive fronts (no matter with or without dynamical stabilization in the wake) is normally followed by the onset of irregular spatiotemporal population oscillations. In the case that dynamical stabilization occurs, a remarkable thing is that there exists a distinct interface separating the unstable plateau from the region occupied by the oscillations; cf. Figures 10.5 and 10.6. A closer look (Petrovskii and Malchow, 2001b) shows that the interface propagates with a constant speed, which we denote as $w$.

Now, the spatial domain where dynamical stabilization takes place is bounded by two moving boundaries: the leading front propagating with a constant speed $c$ and the interface between the plateau and the region of irregular spatiotemporal oscillations propagating with a speed $w$. Therefore, the process is controlled by the relation between $c$ and $w$. In case $w < c$, obviously, the length of the domain grows with time as $(c - w)t$. Since the leading front behaves as a stationary traveling wave, its shape and “width” (i.e., the size of the region occupied by the regular damped oscillations) do not change with time. Correspondingly, an increase in the length of the domain locked between the two moving boundaries is only possible by means of an increase in the length of the unstable plateau. Thus, we obtain that

$$L_{\text{plateau}} = (c - w)t + L_0,$$  

(10.32)

where $L_0$ is a constant.

In the opposite case, $w > c$, dynamical stabilization apparently cannot occur. The length of the plateau, even if it happens to appear as a result of specific initial conditions, should decrease with time until the region of irregular spatiotemporal oscillations would start immediately after the stationary traveling front. The embryo of the unstable plateau, if any, does not grow with time.

Thus, a simple necessary condition for the dynamical stabilization is $w < c$, so that the critical relation is given by

$$w = c.$$  

(10.33)
As it stands, however, relation (10.33) is not of much use because the actual values of speed are not known. The assumption that we are going to make at this point is that the diffusive fronts propagate with their minimum possible speed. Indeed, that is what usually happens in various diffusion–reaction systems (Murray, 1989), although a complete and thorough understanding of the reasons is still missing.

The value $c_{\text{min}}$ is given by Equation (10.31). Now we are going to obtain $w_{\text{min}}$. For that purpose, we consider the system far in front of the interface where the dynamics of the population densities $u(x,t)$ and $v(x,t)$ is described by small perturbations to the steady state $u = \bar{u}$, $v = \bar{v}$:

$$u(x,t) = \bar{u} + \Phi(x,t), \quad v(x,t) = \bar{v} + \Psi(x,t),$$

(10.34)

so that $\Phi$ and $\Psi$ are the solutions of the linearized system

$$\frac{\partial \Phi}{\partial t} = \frac{\partial^2 \Phi}{\partial x^2} + a_{11}\Phi + a_{12}\Psi,$$

(10.35)

$$\frac{\partial \Psi}{\partial t} = \frac{\partial^2 \Psi}{\partial x^2} + a_{21}\Phi + a_{22}\Psi,$$

(10.36)

where $a_{ij}$ are the elements of the linearized system matrix.

Since an exponential function is a generic solution of a system of linear differential equations, and $\Phi$ and $\Psi$ must be, asymptotically, of the same order of magnitude, we look for a solution of (10.35)–(10.36) in the following form:

$$\Phi(x,t) = p(t)e^{-\nu x}, \quad \Psi(x,t) = q(t)e^{-\nu x},$$

(10.37)

where functions $p(t)$ and $q(t)$ are to be determined and $\nu$ is a coefficient.

Having substituted (10.37) into (10.35)–(10.36), we obtain the equations for $p(t)$ and $q(t)$:

$$\frac{dp(t)}{dt} = (\nu^2 + a_{11})p + a_{12}q, \quad \frac{dq(t)}{dt} = a_{21}p + (\nu^2 + a_{22})q.$$  

(10.38)

The general solution of the linear system (10.38) is

$$p(t) = B_1e^{\omega_1 t} + B_2e^{\omega_2 t}, \quad q(t) = C_1e^{\omega_1 t} + C_2e^{\omega_2 t},$$

(10.39)

where $B_1$, $B_2$, $C_1$, $C_2$ are constant coefficients and the exponents $\omega_{1,2}$ are the solutions of the following equation:

$$(a_{11} + \nu^2 - \omega)(a_{22} + \nu^2 - \omega) - a_{12}a_{21} = 0.$$  

(10.40)

Hence,

$$\omega_{1,2} = \left(\nu^2 + \frac{\text{tr}A}{2}\right) \pm \frac{1}{2} \Delta^{1/2},$$  

(10.41)
where $\Delta = (\text{tr} \bar{A})^2 - 4 \det \bar{A}$ and $\bar{A}$ is the matrix of the linearized system.

For simplicity, we restrict further consideration to the case of $(\bar{u}, \bar{v})$ being an unstable focus. Respectively, $\Delta < 0$ and, considering (10.37) and (10.39) together, it is readily seen that the speed of the front is determined by the real part of the exponents. Since $\text{Re } \omega = \nu^2 + (\text{tr} \bar{A})/2$, Equations (10.37) take the form

$$\Phi(x, t) = \tilde{p}(t) \exp \left( -\nu \left[ x - \left( \nu + \frac{\text{tr} \bar{A}}{2\nu} \right) t \right] \right),$$

(10.42)

$$\Psi(x, t) = \tilde{q}(t) \exp \left( -\nu \left[ x - \left( \nu + \frac{\text{tr} \bar{A}}{2\nu} \right) t \right] \right),$$

(10.43)

where the functions $\tilde{p}(t)$ and $\tilde{q}(t)$ are periodical. Therefore, the speed of the front is given by

$$w = \nu + \frac{\text{tr} \bar{A}}{2\nu}.$$  

(10.44)

The spectrum of possible values of the speed depends on the unknown parameter $\nu$. However, the spectrum (10.44) has a lower bound, which corresponds to $\nu_0 = [(\text{tr} \bar{A})/2]^{1/2}$. Thus, the expression for the lower bound is

$$w_{\min} = (2 \text{ tr} \bar{A})^{1/2}.$$  

(10.45)

Note that (10.45) is found analytically without any additional assumptions and without any reference to the particular parametrization of the trophic responses in Equations (10.6)–(10.7)).

Having an expression for $w_{\min}$ at hand, now we can return to the critical relation (10.33). Setting $c = c_{\min}$ and $w = w_{\min}$ and making use of (10.45) and (10.31), after a little algebra from (10.33) we finally obtain

$$\frac{p}{1-p} [(1-h) - p(1+h)] = -2k \left( p - \frac{1}{1+h} \right).$$  

(10.46)

The critical relation (10.46) is shown in Figure 10.8 by the dashed-and-dotted curves (obtained for different $k$). The parameter domain in the $(p, h)$ plane, where the relation between the speeds allows for dynamical stabilization to occur, is on the left of a given dashed-and-dotted curve and below the Hopf bifurcation curve 2.

Obviously, in order to make a prediction of dynamical stabilization more complete, the critical relation obtained from a comparison of speeds should be taken together with the one obtained through the bifurcation analysis; see the previous section. The remaining domain is not that large (between curves 2 and 5, on the left of curves 6 or 7). However, it should be mentioned that, while the position of curve 2 does not depend on $k$ and the position of curve 5 depends on $k$ only slightly, the position of the curve defined by (10.46)
depends on \( k \) significantly. With an increase in \( k \), the critical curve (10.46) moves to the right; as an example, curve 7 shows its position for \( k = 1.2 \). As a whole, the parameter domain corresponding to dynamical stabilization tends to increase along with \( k \).

10.3 Patterns in a competing species community

Predation is a very common type of species interaction but surely not the only possible one. Another ecologically relevant and important type is given by competition. In this section, we consider the dynamics of a community of competing species. Our goal is to find out what kind of spatiotemporal patterns can be observed in that system, and what conditions/restrictions are required for these patterns to emerge.

The first observation we want to make here is that, unlike the case of prey–predator systems, a system of two competing species is not capable of forming a pattern more interesting than a traveling monotonous population front (Namba and Mimura, 1980; Shigesada and Kawasaki, 1997). Therefore, in order to explore the possible scenarios of pattern formation in a competing community, we have to consider a somewhat more complicated model.

Apparently, the next level of complexity is a three-species community. Spatiotemporal dynamics of three competitive species is described by the following equations:

\[
\frac{\partial U_1(X,T)}{\partial T} = D_1 \frac{\partial^2 U_1}{\partial X^2} + A_1(1 - r_{11}U_1 - r_{12}U_2 - r_{13}U_3)U_1, \quad (10.47)
\]

\[
\frac{\partial U_2(X,T)}{\partial T} = D_2 \frac{\partial^2 U_2}{\partial X^2} + A_2(1 - r_{21}U_1 - r_{22}U_2 - r_{23}U_3)U_2, \quad (10.48)
\]

\[
\frac{\partial U_3(X,T)}{\partial T} = D_3 \frac{\partial^2 U_3}{\partial X^2} + A_3(1 - r_{31}U_1 - r_{32}U_2 - r_{33}U_3)U_3 \quad (10.49)
\]

(May, 1973; Hofbauer and Sigmund, 1988), where \( U_1, U_2, \) and \( U_3 \) are the species densities at time \( T \) and position \( X \); coefficient \( A_i \) gives the intrinsic growth rate of the \( i \)th species; coefficients \( r_{ij} \) describe intraspecific (for \( i = j \)) and interspecific (for \( i \neq j \)) competition, respectively. Coefficients \( D_i \) describe the intensity of spatial mixing. Due to their biological meanings, all the parameters in (10.47)–(10.49) are nonnegative.

The system (10.47)–(10.49) is a system of nonlinear partial differential equations, for which effective analytical tools are largely lacking. Therefore, as in most parts of this book, an insight into the system properties is done via results of numerical simulations. On the other hand, Equations (10.47)–(10.49) contain a large number of parameters, which makes its detailed study virtually impossible. Therefore, our first task is to lessen the number of parameters.
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One way to do this is to choose dimensionless variables. Namely, let us consider

\[ u_i = r_{ii}U_i \quad (i = 1, 2, 3), \quad t = A_1 T, \quad x = \left( \frac{A_1}{D_1} \right)^{1/2} X. \] (10.50)

Then, from (10.47)–(10.49) we obtain

\[ \frac{\partial u_1}{\partial t} = \frac{\partial^2 u_1}{\partial x^2} + (1 - u_1 - r_{12}u_2 - r_{13}u_3)u_1, \] (10.51)
\[ \frac{\partial u_2}{\partial t} = \epsilon_2 \frac{\partial^2 u_2}{\partial x^2} + a_2(1 - r_{21}u_1 - u_2 - r_{23}u_3)u_2, \] (10.52)
\[ \frac{\partial u_3}{\partial t} = \epsilon_3 \frac{\partial^2 u_3}{\partial x^2} + a_3(1 - r_{31}u_1 - r_{32}u_2 - u_3)u_3, \] (10.53)

where \( \epsilon_2 = D_2/D_1, \quad \epsilon_3 = D_3/D_1, \quad a_2 = \epsilon_2/\epsilon_1, \) and \( a_3 = \epsilon_3/\epsilon_1. \) In terms of original system (10.47)–(10.49), it means that some of the parameters in the equations can be set to unity without any loss of generality.

Furthermore, here we restrict our study to a special “cyclic” type of interspecific competition originally introduced by May and Leonard (1975) and assume that \( r_{13} = r_{21} = r_{32} = \beta \) and \( r_{23} = r_{31} = \gamma. \) Thus we arrive at the following system:

\[ \frac{\partial u_1}{\partial t} = \frac{\partial^2 u_1}{\partial x^2} + (1 - u_1 - \alpha u_2 - \beta u_3)u_1, \] (10.54)
\[ \frac{\partial u_2}{\partial t} = \epsilon_2 \frac{\partial^2 u_2}{\partial x^2} + a_2(1 - \beta u_1 - u_2 - \gamma u_3)u_2, \] (10.55)
\[ \frac{\partial u_3}{\partial t} = \epsilon_3 \frac{\partial^2 u_3}{\partial x^2} + a_3(1 - \gamma u_1 - \beta u_2 - u_3)u_3, \] (10.56)

where we denote \( \alpha = r_{11} \) to keep notations homogeneous. A fully cyclic competition would correspond to \( \alpha = \gamma; \) however, in order to make the system dynamics richer, we keep the possibility that \( \alpha \neq \gamma. \)

As we did in the case of prey–predator system, we begin with the corresponding nonspatial system:

\[ \frac{du_1}{dt} = (1 - u_1 - \alpha u_2 - \beta u_3)u_1, \] 
\[ \frac{du_2}{dt} = a_2(1 - \beta u_1 - u_2 - \gamma u_3)u_2, \] (10.57)
\[ \frac{du_3}{dt} = a_3(1 - \gamma u_1 - \beta u_2 - u_3)u_3. \]

The system (10.57) has been studied in much detail by May and Leonard (1975) and Hofbauer and Sigmund (1988); here we only briefly recall those of its properties that are required for better understanding the spatiotemporal dynamics.
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It is readily seen that system (10.57) can have as many as eight steady states. The number of steady states in the positive octant \( \mathbf{R}^+_3 = \{(u_1, u_2, u_3) \mid u_i \geq 0, \ i = 1, 2, 3\} \) of the phase space can be different for different parameter values, and, in a general case, the structure of the phase space is rather complicated. However, assuming additionally that

\[ \alpha = \gamma \quad \text{and} \quad \alpha + \beta > 2, \quad \alpha > 1 > \beta, \quad (10.58) \]

it can be shown that the system (10.57) possesses exactly five stationary states. Namely, there are the trivial “no-species” state \((0, 0, 0)\) and three semi-trivial “one-species-only” states \((1, 0, 0)\), \((0, 1, 0)\), and \((0, 0, 1)\). There is also one coexistence steady state \((\bar{u}_1, \bar{u}_2, \bar{u}_3)\), the stationary value of the species concentrations being the solution of the following system:

\[ u_1 + \alpha \bar{u}_2 + \beta \bar{u}_3 = 1, \quad \beta \bar{u}_1 + \bar{u}_2 + \gamma \bar{u}_3 = 1, \quad \gamma \bar{u}_1 + \beta \bar{u}_2 + \bar{u}_3 = 1. \quad (10.59) \]

The trivial steady-state is always an unstable node and the semi-trivial states are saddle-points. Furthermore, under restrictions (10.58), the coexistence steady state is always a saddle-point. May and Leonard (1975) showed that, in this case, the only attractor in the phase space is a heteroclinic cycle consisting of the three “one-species-only” steady states and the trajectories connecting these states. In the \( t \to +\infty \) limit, any trajectory in \( \mathbf{R}^+_3 \) ends at the heteroclinic cycle regardless of the initial conditions, which means that the system (10.57) is not permanent. From the ecological standpoint, it means that in a spatially homogeneous community at least one of the species inevitably goes extinct.

The properties of the system (10.57) change significantly when the restriction \( \alpha = \gamma \) is relaxed. Assuming additionally that \( \det \mathbf{R} > 0 \), where \( \mathbf{R} = (r_{ij}) \), a test of the system permanence is

\[ (\gamma - 1)^2(\alpha - 1) < (1 - \beta)^3 \quad (10.60) \]

(for details, see Hofbauer and Sigmund, 1988), which evidently could not be satisfied under restrictions (10.58). For \( \alpha \neq \gamma \), though, condition (10.60) may determine a nonempty set in the parameter space of the system. Taking into account that under condition \( \det \mathbf{R} > 0 \) the solutions of the system (10.54)–(10.56) are uniformly bounded, permanence means the existence of an attractor belonging to the interior of \( \mathbf{R}^+_3 \). The nature of the attractor then depends on the stability of the coexistence state \((\bar{u}_1, \bar{u}_2, \bar{u}_3)\). Since it is the only steady state in \( \mathbf{R}^+_3 \), in the case that it is unstable, a stable limit cycle is expected to emerge. Following this chain of arguments, the existence of a stable limit cycle in the phase space of the system (10.57) has been proved by K. Kawasaki,\(^1\) and the corresponding parameter range was identified. It should yet be mentioned that the system appears to be rather sensitive to a

\(^1\) Personal communication.
variation of parameter values and the parameter range where the limit cycle exists is rather narrow; outside of that range the heteroclinic cycle is the attractor.

Now we proceed to the dynamics of the spatial system (10.54)–(10.56). Since in this chapter we are mostly interested in patterns generated by propagating population fronts, we use initial conditions described by functions of compact support. Specifically, the results shown below are obtained for

\[ u_i(x, 0) = u_{i0} \text{ if } |x| \leq \frac{d_i}{2}, \quad u_i(x, 0) = 0 \text{ if } |x| > \frac{d_i}{2} \]  

(10.61)

\((i = 1, 2, 3)\), where \(u_{i0}\) and \(d_i\) are the concentration of the \(i\)th species inside the originally invaded domain and the size (diameter) of the domain, respectively. To complete the mathematical formulation of the model, the boundary conditions are chosen to be zero-flux.

Some typical snapshots of the system dynamics are shown in Figures 10.9 to 10.11. (Note that the problem (10.54)–(10.56) with (10.61) is symmetrical with respect to the origin and only half of the domain is shown.) At an early stage, a succession of traveling population fronts is formed; see Figure 10.9. Species 3 wins the competition for open space and it spreads faster than the others. Eventually, however, it loses and is displaced by species 1, being actually put at the brink of extinction – note the very low population density of species 3 in the middle of the domain in Figure 10.9, bottom. Species 2 appears to be the weakest in the competition for space; it is the last to come into play and it spreads slower than the other two. Interestingly, species 2 takes some of the pressure from species 1 and saves it from extinction: In the wake of the last front, all three species coexist.

Each of the fronts has a complex shape formed by regular damped population oscillations. Comparison between species distributions obtained for different moments (e.g., cf. Figure 10.9, middle, and Figure 10.10, top) shows that each front propagates as a stationary traveling wave, so that the form of the oscillations does not change with time and the whole structure moves to the right with a constant speed. Apparently, this is a phenomenon of the same origin as the one observed in the prey–predator system; see Figure 10.2.

Also at a later stage, the system properties appear to be qualitatively similar to those of the prey–predator system when its local dynamics is oscillatory. In the wake of the fronts, after decay of the damped oscillations, dynamical stabilization of the unstable equilibrium \((\bar{u}_1, \bar{u}_2, \bar{u}_3)\) may take place; see Figure 10.9. The length of the unstable plateau can increase up to a very large value so that the species distribution becomes homogeneous nearly over the whole domain; cf. Figure 10.10a. In order to estimate the rate of the plateau growth and to identify the parameter range where dynamical stabilization can or cannot be observed, the method of speeds comparison may be applied; see the previous section. However, since the expressions for the fronts speed (more precisely, for the lower bounds) in the three-species system are rather
FIGURE 10.9: The spatial distribution of the species 1 (top), the species 2 (middle), and the species 3 (bottom) obtained at $t = 800$ for $\alpha = 1.08$, $\beta = 0.8$, $\gamma = 1.24$, $\omega_2 = 1.0$, $\omega_3 = 5.0$, $\epsilon_2 = 1.0$, $\epsilon_3 = 0.2$ and initial conditions (10.61) with $u_{10} = 1.1$, $u_{20} = 1.2$, $u_{30} = 1.3$, $d_{1,2,3} = 100$. For these parameters, the coexistence state is unstable and “surrounded” by a stable limit cycle. The arrows indicate the direction of fronts propagation. Since the problem is symmetrical with respect to the origin, only one half of the domain is shown, i.e. for $x \geq 0$.

complicated (Petrovskii et al., 2001), the critical relation appears to be very cumbersome.

Behind the unstable plateau, irregular spatiotemporal population oscillations emerge that eventually occupy the whole domain; compare the left-hand
FIGURE 10.10: Snapshots of the spatial distributions of the species 2 for $t = 800$ (top), $t = 1800$ (middle), and $t = 16600$ (bottom). Parameters are the same as in Figure 10.9. Note that the plateau behind the oscillating front corresponds to the locally unstable equilibrium ($\bar{u}_1, \bar{u}_2, \bar{u}_3$).

An important observation to be made here is that, after the oscillations invade over the domain, the temporal variations of the population densities also become remarkably irregular. A question naturally arises regarding the degree of this irregularity. This issue was addressed by Petrovskii et al. (2001) by means of checking the sensitivity of solutions to a small perturbation of the initial conditions. It was found that, while at the early stage of system evolution the perturbed and unperturbed solutions are very close to each other, starting from a certain
moment the discrepancy begins to grow very fast (exponentially) so that the difference between the solutions soon becomes of the same order as the solutions themselves. This sort of behavior is a fingerprint of chaos (Nayfeh and Balachandran, 1995). Therefore, the dynamics of the system (10.54)–(10.56) after excitation of irregular spatiotemporal oscillations can be classified as chaotic. The dominant Lyapunov exponent is, however, rather small and is roughly estimated to be on the order of $10^{-4}$.

Numerical simulations show that the formation of chaotic spatiotemporal patterns at a later stage of the system dynamics is a typical phenomenon in the sense that it is robust to variation of the system parameters in a wide range, even if the variation results in a change of the phase space structure. Indeed, while the patterns in Figure 10.10 are obtained for stable limit cycle being the attractor, Figure 10.11 shows the patterns obtained in the case that the criterion (10.60) of permanence is broken and the heteroclinic cycle is an attractor. No unstable plateau is formed in this case, and the chaotic region starts immediately after the regular oscillations at the front.

It should also be mentioned that, as well as in the prey–predator system, the chaotic oscillations are self-sustained. Large-time numerical simulations show that, after the onset of chaos, the system dynamics does not undergo any qualitative changes.

Therefore, in this section we showed that, in a three competing species system where the local kinetics is given either by a heteroclinic attractor or by a stable limit cycle, population fronts are followed by the formation of an unstable plateau and/or by excitation of chaotic spatiotemporal population oscillations. These results provide an important extension to the analysis and inferences of the previous sections showing that the formation of complex spatiotemporal patterns is a more general phenomenon and should not be necessarily attributed to the prey–predator interactions. Even more importantly, they should not be attributed to the existence of a stable limit cycle either: Essentially the same type of system’s dynamics is observed in the case of an heteroclinic attractor.

One ecologically important conjecture of this study must not be overlooked. Let us recall that, in the case of an heteroclinic attractor, in the nonspatial system the system’s trajectory is gradually approaching the boundary of the first octant in the phase space. From the ecological standpoint, it would obviously correspond to species extinction because the system moves through a succession of states with very low population densities (Gilpin, 1972; May, 1972a). Moreover, a species would go extinct without any chance of re-colonization because, due to spatial homogeneity, its population density falls to a dangerously low value simultaneously all over the domain. However, the situation appears to be different in the spatial system: Due to homogeneity breakdown and the subsequent pattern formation, no extinction takes place and all three species coexist throughout the domain; cf. Figure 10.11. Therefore, the predictions obtained from spatial and nonspatial models are qualitatively different. This is a new feature, which we have not observed for the prey–predator system.
FIGURE 10.11: The spatial distributions of the species 1 (top) and the species 2 (bottom) obtained at $t = 3200$ for $\beta = 0.95$; other parameters are the same as in Figure 10.9. The system is not permanent and the heteroclinic cycle is the attractor.

considered in the previous sections. In Chapter 12, we will show that a similar situation may occur for a prey–predator system when the prey growth is hampered by the Allee effect.
10.4 Concluding remarks

We showed that, in a conceptual population community such as a prey– predator system or a system of three competing species, a traveling population front can generate a variety of patterns. The system dynamics is especially rich when the coexistence state is unstable. A typical succession of patterns then looks as follows. Damped oscillations at the front are followed by a plateau emerging as a result of dynamical stabilization of the unstable coexistence state. The plateau is followed by a periodic wavetrain. In the wake of the periodic wave, irregular spatiotemporal oscillations gradually develop. Remarkably, even in the case when the periodic wave is stable with respect to small local perturbations, it often appears to be unstable globally, so that the periodic wavetrain is gradually displaced by irregular oscillations. Having once appeared, the irregular oscillations eventually occupy the whole domain. Depending on parameter values, the plateau and/or the periodical pattern can be missing; however, the irregular spatiotemporal oscillations always arise in the wake of invasion.

Note that the main properties of the system dynamics appear to be robust to details of the phase space structure and, contrary to a widely accepted opinion (Kuramoto, 1984), should not be tightly related to the existence of a stable limit cycle; essentially the same pattern emerges if the attractor is given by a heteroclinic cycle. Moreover, an insight into mathematically similar models of chemical systems shows that damped oscillations at the propagating front, dynamical stabilization, and excitation of irregular oscillations in the wake can be observed as well even in the case that the attractor is just as simple as a stable node, provided the coexistence state is unstable (Malchow and Petrovskii, 2002).

After excitation of irregular spatiotemporal oscillations, the dynamics of the system becomes chaotic. It should be mentioned here that the term “chaos” appeared originally in relation to the temporal behavior of spatially homogeneous systems (Lorenz, 1963; Li and Yorke, 1975; Gilpin, 1979; Hastings and Powell, 1991). In our case, however, the phase space of the homogeneous system does not contain a strange attractor but only a (stable) limit cycle or heteroclinic cycle. Chaotic dynamics becomes possible as a result of homogeneity breaking and the formation of irregular spatial species distribution. It seems reasonable to distinguish between the situations when a given system may or may not exhibit chaos in the nonspatial case. In order to stress the importance of the spatial dimension of the system, to refer to the chaotic patterns considered in this chapter the term “spatiotemporal chaos” is more appropriate.

As a whole, the dynamics of simple few-species model systems continuous in space and time appears to be remarkably rich and capable of generating realistically looking complicated irregular patterns. We want to note that
appreciation of this fact once became a real breakthrough in understanding population dynamics (Pascual, 1993; Sherratt et al., 1995). Until the mid-nineties, irregularities were normally associated with the discreteness of the dynamics either in time, cf. “populations with nonoverlapping generations” (May, 1974), or space (Hassell et al., 1991), rather than with intrinsic instabilities of continuous systems.

In this chapter, we focused on patterns generated by propagating population fronts. Excitation of chaotic oscillations in a prey–predator system used to be essentially attributed to the front wakes (Sherratt, 1994b, 1998) and, correspondingly, to the initial conditions of special type (e.g., finite) generating the fronts. In their influential paper, Sherratt, Lewis, and Fowler (1995) insisted that formation of chaotic patterns in the wake of invasion is a “fundamentally different mechanism” from, for instance, the one considered by Pascual (1993) where spatiotemporal chaos was triggered by a small constant-gradient heterogeneity in the prey growth rate. Later studies, however, showed that it is not so. In the next chapter, we will show that onset of spatiotemporal chaos and the corresponding formation of distinctly heterogeneous, patchy population distribution is a much more general phenomenon and must not be necessarily attributed to propagating fronts.

A question yet remaining open is how the system dynamics may change if the population growth is affected by the Allee effect. Indeed, even in the simplest case of a single-species system continuous in space and time, the system’s properties are significantly different depending on whether the Allee effect is present or not (cf. Lewis and Kareiva, 1993). This issue will be addressed in details in Chapter 12. We will show there that the impact of the Allee effect makes the system dynamics even richer, and, along with the patterns already considered here, it results in a new phenomenon in population spread and the corresponding pattern formation.

Since one of the goals of this book is to “bridge the gaps” between ecological and epidemiological models, another question remains as to whether and to what extent the results of this chapter can be extended onto patterns in the dynamics of epidemics. Here the following observation is to be made. The PDE-based diffusion–reaction models of epidemic spread are similar to the population models in the sense that the spatiotemporal dynamics of a given infectious disease arises as an interplay between dispersal/diffusion and local interaction between different subpopulations or groups, e.g., susceptibles and infected. Consequently, as well as in the case of population models, the possible scenarios of pattern formation in the wake of epidemic spread depend very much on the properties of the local kinetics.

Aiming to give more specific examples, the simplest SI model of disease spread with the mass-action law for the disease transmission rate qualitatively coincides with a prey–predator system where predation is of Holling type I, provided only susceptible individuals are capable of reproduction. Therefore, since in this system the endemic/coexistence state is always stable, the spreading epidemic would form either a monotonous traveling front or a front with
promptly damped oscillations in the wake; cf. Figure 10.2. Numerical simulations in the SI model agree with these predictions perfectly (Murray, 1989). Furthermore, it is readily seen that introduction of the Allee effect into the reproduction rate (for more details see Chapter 12) can destabilize the endemic state and thus makes possible the formation of complicated irregular spatiotemporal patterns in the wake (Petrovskii et al., 2005).

In general, however, epidemic models tend to have local kinetics more complicated than that of population dynamics. One reason is that the above assumption that only susceptibles contribute to population growth actually applies only to a narrow range of diseases (e.g., to those affecting the reproduction system). For most other diseases, contribution of infected to population multiplication cannot be neglected. Obviously, it immediately results in a much more elaborated reproduction term. Another reason can be found in the application of more complicated schemes of disease transmission, such as proportionate mixing or even a combination of the mass-action law and the proportionate mixing (cf. Fromont et al., 1998).

The outcome of the interplay between different factors is very difficult to predict, though. Having been taken separately, the Allee effect, the proportionate mixing, and the contribution of infected to reproduction tend to make the kinetics more complex and the system dynamics richer. Surprisingly, when they are all taken into account, the resultant model may appear to have simpler properties. The simplest SI model with the Allee effect is capable of local limit cycle oscillations and the corresponding formation of complex spatiotemporal patterns; however, an SI model accounting also for the other two factors exhibits nothing more complicated than traveling fronts (Hilker et al., 2007).
Chapter 11

Biological turbulence

Spatial distribution of ecological populations is very rarely homogeneous. On the contrary, patches of high population density quite often alternate with patches of low density or even with uninhabited areas. It can occur on different spatial scales and in various environment and ecosystem types. In population biology and spatial ecology, this phenomenon may come under a variety of names such as plant/animal grouping or aggregation, patchiness, etc. In some cases (e.g., see Section 9.3), the corresponding spatial structure exhibits prominent regularity, even if it might be blurred by environmental noise. Much more often, however, populations are distributed irregularly, without any detectable trace of order.

Remarkably, although environmental and/or landscape properties apparently create a certain “frame” for any population heterogeneity, the properties of observed spatial structures often appear to be uncorrelated or only weakly correlated with those of the environment. This fact has been used as a conceptual basis for considering patchiness as a separate phenomenon, appearing more due to biological interactions rather than being straightforwardly reducible to physical/chemical properties of the environmental settings; see Levin (1990, 1992) and Powell (1995). Although patchiness due to environmental forcing is a practically important and interesting phenomenon, its reasons and the corresponding mechanisms are relatively clear; therefore, in this chapter (as indeed in most parts of this book) we will focus more on its possible biological origin.

Apparently, the mechanisms of self-organized biological patchiness can be different depending, for instance, on how complex are the “social” aspects of the population dynamics of a given species. Perhaps the very first example of animal aggregation that comes into mind is the formation of flocks, herds, schools, swarms, etc. In particular, fish schooling has been paid a lot of attention for obvious commercial reasons. However, here we are more interested in patterns that arise irrespectively of whether the given species may or may not possess this type of behavioral response. It means that the relevant spatial scale should be large enough so that the population density would not be affected by the small-scale spatial variations due to flock/swarm formation. Spatial heterogeneity that appears largely as a result of schooling behavior lies beyond the scope of this book; a reader interested in this issue is advised to use extensive existing literature, e.g. see Radakov (1973); Steele (1977);
Okubo (1986); Huth and Wissel (1994); Reuter and Breckling (1994); Niwa (1996); Parrish et al. (1997); Stöcker (1999); Parrish and Edelstein-Keshet (1999); Saffre and Deneubourg (2002).

As was mentioned above, specific mechanisms and the corresponding theoretical background for understanding self-organized ecological patterns may depend significantly on the environmental properties, in particular, on the degree of its heterogeneity. In a strongly fragmented landscape, the species areal consists of an ensemble of sites or habitats. In case their typical size is small enough so that the population distribution inside each of them can be considered as homogeneous (up to inevitable small fluctuations of purely stochastic origin), the spatial structure is predetermined and the properties of the spatiotemporal dynamics can then be expressed in terms of synchronization between population fluctuations at different sites. A relevant mathematical model should be space-discrete and may be given by coupled systems of ODEs, with each system describing the population dynamics inside the corresponding habitat. We mention here the work by Jansen (1995, 2001), who showed that the local prey–predator oscillatory dynamics becomes desynchronized as soon as the inter-habitat coupling becomes sufficiently weak. As a result of weak coupling, the local dynamics of the subpopulations become chaotic even if the intrinsic dynamics of the corresponding local populations is periodic.

These and other similar results, although very important for understanding the population dynamics on a metapopulation scale, give little information with regards to what can be the mechanisms of self-organized patchiness in a relatively homogeneous environment (e.g., within a given habitat, provided its size is large enough). In that case, a mathematical description continuous in space and time seems to be the most adequate, which results in diffusion–reaction equations. In the previous chapter, using a prey–predator system as a paradigm, we showed that formation of spatially irregular chaotic patterns in such systems can be triggered by biological invasion, i.e., by propagation of a population front emerging from finite initial conditions. Indeed, earlier work on ecological pattern formation essentially attributed self-organized heterogeneous populations distribution\(^1\) to the wakes of propagating fronts (Mark Lewis, personal communication; also Sherratt (1994a); Sherratt et al. (1995, 1997)). It appears, however, that pattern formation in a spatial prey–predator system is a more general, inherent phenomenon and must not necessarily be associated with initial conditions of a special type or any predefined environmental heterogeneity (Petrovskii and Malchow, 1999; Petrovskii and Malchow, 2001b). Rather, it reflects a certain intrinsic instability of a spatially explicit prey–predator system. Remarkably, however, contrary to corresponding non-spatial systems, where an instability may eventually lead to species extinction

\(^1\)In an unstructured environment; phenomenon of self-organized patchiness triggered by (but not correlated with) an environmental heterogeneity of special type was first observed and studied in detail in influential papers by Pascual (1993) and Pascual and Caswell (1997).
(Rosenzweig, 1971; Gilpin, 1972), this instability of spatiotemporal dynamics appears to be beneficial for the community functioning enhancing global species persistence (Petrovskii et al., 2004).

The chapter is organized as follows. In Section 11.1, we give a detailed description of the self-organized patchiness – “biological turbulence” – focusing on the generic scenario of pattern formation and transition to spatiotemporal chaos in a prey–predator system. In Section 11.2, we make a stronger accent on the spatial aspects of the system dynamics after the onset of chaos and also give an extension of the main results onto other similar systems. Section 11.3 makes an insight into possible ecological consequences of the phenomenon. The last section provides a discussion of the main results.

11.1 Self-organized patchiness and the wave of chaos

The scenario of pattern formation resulting in a remarkably irregular patchy spatial structure and transition to chaos, which will be a focus of this section, appear to be generic for a wide class of diffusion–reaction systems where local kinetics is oscillatory. Here “oscillatory” is used in a wider sense and does not necessarily imply the existence of a stable limit cycle in the phase space of the corresponding nonspatial system; in fact, having an unstable focus may be enough. The case when the local kinetics given by a limit cycle can be treated in terms of the so-called $\lambda\omega$ systems (Kopell and Howard, 1973) or its generalization, known as the Ginzburg–Landau equation (Kuramoto, 1984), which are widely regarded as adequate models of spatiotemporal chaos (but see the discussion of the issue in Section 11.4). A more general theory is largely missing, though, and many results are still obtained through numerical simulations in specific systems. Correspondingly, in this section we consider a prey–predator system as the simplest example of a population community with oscillatory kinetics. Some generalizations and extensions of the results onto other systems will be made in Section 11.2.

A general model of a time- and space-continuous system of two interacting species is given by the following equations:

\[
\frac{\partial U(R, T)}{\partial T} = D\nabla^2 U + \phi(U, V), \quad (11.1)
\]

\[
\frac{\partial V(R, T)}{\partial T} = D\nabla^2 V + \psi(U, V), \quad (11.2)
\]

where $U$ and $V$ are the species densities, i.e., presently, the population densities of prey and predator, respectively. Since here we are mostly interested in the possibility of non-Turing patterns, we assume that the diffusivity is the same for both species.
Assuming the logistic growth for prey and the Holling type II for predation, and introducing dimensionless variables in a standard way, the system (11.1)–(11.2) takes the following specific form:

\[
\frac{\partial u(r,t)}{\partial t} = \nabla^2 u + u(1 - u) - \frac{uv}{u + h}, \tag{11.3}
\]

\[
\frac{\partial v(r,t)}{\partial t} = \nabla^2 v + k \left( \frac{uv}{u + h} - pv \right), \tag{11.4}
\]

cf. equations (10.3) to (10.7) of the previous chapter.

The properties of the corresponding nonspatial model were considered in detail in Section 10.1, where it was shown that the only coexistence steady state \((\bar{u}, \bar{v})\) is unstable for \(h < (1 - p)/(1 + p)\) and stable (when feasible) otherwise. When it is unstable, it is surrounded by a stable limit cycle. Since a possibility of the Turing instability has been ruled out, one can hardly expect pattern formation in the case when \((\bar{u}, \bar{v})\) is stable. Therefore, in the computer simulations below we will focus on the parameter range where the steady state is unstable.

We start our insight into the spatiotemporal dynamics of the system (11.3)–(11.4) by considering the one-dimensional case so that \(u = u(x,t), v = v(x,t), \nabla^2 = \partial^2/\partial x^2, \text{ and } 0 < x < L, L\) being the domain size.

Obviously, the spatiotemporal dynamics of the system depends to a large extent on the choice of initial conditions. Contrary to the previous chapter, here we are interested in a situation where, at the beginning, both populations are present over the whole area. In a real ecosystem, the details of the initial spatial distribution of the species can be caused by quite specific reasons. The simplest and, in some sense, most general form of the allocated initial distribution would be spatially homogeneous initial conditions. However, in that case, the distribution of the species would stay homogeneous for any time, and no spatial pattern can emerge. To get a nontrivial spatiotemporal dynamics, one has to perturb the homogeneous distribution.

We begin with a hypothetical “constant-gradient” distribution:

\[
u(x,0) = \bar{v} + \epsilon x + \delta, \tag{11.6}\]

where \(\epsilon\) and \(\delta\) are parameters. Since the system (11.3)–(11.4) is invariant with respect to transformation \(x \rightarrow -x\), we reduce our study to the case \(\epsilon > 0\) without any loss of generality.

It appears that the type of the system dynamics depends significantly on \(\epsilon\) and \(\delta\). In case \(\epsilon\) is small and \(\delta\) is positive, the initial conditions (11.5)–(11.6) evolve to a smooth nonmonotonic spatial distribution of species; see Figure 11.1. The spatial distributions gradually vary in time; the local temporal behavior of the dynamical variables \(u\) and \(v\) is strictly periodical following the limit cycle of the nonspatial system.
A periodic temporal behavior together with a smooth spatial population distribution, like the one shown in Figure 11.1, is perhaps what is intuitively expected from the system (11.3)–(11.4). Indeed, in the field of theoretical ecology, a two-species diffusion–reaction system had long been regarded as too simple to be capable of producing anything more complicated than a regular pattern with regular dynamics, and that was one of the motivations that led researchers to build more complicated models.

However, for a slightly different set of parameters, the dynamics of the system undergoes principal changes; see Figure 11.2. In this case, the initial distribution (11.5)–(11.6) leads to the formation of a strongly irregular “jagged” dynamic pattern inside a subdomain of the system. The size of the region occupied by this pattern steadily grows with time (cf. top and bottom of Figure 11.2), so that finally irregular spatiotemporal oscillations invade over the whole domain.

Also, the temporal behavior of the densities \( u \) and \( v \) becomes completely different. Figure 11.3 shows the “local” phase plane of the system obtained at a fixed point \( \bar{x} \) inside the region invaded by the irregular spatiotemporal oscillations, the trajectory now filling nearly the whole area inside the limit cycle.

This remarkable irregularity invokes a question of whether it is actually chaotic. In order to clarify the issue, sensitivity of the solutions to a small perturbation has been checked using different measures; for details, see Med-
vinsky et al. (2002). It was shown that, while during an initial stage of the system dynamics the difference between the perturbed and unperturbed solutions remains small, for a larger time it starts growing promptly so that shortly thereafter the difference becomes on the order of the solutions them-

FIGURE 11.2: Population distribution over space at \( t = 1000 \) (top) and \( t = 2000 \) (bottom) obtained for the initial conditions (11.5)–(11.6) with \( \epsilon = 10^{-5} \) and \( \delta = -1.5 \cdot 10^{-2} \); other parameters are the same as in Figure 11.1). Solid line for predator, dashed for prey.
FIGURE 11.3: Local phase plane of the system (11.3)–(11.4) after formation of irregular sharp pattern; the trajectory is shown for time interval between \( t = 3000 \) and \( t = 5000 \). Parameters are the same as in Figure 11.2. The envelope of the domain filled with the system’s trajectory coincides with the limit cycle.

selves; see Figure 11.4. This behavior obviously complies with the definition of chaos. Also, chaos in the dynamics of the jagged pattern can be demonstrated by considering the spectra of the corresponding time series (Petrovskii and Malchow, 1999). Note that chaos in the system (11.3)–(11.4) is essentially spatiotemporal because the nonspatial system can only exhibit a simpler dynamics as given by steady states and limit cycles.

A curious property of the system dynamics is that, for each moment of time, there exist distinct boundaries separating the regions occupied by different dynamic regimes, i.e., by jagged/chaotic and smooth/regular patterns. Both numerical results and analytical estimates (Petrovskii and Malchow, 2001b) show that these boundaries propagate with an approximately constant speed in opposite directions, so that the size of the region with chaotic dynamics is always growing until it occupies the whole domain. Thus, the chaotic regime arises as a result of the propagation of the “wave of chaos,” i.e., of the moving interface between the two regions. The scenario is essentially spatiotemporal: The chaos prevails as a result of displacement of the regular regime by the chaotic regime.

An issue to address is what factors determine the position of the “chaotic embryo” when it first appears in the course of the system dynamics. A careful analysis of the computer simulation results show that, in case the initial
species distribution contains a point of “phase singularity” (cf. Kuramoto, 1984), it is in a vicinity of this point that the jagged chaotic pattern first develops. For the initial conditions given by (11.5)–(11.6), such a point $x_*$ is obviously determined as a solution of the following equation:

$$v(x,0) = \bar{v} + \epsilon x_* + \delta = \bar{v} \quad \text{so that} \quad x_* = -\frac{\delta}{\epsilon} \quad (11.7)$$

(hence the empirical observation that, for $\epsilon > 0$ and $\delta > 0$, the system may sometimes stay in the regular regime; cf. the lines below Equations (11.5)–(11.6)).

The existence of a phase-singularity point is not a necessary condition of the onset of chaos, though. Interestingly, when the initial conditions do not possess such a point (e.g., when $\epsilon \delta > 0$), the chaotic pattern can develop anyway provided $\epsilon$ is not very small. However, in this case, the factors determining the position of the chaotic embryo remain obscure.

The dynamics with more general initial conditions (e.g., nonmonotonic) can be even more complicated, showing a phenomenon that may be called intermittency, when the domains occupied by regular and chaotic patterns alternate in space. Indeed, this sort of spatially intermittent dynamics should
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be expected if we take into account that nonmonotonic initial conditions may include more than one point of phase singularity and, consequently, may evolve to a few chaotic embryos. As a particular example, we consider the initial conditions in the following form:

\[
    u(x, 0) = \bar{u},
    \]
\[
    v(x, 0) = \bar{v} + \epsilon x + \delta + \epsilon_1 \cos \left( \frac{2\pi x}{L} \right).
    \]

In this case, a slightly perturbed homogeneous initial species distribution evolves to a pattern where two domains occupied by the jagged chaotic pattern are separated by regions with a smooth pattern; see Figure 11.2. As in the previous case, the size of the chaotic domains steadily grows, so that they eventually displace the regular dynamics and occupy the whole region.

\[
\text{FIGURE 11.5: Intermittent population spatial distribution (solid curve for predator, dashed-and-dotted one for prey) obtained at } t = 450 \text{ for slightly heterogeneous non-monotonic initial conditions (11.8)–(11.9) with } \epsilon_1 = 0.07, \epsilon = 10^{-5}, \delta = 0, \text{ and } L = 3000. \text{ Other parameters are the same as in Figures 11.1 to 11.3.}
\]

We want to emphasize, based on extensive evidence from numerous computer simulations, that the onset of chaos in the system (11.3)–(11.4) follows exactly the same scenario for any parameter value when the local kinetics
of the system is oscillatory. Therefore, the type of dynamics shown in Figures 11.2 to 11.5 should be regarded as typical rather than exotic. In addition, we note that apparent asymmetry between $u(x, 0)$ and $v(x, 0)$ in the initial conditions considered above, when $u(x, 0)$ is constant and $v(x, 0)$ is perturbed, is only a matter of choice and does not have any special meaning. Other forms of initial conditions, e.g., when $u(x, 0)$ is perturbed and $v(x, 0)$ is constant, or the distribution is perturbed for both species, lead to qualitatively the same dynamics of the system.

It should also be mentioned that the above results of computer simulations were very carefully checked and tested in order to exclude any significant numerical artifacts. In particular, sensitivity of the results to the size of the mesh steps was checked and they were chosen small enough in order to reach the required degree of approximation and accuracy. The numerical mesh used in the simulations was large enough (typically, with the number of nodes on the order of $10^4$) so that each peak in the chaotic pattern was approximated by a few dozens of nodes.

11.1.1 Stability diagram and the hierarchy of regimes

So far we have shown that, for a rather general class of initial conditions, the dynamics of the system (11.3)–(11.4) leads either to the formation of a time-periodical smooth spatial pattern or, via propagation of the “wave of chaos,” to the formation of jagged chaotic spatiotemporal patterns. In this section, we will make a further insight into the conditions for the generation of the two regimes and into their persistence.

In order to address the first of these issues, we calculate the stability diagram, i.e., what can be the type of system dynamics in response to a perturbation of a spatially homogeneous initial distribution. For that purpose, we consider the following initial conditions:

\[ u(x, 0) = \bar{u} , \]
\[ v(x, 0) = \bar{v} + A \sin \left( \frac{2\pi(x - x_0)}{S} \right) \quad \text{for} \quad x_0 \leq x \leq x_0 + S , \]
\[ v(x, 0) = \bar{v} \quad \text{for} \quad x \leq x_0 \quad \text{or} \quad x \geq x_0 + S , \]

and check the type of the system dynamics for the parameters $A$ and $S$ varying in a wide range.

The whole parameter plane $(S, A)$ appears to be divided into three regions: see Figure 11.6. If at least one of the parameters is small enough (region I),
FIGURE 11.6: Stability diagram. A sketch of the map in the parametric ($S, A$) plane (semilogarithmic) for the locally disturbed initial conditions (11.10)–(11.11) obtained for parameters $k = 2.0, p = 0.3$, and $h = 0.4$. Different domains correspond to the excitation of different regimes; see comments in the text. With permission from Petrovskii and Malchow (2001b).

The initial perturbation (11.10)–(11.11) of the stationary homogeneous spatial distribution does not lead to any pattern formation. In this case, the spatially homogeneous distribution is restored and the only consequence is that the local dynamics now becomes periodical corresponding to the limit cycle. If the values of amplitude $A$ and magnitude $S$ are larger (region II), then the initial perturbation leads to excitation of the smooth pattern; see Figure 11.1 as an example. Finally, for even larger values of $A$ and $S$ (region III), the initial condition (11.10)–(11.11) leads to excitation of the jagged chaotic pattern according to the scenario described above. These results are also summarized in the diagram in Figure 11.7; lines 1, 2, and 3 correspond to parameters from domains I, II, and III respectively. Note that the steady-state population densities for the parameters of Figure 11.6 are $\bar{u} = 0.171$ and $\bar{v} = 0.473$ whereas the minimum value of the perturbation amplitude $A$ sufficient to trigger the formation of chaotic pattern is typically quite small, i.e., on the order of $10^{-4}$. Numerical experiments show that, although particular figures can be somewhat different for a different set of parameters, the critical values of $A$ and $S$ remain of the same order.

These results lead to the conclusion that the formation of a jagged irregular dynamical spatial pattern is a rather typical, “natural” phenomenon for a locally oscillating, spatially explicit prey–predator community. The regular
dynamics is stable with respect to perturbations of very small amplitude. However, a larger perturbation drives the system to spatiotemporal chaos. Note that, conceptually, it is in very good agreement with the conclusion made earlier regarding stability of periodic wakes (cf. Section 10.1): The regular (periodic) regime may be stable linearly but the basin of stability is very small, so that it is not persistent in computer simulations and would normally give way to chaos due to the impact of perturbation of various origins such as the impact of boundary conditions, imperfect numerical approximation, etc.

Figure 11.6 gives an account of system stability with respect to a small heterogeneous perturbation of a spatially homogeneous, locally unstable steady state. However, the situation when, prior to the perturbation, the system is in an unsteady state may look rather elaborate. Probably more natural would be an option when the initial spatially homogeneous population distribution would correspond to a stable limit cycle. Indeed, even if the homogeneity itself is preserved (cf. domain I in Figure 11.6), the perturbation drives the system from the equilibrium state to homogeneous oscillations. This case has been addressed as well, with similar conclusions about system stability, although it is more difficult to present the results in a compact form because the critical amplitude is different for different oscillation phases, i.e., for different positions of the system on the cycle. On the whole, the critical amplitude now appears to be larger than it was previously (i.e., for the initial conditions corresponding to the homogeneous unstable steady state), with the critical amplitude typically being between $10^{-3}$ and $10^{-2}$. Note that it is still well within a few per cent of the population density steady-state value, which confirms the above conclusion that the spatially homogeneous state is stable linearly but is unstable "globally."

The next point is the persistence of the regimes. In order to address this issue, the system dynamics has been checked in long-term numerical simulations. Our results show that the smooth spatial pattern (see Figure 11.1) is not self-sustained. This regime of the system dynamics is, in fact, the process of a very slow relaxation (with a characteristic time $\tau_{\text{phase}} \sim 10^3$ to $10^6$) to the spatially homogeneous temporary periodical solution. The corresponding dynamics looks as follows. First, after a perturbation of the initial homogeneous state, the process of local relaxation to the stable limit cycle takes place (transition 2 in Figure 11.7). In each point $x$, the dynamical variables $u$ and $v$ approach the periodical solution of the homogeneous system. The local relaxation occurs almost simultaneously over the whole domain, and the time scale of this process is $\tau_{\text{amp}} \sim 10^2$ to $10^3$, depending on the problem parameters. This stage can be regarded as the "amplitude relaxation"; as a result, the amplitude of the local oscillations becomes the same. However, the oscillations at different positions take place with different phases. Then, the process

\[\text{in the standard terminology of linear stability theory it would correspond to the "local stability"; however, here we reserve the word "local" for nonspatial systems.}\]
of “phase relaxation” begins, cf. transition 4 in Figure 11.7, which is much slower but eventually leads, after time $\tau_{\text{phase}} \gg \tau_{\text{ampl}}$, to synchronization of the local oscillations all over the domain.

Conversely, the regime of chaotic spatiotemporal oscillations is persistent. In this case, there are also two different time scales. The first scale, $\tau_{\text{emb}}$, corresponds to the formation of the chaotic embryo(s); this time is roughly estimated to be of the same order as $\tau_{\text{ampl}}$. The second time scale, $\tau_{\text{dis}}$, corresponds to the growth of the chaotic domain(s) and to the displacement of the regular pattern by chaos through the propagation of the “wave of chaos”; cf. Figure 11.2. The specific value of $\tau_{\text{dis}}$ apparently depends on the domain length $L$ and also on other parameters through the speed of the wave propagation (Petrovskii and Malchow, 2001b). After the chaotic phase occupies the whole domain, the dynamics of the system does not undergo any further quantitative changes. In particular, the long-time numerical simulations (up to $t \sim 10^6$) show that the form and size of the chaotic attractors both in a local $(u,v)$ plane (i.e., at a fixed $\bar{x}$) and in the plane $<u>, <v>$ of the spatially averaged values remain unchanged.

Therefore, based on the results of extensive numerical experiments, we have

\begin{figure}
\centering
\includegraphics[width=0.5\textwidth]{figure11_7.png}
\caption{Hierarchy of regimes. The arrows show all possible transitions between different regimes observed in the course of the system dynamics. With permission from Petrovskii and Malchow (2001b).}
\end{figure}
shown that the parameter range where spatial homogeneity in the system (11.3)–(11.4) is either maintained or gives way to a regular spatiotemporal pattern is quite narrow. For the parameters outside of this range, the system is driven to spatiotemporal chaos that is self-sustained and persistent.

We also mention that, having once emerged, the chaotic pattern appears to be robust with respect to moderate variations of the problem parameters $k$, $p$, and $h$. Interestingly, however, chaos may be suppressed when the parameters get sufficiently close to the Hopf bifurcation point, even if the local kinetics still remains oscillatory. In Section 11.2, we will address this issue in detail and show that it happens when the correlation length of the system exceeds the domain length.

11.1.2 Patchiness in a two-dimensional case

Thinking about the possible ecological implications of the above results, a question of immediate interest is to what extent they can be extended onto the case of two spatial dimensions and how the self-organized patchiness, if any, may look in a two-dimensional prey–predator system. Indeed, the dynamics of ecological populations rarely takes place along a line (but see Lubina and Levin, 1988); however, it is well known that the system’s properties can be rather different depending on the number of spatial dimensions.

In order to address this issue, we consider the full two-dimensional version of Equations (11.3)–(11.4), where now $\nabla^2 = \partial^2/\partial x^2 + \partial^2/\partial y^2$, $0 < x < L_x$, and $0 < y < L_y$. At the domain boundary, zero-flux conditions are imposed. The equations are solved numerically. As well as in the one-dimensional case, the type of system dynamics depends on the choice of the initial conditions. For a purely homogeneous population initial distribution, the system stays homogeneous forever. For a slightly perturbed homogeneous initial distribution (the shape of the perturbation can be different; cf. (11.5)–(11.6) and (11.10)–(11.11)), a smooth pattern arises, which is not persistent and gradually converges to a homogeneous distribution. For a greater initial perturbation, the system evolves to the formation of an irregular patchy spatial pattern. A typical example is shown below.

We consider the initial conditions describing a dome-shaped distribution of prey placed into a domain with a constant-gradient predator distribution:

\[ u(x, y, 0) = \bar{u} + \epsilon_1(x - 0.2L_x)(x - 0.8L_x) + \epsilon_2(y - 0.3L_y)(y - 0.7L_y), \]
\[ v(x, y, 0) = \bar{v} + \epsilon_3(x - 0.5L_x) + \epsilon_4(y - 0.45L_y), \]

where $\epsilon_1 = -2 \cdot 10^{-7}$, $\epsilon_2 = -6 \cdot 10^{-7}$, $\epsilon_3 = -3 \cdot 10^{-5}$, and $\epsilon_4 = -6 \cdot 10^{-5}$ are parameters.

Figure 11.8 shows snapshots of the prey density distribution over space at $t = 0$, 150, 350 and 1500 (top to bottom) for the same parameter values as in Figures 11.1 to 11.6, i.e., for $k = 2.0$, $p = 0.3$, $h = 0.4$. Obviously, the
**FIGURE 11.8:** (See color insert.) Snapshots of the prey distribution over two-dimensional space for (top to bottom) \( t = 0 \), \( t = 150 \), \( t = 350 \), and \( t = 1500 \). Parameters are given in the text; see Equations (11.12)–(11.13) and below. Predator density shows qualitatively similar behavior except for very early stages of the system dynamics when the impact of initial conditions is essential.
Spatiotemporal patterns in ecology and epidemiology

System dynamics follows a scenario of pattern formation that is qualitatively similar to the one observed in the one-dimensional case. At an early stage, the population distribution is smooth and regular, forming spiral-like spatial structures. The chaotic embryos then appear in the vicinity of the spiral centers, which are readily seen to correspond to the points of the phase singularity \((x_*, y_*)\), where \(u(x_*, y_*) = u_*\) and \(v(x_*, y_*) = v_*\). A circular boundary separates the domains with jagged, patchy chaotic structures from the rest of the system where the dynamics is smooth in space and periodic in time. The domains grow gradually, and, finally, the chaotic pattern invades over the whole available space.

We want to emphasize that, although Figure 11.8 shows the results of a single simulation run accomplished for a given parameter set, the succession of patterns like “spirals → chaotic embryos → spatiotemporal chaos” is typical for a two-dimensional diffusion–reaction system with oscillatory kinetics. More examples (obtained for different initial conditions, different parameter values, and by different numerical methods) can be found in Medvinsky et al. (2002) and Garvie (2007); for an earlier reference see also Kuramoto (1984).

11.2 Spatial structure and spatial correlations

In the case of spatiotemporal chaos, the population densities fluctuate or oscillate with time in an irregular, stochastic-like manner. Furthermore, they also show a qualitatively similar behavior in space. The fact that the populations’ spatial distribution exhibits a prominent irregular patchy structure seems to indicate that the local oscillations may turn out to be “out of phase” when the distance between any two positions becomes sufficiently large. This spatial aspect of the population dynamics is usually quantified in terms of the correlation length: Population fluctuations in points \(r_1\) and \(r_2\) can be regarded as independent (uncorrelated) in the case \(|r_1 - r_2| > L_{corr}\). Within the distance \(L_{corr}\), the population oscillations are correlated due to the diffusive coupling between neighboring points.

Clearly, the correlation length is an important characteristic of the system’s spatiotemporal dynamics. In order to calculate \(L_{corr}\), a variety of approaches can be used (Pascual and Levin, 1999; Durrett and Levin, 2000); for a discussion of related issues also see Rand and Wilson (1995). Perhaps the most common way is to derive the value of \(L_{corr}\) from the properties of the spatial correlation function of the population fluctuations. It should be noted here that the state of a prey–predator community is naturally described by the two dynamical variables \(u\) and \(v\), i.e., the densities of prey and predator, respectively. Thus, for a general situation, one has to consider two autocorrelation functions as well as the cross-correlations. However, since both
variables show a qualitatively similar behavior, here we restrict our analysis to the autocorrelation function for prey density \( u \).

An immediate application of the standard definition leads to a problem. According to the usual approach, in case a dynamical variable \( \psi \) is a function of a variable \( \tau \) (which may have the meaning of time or position), the autocorrelation function is defined by the following expression:

\[
F(\xi) = \lim_{Z \to \infty} \frac{1}{Z} \int_0^Z \psi(\tau + \xi)\psi(\tau) d\tau .
\] (11.14)

In the problem under consideration, the prey density depends on two variables, position and time. Thus, applying definition (11.14) straightforwardly, we arrive at

\[
\tilde{F}(\xi, t) = \lim_{Z \to \infty} \frac{1}{Z} \int_0^Z u(x + \xi, t)u(x, t) dx .
\] (11.15)

Equation (11.15) has a few evident drawbacks. First, the autocorrelation function calculated according to (11.15) depends not only on the distance \( \xi \) but also on time. The situation when the properties of \( \tilde{F} \), considered as a function of \( \xi \), are explicitly time dependent appears rather exotic and makes the interpretation of the results highly difficult. On the other hand, since the problem is essentially nonstationary, it seems reasonable that a proper definition of the autocorrelation function should take into account both spatial and temporal aspects of the system dynamics. Another problem is that, in order to obtain reliable results in computer simulations, the value of \( Z \) in Equation (11.15) must be chosen sufficiently large. In practice, this means that the numerical grid must consist of at least a few dozen thousands of nodes (more likely, even tens of thousands), which would bring a considerable technical difficulty.

In order to overcome these problems, we use a modified definition of the autocorrelation function where the averaging over space is changed to the averaging over time:

\[
K(\xi) = \lim_{T \to \infty} \frac{1}{T} \int_0^T u(x_0 + \xi, t)u(x_0, t) dt .
\] (11.16)

Note that Equation (11.16) includes the standard definition as a particular case provided the system exhibits ergodic behavior. Let us also mention here that, although the value of \( K \) formally depends on the parameter \( x_0 \), the results of the numerical simulations do not show any dependence on \( x_0 \). This fact has a clear interpretation indicating the “statistical homogeneity” of the system dynamics.

The autocorrelation function \( K(\xi) \) calculated according to (11.16) in the case when the system is in the regime of spatiotemporal chaos is shown in Figure 11.9. Its properties (i.e., a fast decay for small \( \xi \) followed by some
irregular oscillations of relatively small amplitude) are typical for chaotic dynamics (Nayfeh and Balachandran, 1995). It should be mentioned here that the irregular oscillations of finite (nonzero) amplitude in $K(\xi)$ are the consequence of the finiteness of the averaging interval $T$; results of numerical experiments show that their amplitude tends to zero for increasing $T$. The dynamics of the system can now be characterized by the correlation length, its value being given by the first minimum of the autocorrelation function (Nayfeh and Balachandran, 1995; Abarbanel, 1996). For the parameters of Figure 11.9, $L_{corr} \approx 30$.

Existence of a finite correlation length means that the whole domain appears to be dynamically split into a number of subdomains, $N \approx L/L_{corr}$ and $N \approx (L/L_{corr})^2$ for one- and two-dimensional systems, respectively, their temporal behavior being virtually independent of each other. This self-organized splitting explains how the onset of chaos becomes possible in a time- and space-continuous prey–predator system: While the corresponding nonspatial dynamical system is of dimension 2 (in the sense of the dynamical systems theory), which makes chaos impossible, in the patchy regime its dimension grows with the number of subdomains and, therefore, can be much larger.

(It should be mentioned that, in case of the regular dynamics, the spatiotemporal behavior of the system (11.3)–(11.4) is highly correlated over the whole domain. Equation (11.16) then typically results in a monotonous,
slowly decreasing curve. Since the regime of smooth patterns is a process of slow relaxation to the homogeneous spatial distribution, the autocorrelation function gradually changes with time so that the degree of correlation between different points increases. Correspondingly, the actual shape of the autocorrelation function depends significantly on both the initial conditions and the averaging time; for that reason, we do not show it here. In any case, however, in the large-time limit, temporal oscillations become synchronized throughout the system and $K(\xi)$ approaches 1 for any finite $\xi$.

FIGURE 11.10: The value of the correlation length (#) in the chaotic prey–predator system (11.3)–(11.4) calculated for different parameter values along the line $h = p + 0.1$. Curve 1 shows the analytical prediction, see (11.26)–(11.27), and vertical line 2 shows the position of the Hopf-bifurcation point. With permission from Petrovskii and Malchow (2001a).

The next point is how $L_{corr}$ may depend on parameter values. A visual comparison between the spatial population distributions obtained for different parameters shows that they become “more patchy” when the parameters move away from the Hopf-bifurcation curve. In order to address this issue, the correlation length was calculated for the parameters varying along a certain line in the $(p, h)$ parameter plane. The results are shown in Figure 11.10. Indeed, it is readily seen that, although $L_{corr}$ changes only slightly in the intermediate parameter range, it tends to increase significantly when approaching the bifurcation point. This behavior also helps to explain why chaos is always suppressed in a close vicinity of the Hopf bifurcation; cf. the last paragraph in Section 11.1.1. As the system’s parameters get closer to the bifurcation point,
for any fixed length $L$ of the domain it appears that $L < L_{\text{corr}}$. This means that the oscillations get synchronized all over the domain and the system becomes dynamically equivalent to the corresponding homogeneous system where chaos is impossible.

So far as, we have shown that the onset of chaos may depend critically on the ratio $L/L_{\text{corr}}$. In Section 11.3, we will show that de-synchronization of population oscillations for $|r_1 - r_2| > L_{\text{corr}}$ due to transition to self-organized patchiness – onset of “biological turbulence” – has also crucial ecological implications. Altogether, it appeals for a method that would provide a fast and reliable estimate of the correlation length in a given system. Note that, theoretically speaking, its value can always be derived from the properties of a (exponentially) decaying correlation function. In turn, the spatial correlation function can be calculated either based on spatiotemporal data of field observations (cf. Ranta et al., 1997) or from a relevant model of the population dynamics. However, sufficiently detailed data are rarely available and obtaining $L_{\text{corr}}$ from a model usually takes a lot of computer simulations for each parameter set. Moreover, numerical data alone usually provide only limited information about the dependence of $L_{\text{corr}}$ on the parameters.

In the following section, we consider an alternative way to estimate the value of the correlation length. Using a general model of population dynamics and considering the stability of the population homogeneous distribution at equilibrium, we obtain a simple analytical formula for a certain “intrinsic length,” which distinguishes between subcritical perturbations (preserving homogeneity) and supercritical ones (driving the system to pattern formation). We will then show that this “intrinsic length” appears to coincide with $L_{\text{corr}}$ up to a numerical coefficient on the order of unity.

### 11.2.1 Intrinsic lengths and scaling

We begin with a general case of an $n$-species system. The mathematical model is given by the following equations:

$$
\frac{\partial U_i(R,T)}{\partial T} = D_i \nabla^2 U_i(R,T) + f_i(U), \quad i = 1, \ldots, n
$$

(cf. Chapter 2), where $U = (U_1, U_2, \ldots, U_n)$, $U_i$ is the density of the $i$th species, $R$ is the position in space, $T$ is the time, and the nonlinear functions $f_i$ describe the local “kinetics” of the system as given by multiplication, mortality, predation, etc.

We assume that the form of the functions $f_i$ provides the existence of at least one coexistence steady state $\bar{U} = (\bar{U}_1, \bar{U}_2, \ldots, \bar{U}_n)$, so that

$$
f_1(\bar{U}) = f_2(\bar{U}) = \ldots = f_n(\bar{U}) = 0,
$$

where $\bar{U}_i > 0$, $i = 1, \ldots, n$. In order to induce a nontrivial local dynamics of the system, we assume below that $n \geq 2$. Obviously, each steady state defined
by Equation (11.18) corresponds to the homogeneous stationary state of the system (11.17).

A coexistence state can be of a different type, either stable or unstable. We begin with the purely temporal stability of the spatially homogeneous system. Applying the standard linear analysis to the system (11.17) without diffusion, i.e., to

$$\frac{dU_i(t)}{dt} = f_i(U), \quad i = 1, \ldots, n,$$

(11.19)

stability of the given steady state means that all the eigenvalues of the system (11.19) linearized in the vicinity of $\bar{U}$ have negative real parts. Denoting $U_i(t) - \bar{U}_i = \epsilon_i(t)$, from system (11.19) we obtain

$$\frac{d\epsilon_i(t)}{dt} = a_{i1}\epsilon_1 + a_{i2}\epsilon_2 + \ldots + a_{in}\epsilon_n, \quad i = 1, \ldots, n,$$

(11.20)

where $a_{ij} = \frac{\partial f_i(\bar{U})}{\partial U_j}$ are the elements of the Jacobian of the kinetic system at the steady state. Then, the eigenvalues $\lambda_i$ ($i = 1, \ldots, n$) are given by the following equation:

$$\det(A - \lambda E) = 0,$$

(11.21)

where $A = (a_{ij})$ and $E$ is the identity matrix.

Since the coefficients $a_{ij}$ evidently depend on a number of parameters (such as the population growth and mortality rates, predation rates, etc.), the eigenvalues also depend on the parameter values. Let us consider the case when a given steady state can change its stability depending on the values of parameters. Loss of stability means that the real parts of some eigenvalues become positive when a point in the parameter space crosses a certain critical hypersurface. An example is given by the Hopf bifurcation. Denoting the maximum real part of the eigenvalues as $\bar{\lambda}$, the system becomes (linearly) unstable when $\bar{\lambda}$ becomes positive.

Note that stability of the system (11.19) means stability of the stationary homogeneous state $\bar{U}_i(x,t) = \bar{U}_i, \quad i = 1, \ldots, n$ of the system (11.17) with respect to small spatially homogeneous perturbations. However, the situation becomes somewhat more complicated when we consider spatially heterogeneous perturbations. Now, $\bar{U}_i(x,t) = \bar{U}_i + \epsilon_i(x,t)$, and, substituting it into (11.17), we obtain

$$\frac{\partial \epsilon_i(r,t)}{\partial t} = D_i \nabla^2 \epsilon_i(r,t) + a_{i1}\epsilon_1 + a_{i2}\epsilon_2 + \ldots + a_{in}\epsilon_n, \quad i = 1, \ldots, n.$$  

(11.22)

Considering a perturbation with a certain wavenumber $|k|$, i.e., $\epsilon_i = C_ie^{ikr}$
cos kr, \( i = 1, \ldots, n \), from (11.22) we arrive at the following system:

\[
\begin{align*}
(a_{11} - \nu - D_1 k^2) C_1 + a_{12} C_2 + \ldots + a_{1n} C_n &= 0, \\
a_{21} C_1 + (a_{22} - \nu - D_2 k^2) C_2 + \ldots + a_{2n} C_n &= 0, \\
&\vdots \\
a_{n1} C_1 + a_{n2} C_2 + \ldots + (a_{nn} - \nu - D_n k^2) C_n &= 0.
\end{align*}
\]

A nontrivial solution exists if and only if

\[
\det \left[(A - k^2 B) - \nu E\right] = 0, \tag{11.23}
\]

where \( B = (b_{ij}) \), \( b_{ij} = D_i \delta_{ij} \) and \( \delta_{ij} \) is the Kronecker symbol. Denoting, for convenience, \( \bar{\nu} = \max(\Re \nu_i) \) \((i = 1, \ldots, n)\), a stationary homogeneous distribution is linearly stable with respect to the heterogeneous perturbation with given wavelength \( l = 2\pi / |k| \) for \( \bar{\nu}(k) < 0 \) and unstable for \( \bar{\nu}(k) > 0 \).

Assuming here, for simplicity,\(^5\) that \( D_1 = \ldots = D_n = D \), it is readily seen that \( B = DE \) and Equation (11.23) coincides with (11.21) provided \( \lambda = \nu + D k^2 \). As an immediate consequence, it means that the maximum real parts of the eigenvalues of the homogeneous and heterogeneous problems are related by means of the following equation:

\[
\bar{\nu} = \bar{\lambda} - D k^2. \tag{11.24}
\]

Relation (11.24) has a clear meaning. When the steady state \( \bar{U} \) becomes locally unstable due to a change in parameter values, i.e., \( \bar{\lambda} \) becomes positive, it still remains stable in the distributed system with respect to heterogeneous short-wave perturbations, i.e., for

\[
|k| > k_0 = (\bar{\lambda} / D)^{1/2}. \tag{11.25}
\]

From (11.25), we arrive at the following formula for the critical wavelength:

\[
l_0 = 2\pi \left( D / \bar{\lambda} \right)^{1/2}. \tag{11.26}
\]

The value of \( l_0 \) may be regarded as a certain “intrinsic length” of system (11.17). It gives the upper limit of the system stability with respect to small spatially heterogeneous perturbations, i.e., the perturbations with wavelengths less than \( l_0 \) are damped by diffusion. Note that, in order to obtain (11.26), we did not make any specific assumptions about the local kinetics of the system, e.g., about the type of inter-specific interactions.

In case a perturbation exceeds the stability limits, the system may be driven into spatiotemporal chaos. Although the dynamics of the general system

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\(^5\)An extension of this analysis onto the case of unequal diffusion coefficients can be found in Petrovskii et al. (2003).
Biological turbulence

(11.17) remains to be investigated, existence of chaotic patterns was previously shown for a few particular cases such as a prey–predator system and a three competing species system. In the chaotic regime, the spatial properties of system (11.17) can be quantified by the correlation length $L_{\text{corr}}$ which has a meaning similar to the “intrinsic length” $l_0$, i.e., it gives the maximum distance between two positions in space where the diffusive coupling is still essential. Thus, the effect of diffusion is quantified by $l_0$ at the early stage of the system dynamics and by $L_{\text{corr}}$ at its later stage, after transients have disappeared. The assumption we are going to make now is that some properties of the early stage dynamics may stay important at the large-time stage as well.\(^6\) Then one can expect that the length $l_0$ remains intrinsic for the system after the onset of chaos. Since both $l_0$ and $L_{\text{corr}}$ are related to the same process, i.e., diffusive coupling, we then make a somewhat stronger hypothesis about the existence of a “scaling law:"

$$\frac{L_{\text{corr}}}{l_0} = c^*, \quad (11.27)$$

where $c^*$ is a “structural” coefficient of the order of unity, its value depending on the type of the interspecific interactions but not on particular parameter values once the system is specified.

The above speculations can hardly be accepted as a proof, though. Therefore, in order to test hypothesis (11.27), now we are going to consider a few particular cases of the system (11.17) where dependence of the maximum real part of the eigenvalues $\bar{\lambda}$ on system parameters can be followed explicitly.

**Prey–predator system.** For a prey–predator system described by Equations (11.3)–(11.4), the real part of the eigenvalues (in dimensionless units) of the system linearized in the vicinity of ($\bar{u}, \bar{v}$) is given by the following equation:

$$\bar{\lambda} = \frac{\text{tr}A}{2} \text{ where } \text{tr}A = \frac{p}{1-p} [(1-p) - h(1+p)], \quad (11.28)$$

where $A$ is the matrix of the linearized system. Figures 11.10 and 11.11 show $L_{\text{corr}}$ obtained from numerical data (as the position of the first minimum of the spatial autocorrelation function) and $l_0$ calculated analytically from (11.26) and (11.28). It is readily seen that the ratio $L_{\text{corr}}/l_0$ remains constant (up to small fluctuations most likely caused by the computational error) if $\bar{\lambda}$ is sufficiently small.

**Three competing species system.** Another example of a biological system showing the formation of chaotic spatiotemporal patterns is given by a community of three competitive species; see Section 10.3. For simplicity, here we

\(^6\)Although this assumption may seem exotic, a recent paper by Neubert et al. (2002) reveals another link between the early stage dynamics and the large-time dynamics of a diffusion–reaction system.
restrict our analysis to the special case of cyclic competition. The maximum real part of the eigenvalues of the linearized dimensionless system is then given by the following formula:

$$\lambda = \frac{1}{1 + \alpha + \beta} \left( -1 + \frac{\alpha + \beta}{2} \right).$$ \hspace{1cm} (11.29)

When $\alpha + \beta > 2$, the steady state becomes locally unstable and perturbations of the homogeneous initial state can drive the system into spatiotemporal chaos (Petrovskii et al., 2001). Note that the structure of the phase space of system (10.54)–(10.56) is different compared to the prey–predator system: In the case of cyclic competition, no stable limit cycle exists and the attractor is given by a heteroclinic trajectory connecting the three unstable “one-species-only” states.

Table 11.1 and Figure 11.11 show the correlation length $L_{corr}$ calculated for different parameters $\alpha$ and $\beta$ as well as the corresponding value of $l_0$ given by (11.26) and (11.29). Apparently, both quantities exhibit essentially the same
TABLE 11.1: Relation between the (dimensionless) values of the correlation length $L_{corr}$ and the intrinsic length $l_0$ in the three competitive species community.

With permission from Petrovskii et al. (2003).

<table>
<thead>
<tr>
<th>parameters</th>
<th>$\alpha$</th>
<th>$\beta$</th>
<th>$\lambda$</th>
<th>$l_0$</th>
<th>$L_{corr}$</th>
<th>$L_{corr}/l_0$</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\alpha = 1.20$, $\beta = 0.90$</td>
<td>0.0161</td>
<td>49</td>
<td>30 ± 2</td>
<td>0.61</td>
<td></td>
<td></td>
</tr>
<tr>
<td>$\alpha = 1.30$, $\beta = 0.80$</td>
<td>0.0161</td>
<td>49</td>
<td>30 ± 2</td>
<td>0.61</td>
<td></td>
<td></td>
</tr>
<tr>
<td>$\alpha = 1.15$, $\beta = 0.90$</td>
<td>0.0082</td>
<td>69</td>
<td>41 ± 2</td>
<td>0.59</td>
<td></td>
<td></td>
</tr>
<tr>
<td>$\alpha = 1.22$, $\beta = 0.80$</td>
<td>0.0033</td>
<td>109</td>
<td>65 ± 2</td>
<td>0.60</td>
<td></td>
<td></td>
</tr>
<tr>
<td>$\alpha = 1.07$, $\beta = 0.95$</td>
<td>0.0033</td>
<td>109</td>
<td>67 ± 2</td>
<td>0.61</td>
<td></td>
<td></td>
</tr>
<tr>
<td>$\alpha = 1.21$, $\beta = 0.80$</td>
<td>0.0017</td>
<td>154</td>
<td>93 ± 5</td>
<td>0.60</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Type of dependence on the parameters, so that their ratio stays constant. In particular, the scaling hypothesis (11.27) succeeds in predicting that the correlation length depends on the sum $\alpha + \beta$ rather than on $\alpha$ and $\beta$ separately, and that the value of $L_{corr}$ increases when $\alpha + \beta$ tends to 2.

A two-species chemical system. The two cases considered above, although essentially different in the type of interspecific interactions, still exhibit a certain mathematical similarity. Namely, in both cases there is only one stationary coexistence state. Meanwhile, it is well known that the existence of another steady state, particularly, the existence of a saddle-point, can change the dynamics significantly (Rai and Schaffer, 2001). A question thus arises whether the validity of Equation (11.27) is restricted to a specific structure of the phase space of the system, or it may have a wider application.

In order to address this issue, now we consider an example of different origin, i.e., the Gray–Scott model of an autocatalytic reaction in an open one-dimensional flow reactor:

\[
\frac{\partial u}{\partial t} = \frac{\partial^2 u}{\partial x^2} + F(1 - u) - uv^2 , \quad (11.30)
\]

\[
\frac{\partial v}{\partial t} = \frac{\partial^2 v}{\partial x^2} + uv^2 - (F + k)v \quad (11.31)
\]

(Gray and Scott, 1990) with properly chosen dimensionless variables (cf. Pearson, 1993). Now $u$ and $v$ are the concentrations of the substrate and the autocatalyst, respectively, $F$ is the flow rate, and $k$ is the effective rate constant of the decay of the autocatalyst. Although the system (11.30)–(11.31) does not immediately apply to a community of biological species, it provides a convenient test to check the generality of our scaling hypothesis (11.27).

One can readily see that, under condition $d = 1 - 4(F + k)^2/F > 0$, there are three spatially homogeneous steady states in the system (11.30)–(11.31): “substrate-only” state $(1,0)$, “substrate-dominated” state $(u_s, v_s)$

and “autocatalyst-dominated” state \((u_a, v_a)\), where

\[
\begin{align*}
    u_a &= \frac{1 + \sqrt{d}}{2}, \quad v_a = \left( \frac{F}{F + k} \right) \frac{1 - \sqrt{d}}{2}, \\
    u_a &= \frac{1 - \sqrt{d}}{2}, \quad v_a = \left( \frac{F}{F + k} \right) \frac{1 + \sqrt{d}}{2}.
\end{align*}
\]  

(11.32)  

(11.33)

When crossing the critical curve \(d = 0\) in the \((k, F)\) plane towards smaller values of \(k\), the two nontrivial states appear through a saddle-node bifurcation, the autocatalyst-dominated state being an unstable node.

The substrate-only state is always a stable node and the substrate-dominated state is always a saddle-point. A change in the local dynamics can be associated with the change of the type of the autocatalyst-dominated state, first of all, with the change of its stability. It is straightforward to see that the maximum real part of the eigenvalues at the autocatalyst-dominated state is
given by the following equation:

\[ \tilde{\lambda} = \frac{1}{2} (k - v^2_a) . \]  

(11.34)

The change of stability takes place when \( \tilde{\lambda} = 0 \), which, after a little algebra, takes the following form:

\[ \frac{k - F}{k + F} = \sqrt{d} . \]  

(11.35)

The Hopf bifurcation that takes place when crossing the curve (11.35) is predicted to be supercritical for \( k < k_{cr} \) (where \( k_{cr} \) is estimated as about 0.035), and only in this case does a stable limit cycle appear (Rasmussen et al., 1996). Otherwise, no limit cycle arises and any trajectory starting in the vicinity of the autocatalyst-dominated state, after a number of twists of increasing amplitude, is finally attracted to the substrate-only state. Therefore, the structure of the local phase plane of the Gray–Scott model is essentially different from the prey–predator system (11.3)–(11.4).

For those parameter values when the autocatalyst-dominated state is unstable, the system (11.30)–(11.31) can exhibit spatiotemporal chaos (Merkin et al., 1996; Davidson, 1998). In the chaotic regime, the spatial and temporal behavior of the reacting species is qualitatively similar to the behavior of the biological species; see Figure 11.12. Although our main interest is focused on the dynamics of biological communities, the model (11.30)–(11.31) can be used for testing the scaling hypothesis (11.27). Figure 11.11 shows the value of \( L_{corr} \) obtained in numerical simulations as well as the intrinsic length \( l_0 \). One can see that the ratio \( L_{corr}/l_0 \) stays constant, which “proves” the relation (11.27).

Thus, the results of our numerical simulations of chaotic spatiotemporal dynamics in different systems show that the hypothesis (11.27) about the relation between the intrinsic length \( l_0 \) and the correlation length \( L_{corr} \) of the system is valid in a wide variety of cases; see Figure 11.11. We also want to mention that relation (11.27) remains valid for populations with different diffusivity, although the analytical expression for \( l_0 \) is less elegant in that case (Petrovskii et al., 2003).

An immediate consequence of the above conclusion is that the following equation can be used in order to estimate the value of \( L_{corr} \):

\[ L_{corr} = c^* l_0 , \]  

(11.36)

where \( l_0 \) is given by Equation (11.26) and \( c^* \) is a constant on the order of unity, e.g., \( c^* \approx 1 \) for the prey–predator system, \( c^* \approx 0.6 \) for the system of three competitive species, and \( c^* \approx 0.47 \) for the autocatalytic chemical system.

When the parameters approach the bifurcation point where the corresponding steady state becomes stable, \( \lambda \) tends to zero. Correspondingly, \( l_0 \) tends to infinity and (11.36) predicts that \( L_{corr} \) grows unboundedly. This is in good
agreement with our numerical results; see Figure 11.11. There is, however, a certain discrepancy between $L_{corr}$ and $l_0$ that is not described by the linear relation (11.27) further away from the bifurcation point, i.e., in the parameter range where $\bar{\lambda}$ becomes sufficiently large (correspondingly, $L_{corr}$ sufficiently small). It may indicate that $c^*$ is, in fact, a function of the system parameters with scaling properties so that $c^* \approx const > 0$ for $\bar{\lambda} \ll 1$.

11.3 Ecological implications

The importance of space in ecology has been increasingly recognized over the last two decades. When considered in space and time, ecological interactions may exhibit considerably different properties compared to the corresponding nonspatial systems. We have already demonstrated that a spatially homogeneous distribution of interacting species often appears to be unstable to heterogeneous perturbations and is prone to generating spatiotemporal patterns. In case a system’s kinetics is oscillatory, these patterns are remarkably irregular in space and chaotic in time. In order to study this phenomenon of “biological turbulence,” a prey–predator system was used as a paradigm. However, self-organized patterning is clearly a more general phenomenon and not restricted to prey–predator interactions; in particular, extension of the main results has been made onto a system of competing species. Moreover, the prey–predator interactions are among the most common ones in population biology and are present in virtually every population community. Therefore, we expect that the mechanism of self-organized patchiness considered in the previous sections is likely to have a variety of profound implications for population dynamics and ecology. In this section, we will give two examples where the properties of biological turbulence enhance our understanding of ecological dynamics and may provide a solution for some long-standing ecological problems.

11.3.1 Plankton patchiness on a biological scale

Probably the most notorious example of ecological patterning, which has been studied intensively for several decades, is the heterogeneity of aquatic populations. In particular, the horizontal spatial distribution of plankton in oceans, seas, and large lakes is remarkably inhomogeneous. This phenomenon, which is often referred to as “plankton patchiness,” has fundamental implications for almost every aspect of marine ecosystems organization (Valiela,
Correspondingly, a number of attempts have been made to explain the plankton patterns by means of reducing them to the heterogeneity of the temperature field (Denman, 1976), to specific ocean hydrodynamics (Wyatt, 1973; Abbott and Zion, 1985), or turbulent mixing (Platt, 1972; Abraham, 1998; Neufeld, 2001; Reigada et al., 2003).

However, the problem as a whole seems to be much more complicated. Prominent spatial heterogeneity of plankton distribution occurs on different scales ranging from a few centimeters to thousands of kilometers. Analysis of field data (Powell et al., 1975; Weber et al., 1986) leads to the conclusion that the plankton patchiness on different scales is caused by the processes of different nature (Levin, 1990; Powell, 1995). On a scale of dozens of meters and less, plankton spatial distribution is indeed mainly controlled by turbulence (cf. Platt, 1972; Powell et al., 1975). On a scale of hundreds of kilometers and more, the heterogeneity of plankton distribution follows the heterogeneity of the temperature field (Weber et al., 1986). However, on the intermediate “biological scale,” i.e., from hundreds of meters to dozens of kilometers, plankton patchy distribution is significantly affected by the interplay between the biological processes (such as population growth, predation, etc.) and the species dispersal, mostly due to turbulent mixing; see Levin and Segel (1976); Steele and Henderson (1992b); Powell (1995); Pascual and Caswell (1997); also see recent reviews by Medvinsky et al. (2002) and Martin (2003) for further references.

Remarkably, there is an opinion (Valiela, 1995) that the phenomenon of plankton patchiness is a manifestation of marine ecosystem’s spatiotemporal self-organization, which is beneficial for the community as a whole. However, what particular mechanisms are behind this self-organization? Here we hypothesize that the dynamics of a plankton system is to a large extent controlled by the trophic interaction between phyto- and zooplankton acting as prey and predator, respectively. Consequently, we can apply the results obtained above for the prey–predator system and associate the chaotic spatiotemporal patterns with the plankton patterns on the biological scale. Indeed, there is a striking visual coincidence between the irregular spatial patterns generated in a prey–predator system (cf. Figure 11.8) and the patterns of spatial plankton distribution usually observed in the ocean; see Figure 11.13. It should also be mentioned that, although conclusive evidence is still lacking, there is a growing number of indications of chaos in the dynamics of aquatic populations (Medvinsky et al., 2002).

A visual coincidence can hardly be regarded as an acceptable proof, though. However, our hypothesis of the plankton patchiness origin can be tested in a more quantitative way. Recall that the biological scale has lower and upper bounds. The lower bound is called the KISS length; it corresponds to the spatial scale where the distractive impact of turbulent eddies on plankton patches is balanced by their growth due to population multiplication. The value of the KISS length (being on the order of a hundred meters) has been estimated consistently from a few different approaches (Kierstead and
Slobodkin, 1953; Powell et al., 1975; Denman and Platt, 1976; Steele, 1978; Powell and Okubo, 1994). However, the origin of the upper bound remains poorly understood. Although suggestions have been made that it may reflect the impact of zooplankton on the system dynamics (cf. Steele, 1978), a particular mechanism has never been identified. Now, if we assume that plankton patchiness is a manifestation of the biological turbulence resulting from phytoplankton-zooplankton (prey–predator) interactions, the value that has a meaning similar to the upper bound of the biological scale is the correlation length.

In order to calculate $L_{\text{corr}}$, we use Equations (11.36), (11.26), and (11.28). Referring to the parameter estimates provided by different authors (Nisbet et al., 1991; Truscott and Brindley, 1994; Sherratt, 2001), we choose $\alpha = 1.0$ day$^{-1}$, $\gamma = 0.7$ day$^{-1}$, $\mu = 0.05$ day$^{-1}$, $\kappa = 0.15$, and $H/K = 0.3$ as typical values. The turbulent diffusivity is roughly estimated as $D = 10^5 \text{cm}^2\text{sec}^{-1}$ (Nihoul 1980). Using these values, we arrive at $L_{\text{corr}} \approx 30 \text{ km}$ (cf. Petrovskii et al., 2003), which is in a very good agreement with the estimates of the
The above agreement is encouraging; however, we also want to mention that a marine ecosystem is an extremely complex object where a wide range of complicated physical processes interplay with equally complicated biological ones. Therefore, it would hardly be realistic to relate plankton patchiness to a single reason. The “biological turbulence” arising due to the coupling between spatial mixing and local phyto-zooplankton oscillations is likely to manifest itself under conditions of statistically homogeneous and isotropic turbulent ocean flows. In case the ocean hydrodynamical flows exhibit some sort of anisotropy, other mechanisms may apply, creating an alternative or complementary framework for the plankton patchiness. In particular, shear flows at the edges of large ocean currents may also result in pattern formation on the scale of dozens of kilometers (Biktashev et al., 1999).

Moreover, the horizontal spatial mixing in plankton systems in oceans, seas, and large lakes takes place mainly due to the turbulent eddy diffusivity. Contrary to the usual Brownian diffusion, the intensity of the turbulent diffusion typically increases with the scale of the process because of the impact of larger eddies (Okubo, 1971). This can be quantified in terms of turbulence spectrum, i.e., velocity variance per unit wavenumber. While a conservative passive tracer such as water temperature or salinity has the spatial spectrum coinciding with that of the turbulent flow, the spectra of phyto- and zooplankton patchiness are different on the biological scale (Weber et al., 1986), presumably due to the impact of the local population growth (Levin, 1990). Having considered a phyto-zooplankton system near its steady equilibrium, Vilar et al. (2003) showed theoretically that, by means of including details of the turbulent spectrum into the model, the spectral properties of Equations (11.1)–(11.2) can indeed be made very similar to those observed in field data.

Finally, we mention that the whole concept of turbulent “diffusion” is a sort of mean-field approach containing very little information about the inherent stochastic fluctuations of the turbulent flows. Therefore, solutions of the diffusion–reaction models should be regarded as population densities averaged over the corresponding statistical ensemble rather than snapshots of actual plankton distributions. In order to make an insight into the transient spatial structures associated with turbulent pulsations, one should go beyond the mean-field approximation (Abraham, 1998), e.g., considering turbulent transport as “stochastic advection” (Neufeld, 2001).
11.3.2 Self-organized patchiness, desynchronization, and the paradox of enrichment

The paradox of enrichment (Rosenzweig, 1971), when an increase in the nutrient input into a prey–predator system destabilizes the community functioning and may even lead to extinction of the species, has been a challenge for a few generations of ecologists; cf. Gilpin (1972); May (1972a); Luckinbill (1974); Brauer and Soudack (1978); Jansen (1995); Abrams and Waters (1996); Bohannan and Lenski (1997); Nisbet et al. (1997); Genkai-Kato and Yamamura (1999); Holyoak (2000); Petrovskii et al. (2004); Jensen and Ginzburg (2005); and Morozov et al. (2007).

Theoretical arguments behind the paradox are simple, clear, and apply to a rather general case. Let us consider the following prey–predator system:

$$\frac{dU}{dt} = f(U) - g(U) V , \quad \frac{dV}{dt} = \kappa g(U) V - MV , \quad (11.37)$$

where all terms have their usual meanings. For biological reasons, $g(U)$ is a monotonously increasing function; additionally, we assume that $g(U)$ increases linearly for small $U$, i.e., $g(U) = \text{const} \cdot U + o(U)$ (cf. Holling type II). We also assume that prey growth is not damped by the Allee effect; therefore, $f(U)$ is a monotonously decreasing function turning to zero for $U = K$, where $K$ is the carrying capacity. These assumptions are not necessary conditions for the paradox of enrichment to occur, but they make analysis easier and more straightforward.

The coexistence steady state of the system is given by the intersection of the isoclines. Moreover, it is readily seen that the steady state is stable when the intersection takes place on the right of the hump (i.e., where function $f(U)U/g(U)$ decreases) and it is unstable on the left of the hump (where $f(U)U/g(U)$ increases). For the parameter values when it is unstable, it is surrounded by a stable limit cycle (see Figure 11.14, top), which appears through the Hopf bifurcation when the steady state passes the hump from right to left.
Now, how do the system properties change in response to enrichment? System eutrophication is thought\(^8\) to increase the carrying capacity \(K\). Since \(K\)

\[^8\] It may also increase the linear per capita growth \(r\), but see the comments after Equations (11.40)–(11.41).

**FIGURE 11.14**: A sketch of the system’s response to enrichment: (a) A typical phase plane of a prey–predator system. The dashed-and-dotted lines show the null-isoclines of the system, and the solid curve shows a stable limit cycle. The curves are obtained for the nonspatial version of (11.40)–(11.41) with parameters \(k = 2\), \(m = 0.6\), and \(h = 0.43\). (b) Periodical oscillations of the prey density; enrichment of the system (when \(K\) increases instantaneously by about 50 per cent at \(t = 200\)) results in oscillations of considerably larger amplitude.
is a natural scaling factor for the prey density, an increase in $K$ obviously “pulls” the whole plot of the first isocline to the right; in particular, the position of the hump moves to the right as well. Then, for any fixed position of the vertical isocline, a sufficiently large increase in $K$ will inevitably destabilize the steady state. As a result, the population densities start oscillating in time. The dynamics that may then lead to species extinction corresponds to the case when the trajectory in the phase space of the system comes close to the boundary of the biologically meaningful domain $U \geq 0$, $V \geq 0$. Remarkably, a further increase in $K$ leads to a fast increase in the size of the limit cycle, so that the minimum value of the population density reached in the course of oscillations eventually tends to zero (May, 1972b; Gilpin, 1972); see Figure 11.14, bottom. In terms of the dynamics of real ecosystems, as the minimum value of the population density decreases, population extinction becomes more probable due to stochastic environmental perturbations (Goel and Richter-Dyn, 1974; Lande, 1993). Moreover, the situations when the minimum population size falls to a value $\ll 1$ should be regarded as actual extinction even without any stochasticity.

The apparent contradiction between the intuitively expected positive impact of increasing nutrient input and its actual destabilizing effect inspired a number of modifications of the original prey–predator model. For instance, it was shown that enrichment of a prey–predator community does not necessarily diminish the minimum value of oscillating population densities in the cases of either the existence of invulnerable individuals within the prey population (Abrams and Waters, 1996) or in the presence of an alternative “unpalatable” prey (Genkai-Kato and Yamamura, 1999). Another way to increase the system’s stability is to include ratio-dependence into the predation term (cf. Jensen and Ginzburg, 2005). However, these modifications have left open the question of whether the simplest one-predator–one-prey system is intrinsically unstable with respect to eutrophication. Although this kind of system response to eutrophication is not commonly seen in nature (McCauley and Murdoch, 1990), the self-regulating mechanisms of the system are not always clear. Furthermore, extinction of a prey–predator community following system eutrophication has been seen in some biological data (Luckinbill, 1974; Bohannan and Lenski, 1997).

The theoretical results mentioned above were obtained under the assumption that the interacting populations were homogeneous in space. A salient point of contemporary ecology has become the growing understanding that the dynamics of any biological community takes place not only in time but also in space, and that the properties of a spatiotemporal system can be essentially different from those of its nonspatial counterpart. The impact of space on the persistence of enriched prey–predator systems was indeed seen in laboratory experiments (Luckinbill, 1974), although the corresponding mechanisms remained unclear. More recently, it has been shown both in experimental studies (Holyoak, 2000) and theoretically (Jansen, 1995, 2001) that the existence of a predefined patchy spatial structure makes a prey–predator system
system less prone to extinction. In a spatially structured metapopulation, the temporal variations of the density of different subpopulations can become asynchronous and the events of local extinction can be compensated for due to re-colonization from other sites (Allen et al., 1993).

**FIGURE 11.15:** Spatial (a, c) and temporal (b, d) variations of the population densities in case of regular (a, b) and chaotic (c, d) dynamics as described by Equations (11.40)–(11.41). Snapshots of the species distribution in (a) and (c) (solid line for prey, dotted for predator) are taken at moment $t = 2000$. The corresponding initial conditions were chosen in the form of constant-gradient perturbation of the coexistence steady state, i.e., $u(x, 0) = \bar{u}$, $v(x, 0) = \bar{v} + \nu x + \delta$, with parameter values $\nu = 10^{-5}$ and (a) $\delta = 0.01$ and (c) $\delta = -0.005$. Other parameters are $k = 2.0$, $m = 0.6$, and $h = 0.43$ (with permission from Petrovskii et al., 2004).

Our goal here is to investigate the impact of enrichment on persistence / extinction of the species in a model prey–predator system taking into account the existence of the two different types of dynamics; see Figure 11.15. The idea is that, in the case that the temporal variations of the species are synchronized or strongly correlated over the whole space, as it takes place in the regular
regime, a fall of the population density to a dangerously small value takes place almost simultaneously throughout the system and thus the species may go extinct without any chance of subsequent re-colonization (Allen et al., 1993). On the contrary, if the temporal variations are desynchronized, as it takes place in the chaotic regime, a local extinction does not necessarily lead to global extinction because the empty sites can be re-colonized via “diffusion” of the individuals from other sites. A related but somewhat more general question is whether these two types of dynamics are robust with respect to the system’s enrichment.

In order to make a further insight into this problem, we use the prey–predator model from the preceding sections:

\[
\frac{\partial u}{\partial t} = \frac{\partial^2 u}{\partial x^2} + u(1 - u) - \frac{u}{u + h} v, \quad (11.40)
\]

\[
\frac{\partial v}{\partial t} = \frac{\partial^2 v}{\partial x^2} + k \frac{u}{u + h} v - m v. \quad (11.41)
\]

Note that the system (11.40)–(11.41) is in dimensionless form where \( h, k, \) and \( m \) are expressed through the original parameters as \( h = H/K, k = \kappa A/\alpha, \) and \( m = M/\alpha; \) see Section 10.1 for details. Enrichment of the system leads to an increase in the prey carrying capacity \( K \) and thus to a decrease in \( h. \) A reasonable biological alternative would be an increase in the prey linear growth rate \( \alpha \) and thus a decrease in \( k \) and \( m. \) However, in Section 10.1 we showed that variations in \( k \) do not have any significant impact on the stability of the steady state. In its turn, a decrease in \( m \) has an impact similar to that of a decrease in \( h \) (a point in the parameter plane of the system moves further away from the Hopf bifurcation curve; see Figure 10.1). Therefore, for the sake of simplicity, here we restrict our consideration to the case when enrichment only affects \( h. \)

In order to distinguish between different types of system dynamics, we use the spatially averaged population density as a convenient “measure” describing correlations between the temporal variations in population density at different positions in space. Indeed, in case temporal variations are strongly correlated throughout the domain (cf. Figure 11.15a), the amplitude of the variation of the average density is close to the amplitude of the local variations, which tends to grow with the system eutrophication. However, if the population oscillations are not correlated throughout the domain (cf. Figure 11.15c), the amplitude of the variation of the average density is significantly smaller than that of the local density.

We begin with the case when eutrophication occurs instantaneously so that the value of \( h \) changes from an initial value \( h_0 \) to a new value \( h_1 = h_0 - \Delta h \) at a certain moment \( t_0. \) We assume that, prior to eutrophication, the system is in the regime of regular spatiotemporal oscillations and use the spatial distribution shown in Figure 11.15a as the initial condition. The system dynamics is then studied through extensive numerical simulations, with the values of \( r, h_0, \) and \( \Delta h \) varying in a wide range.
Some typical results are presented in Figure 11.16. Only temporal variations of the prey density are shown; the density of predator exhibits qualitatively similar behavior. Figures 11.16a and 11.16b show the system response after $h$ changed from $h_0 = 0.43$ to $h_1 = 0.3$ at the moment $t_0 = 200$. The amplitude of the local population oscillations increases considerably, so that the prey density periodically falls to a very small value. Interestingly, however, the minimum value of oscillating average prey density appears to be somewhat larger than it was before eutrophication: As a result of enrichment, the population fluctuations over the domain become less correlated. According to the above arguments, it makes global species extinction less probable. For comparison, enrichment in the corresponding nonspatial system would inevitably lead to oscillations of larger amplitude with a smaller minimum value of the
population density; therefore, the impact of space has a clear positive effect on species survival.

Remarkably, the positive impact of space becomes much more prominent for eutrophication of a larger magnitude, i.e., for larger values of $\Delta h$. Figures 11.16c and 11.16d show the species temporal variations when at $t_0 = 200$ the value of $h$ decreases from $h_0 = 0.43$ to $h_1 = 0.16$. The system dynamics now changes from regular to chaotic, the properties of the species spatial distribution undergoing the corresponding changes (cf. Figures 11.15a and 11.15c). Although the local population density may fall to a very small value (see Figure 11.16c), the amplitude of the temporal variation of the average density decreases significantly; this reflects the fact that the oscillations of the species density at different positions in space become desynchronized. Desynchronized dynamics practically excludes the situation when the species density falls to a small value simultaneously at each position in space. In fact, the minimum value of the average population density now appears to be considerably larger than it was before eutrophication. Thus, following the above arguments, enrichment has made the probability of species extinction smaller, not larger.

Besides instantaneous changes, we also consider the case when eutrophication takes place gradually, during a certain finite time interval $\Delta t$. For simplicity, we assume that the value of $h$ changes from $h_0$ to $h_1$ linearly with time between $t_0$ and $t_1 = t_0 + \Delta t$. Typical results are shown in Figure 11.17. It is readily seen that, in this case, the type of system response depends also on the value of $\Delta t$. Simulations show that, while for small $\Delta t$ the system response is the same as for instantaneous enrichment (i.e., transition to chaos for sufficiently large $\Delta h$; cf. Figures 11.16d and 11.17, top), for sufficiently large $\Delta t$ the dynamics of the system remains regular; see Figure 11.17, bottom. Contrary to the case of instantaneous enrichment, the minimum value of the average population density now decreases drastically. That leads to a rather counter-intuitive conclusion that a fast eutrophication may appear to be less dangerous for the community functioning than a slow one.

The above results about the role of space and self-organized patchiness in resolving the paradox of enrichment are sketched in Figure 11.18, with the curves showing the “probability” (in a loose sense) of population extinction. Here curve 1 corresponds to the spatially homogeneous cases, i.e., to the classical paradox of enrichment. A monotonous increase in the probability of global extinction along with the magnitude of enrichment follows from the fact that the minimum average population density decreases when $\Delta h$ grows. The probability of extinction becomes dangerously high (above the horizontal dotted line) when the magnitude of enrichment exceeds a certain critical value $\Delta h_1$.

Curve 2 shows how the situation changes in a spatially heterogeneous system in the case of a fast (instantaneous) enrichment. Prior to eutrophication, the system is assumed to be in the regime of regular oscillations. A sudden increase in $K$ (decrease in $h$) makes the population oscillation less correlated
and thus increases the minimum average population density; hence the probability of global extinction decreases. A sufficiently large drop in \( h \) drives the system into spatiotemporal chaos with an even larger value of minimum average density and, correspondingly, smaller probability of extinction.

Curve 3 outlines a typical response of a heterogeneous system to a slow (gradual) enrichment. In this case, a decrease in \( h \) is accompanied by a decrease in the minimum average density; therefore, the larger the enrichment
FIGURE 11.18: A sketch of the extinction probability dependence on the magnitude of a system’s enrichment for spatially homogeneous (curve 1) and spatially heterogeneous (curves 2 and 3) systems; curves 2 and 3 show the system response for fast (instantaneous) and slow enrichments, respectively.

Apart from the fact that the impact of space denounces the negative effect of eutrophication on global species persistence, the above results clearly show that enrichment can drive the system to spatiotemporal chaos. An issue of interest is then to understand how this type of system response can be modified by a variation in the length $L$ of the spatial domain. Since the system possesses an intrinsic length $L_{\text{corr}}$, one can expect that the properties of the system dynamics can be somewhat different for different values of the ratio $L/L_{\text{corr}}$, i.e., for different $L$ provided all other parameters are fixed. In order to address this issue, we have repeated simulations for various $L$. The results are summarized in Table 11.2. It is readily seen that regular dynamics in small habitats tends to be more stable to enrichment than it is in large ones. This is in a good agreement with more general conclusions about possible transitions to chaos in the spatially explicit prey–predator system; see Section 11.1.1 and also the paragraphs preceding Section 11.2.1. Consequently, since transition
TABLE 11.2: Chaos versus order. Pluses mark the cases when enrichment drives the system to spatiotemporal chaos, and minuses stand for the cases when the dynamics remains regular; \( L \) is the size of the domain. With permission from Petrovskii et al. (2004).

<table>
<thead>
<tr>
<th>( L ), dimensionless</th>
<th>( \Delta h ), dimensionless</th>
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</thead>
<tbody>
<tr>
<td>300</td>
<td>– – – – – – +</td>
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<tr>
<td>500</td>
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<tr>
<td>1000</td>
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<td>1500</td>
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</tr>
<tr>
<td>2000</td>
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</tr>
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The above analysis corresponds to the case when prior to eutrophication the system dynamics is regular. Now, what would be the difference if the system had already been in the regime of spatiotemporal chaos? In the previous sections we have shown that chaos is robust to parameter perturbations, unless either the steady state becomes stable and the local population oscillations are totally suppressed or the corresponding point in the parameter space comes very close to the Hopf-bifurcation curve so that \( L_{corr} > L \). This is clearly not the case with enrichment when \( h \) is decreasing, the system goes further away from the Hopf bifurcation, and the local oscillations become more prominent. Therefore, intuitively, it is rather obvious that a chaotic prey–predator system is unlikely to be sensitive to enrichment. Indeed, computer simulations show that the regime of spatiotemporal chaos is stable with respect to a decrease in \( h \). In this case, although eutrophication of the system increases the amplitude of local population oscillations, it does not increase the danger of global species extinction in any significant way because the dynamics of the system remains chaotic and the population oscillations throughout the domain remain uncorrelated.

11.4 Concluding remarks

In this chapter, we have shown that the onset of “biological turbulence”–the formation of a prominent irregular spatial structure with the population oscillations desynchronized in space and chaotic in time–is an inherent property of a community of interacting populations. It should not be necessarily
attributed to any environmental forcing (such as any predefined spatial heterogeneity), any special type of the initial conditions, or any particular type of interspecific interactions. The only necessary conditions that we were able to identify are that the system should possess a coexistence steady state and the corresponding system’s kinetics must be oscillatory in a “broad sense,” i.e., the coexistence state should be an unstable focus. The regular dynamics then appears to be unstable with respect to supercritical (but still very small) perturbations that drive the system into spatiotemporal chaos.

Note that, although conclusive evidence of chaos in the dynamics of real ecological populations is still lacking, chaotic population dynamics under laboratory conditions have indeed been observed (Costantino et al., 1997; Dennis et al., 2001; Becks et al., 2005; Cushing et al., 2003). The factors that may prevent observation of chaos in ecological data are not well understood yet. One plausible explanation is that the stationary ecological time series appropriate for the analysis simply cannot be made long enough due to the transient nature of the environmental conditions (Hastings, 2001, 2004). Nevertheless, here we have shown that spatiotemporal dynamics of a population community is intrinsically chaotic. This is likely to have crucial ecological implications (Scheffer, 1991b; Hastings et al., 1993): Chaos means sensitivity to the initial condition, and that makes an accurate long-term forecast of ecological dynamics principally impossible. For instance, we can calculate an average/expected size of a single population patch and the expected time of its existence, but we are not able to predict when and where it will actually appear; “we can indicate the range of things that can happen, but cannot predict when they will happen” (Scheffer, 1991b).

A more specific implication of our results concerns the choice of an adequate model to study chaotic dynamics. Namely, many theoretical results concerning spatiotemporal chaos in a system of interacting chemical or biological species have been obtained in terms of so-called $\lambda$-$\omega$ systems (the complex-variable analogue of the $\lambda$-$\omega$ system is called the Ginzburg–Landau equation); see Kopell and Howard (1973); Kuramoto (1984); Bohr et al. (1998), also Sherratt (2001) and the references therein. Briefly, a $\lambda$-$\omega$ system arises as the first order approximation of a general diffusion–reaction system considering the size of the limit cycle as a small parameter. An advantage of this approach is that $\lambda$-$\omega$ systems often appear to be mathematically much simpler than the original diffusion–reaction systems, especially in the case when the original system consists of many species. The $\lambda$-$\omega$ systems are formally valid for parameter values near the Hopf bifurcation, but there is also a strong opinion that they provide an adequate description of the corresponding diffusion–reaction systems in a much wider parameter range. Our results, however, seem to indicate that spatiotemporal chaos in the $\lambda$-$\omega$ systems is just a mathematical artifact without any clear relation to reality. It follows from Equations (11.26) and (11.36), as well as from simulations (e.g., see Figures 11.10 and 11.11), that the value of $L_{\text{corr}}$ grows unboundedly in vicinity of the Hopf bifurcation; therefore, in any finite spatial system (which is always the case for real biological
or chemical systems) its size $L$ will be less than the correlation length. That makes the onset of chaos impossible exactly in the parameter range where the $\lambda$-$\omega$ systems are formally valid. This inconsistency might explain why the approach based on the $\lambda$-$\omega$ systems fails to describe the type of population dynamics correctly (cf. Sherratt, 2001), predicting spatiotemporal chaos for those cases where field observations report regular patterns.
Chapter 12

Patchy invasion

In the previous few chapters, we have demonstrated that self-organized patchiness uncorrelated with the environment is an inherent property of spatiotemporal population dynamics. Scenarios of pattern formation as well as the properties of the emerging heterogeneous population distribution can be rather different, though. In a system of nonlinearly interacting populations with unequal diffusivities, the Turing instability can destabilize a locally stable coexistence state and lead to the formation of a stationary, regular, spatially periodic structure. Conversely, destabilization of a population system with locally oscillatory kinetics normally results, even in the case of equal diffusion coefficients, in spatiotemporal chaos ("biological turbulence"), i.e., a spatially irregular patchy structure with the population densities fluctuating in time chaotically.

Whether intrinsic population cycles (e.g., due to the prey–predator interactions) are really common in nature remains a controversial issue (cf. White, 2001, 2004). Throughout this book, we strongly support the opinion that population oscillations are typical in population dynamics, even if they sometimes may arise as a response of a given community to some unfavourable, destabilizing factors (Gillpin, 1972; May, 1972a, 1973). The self-organized irregular patchiness then becomes a simple consequence of local oscillations as the corresponding spatially extended system is driven to spatiotemporal chaos by virtually any heterogeneous perturbation. For a few biologically reasonable situations, we have shown that the onset of biological turbulence follows the propagation of a certain traveling front — “the wave of chaos” — which separates the domains filled with chaotic population patches from the rest of the system where dynamics is regular.

Curiously, there can be another scenario when a prominent irregular patchy structure is formed straightforwardly, i.e., is not preceded by the propagation of a front. This scenario is usually associated with the existence of the Allee effect in the population growth (Petrovskii et al., 2002, 2005; Morozov et al., 2006). In this chapter, we revisit this phenomenon of “patchy invasion,” identify biological situations when it is likely to occur, and make an insight into its basic properties. In particular, we will show that the patchy invasion may appear as a result of an overpressure on the invasive species in the course of biological control efforts. We will also show that the patchy invasion is invasion “at the edge of extinction,” so that it takes place when no other scenario can
apply and the invasive species would go extinct otherwise. Finally, we will have a look at some field data and show that this scenario of species spread and the corresponding pattern formation agree very well with what is often observed in nature.

### 12.1 Allee effect, biological control, and one-dimensional patterns of species invasion

Most of the analysis in previous chapters was done under assumptions that the population per capita growth rate \( f(U) \) reaches its maximum when the population density \( U \) tends to zero and that it decreases monotonically when the population density increases (turning to zero for \( U = K \), where \( K \) is the population carrying capacity). In mathematical terms, it means that the growth rate \( P(U) \) should be a convex function. The simplest parametrization for the growth rate \( P(U) \) is then the square polynomial, which is known as the logistic growth.

However, this is not always the case with real ecological populations. There has been a growing number of examples (Courchamp et al., 1999; Stephens and Sutherland, 1999), along with relevant theoretical arguments (Dennis, 1989; Stephens et al., 1999), where the per capita growth rate reaches its maximum value for an intermediate (nonzero) value of the population density. This is called the Allee effect (Allee, 1938) and the corresponding population dynamics is sometimes called the Allee dynamics. Special attention has been paid to the case of a “strong” Allee effect (when the population growth becomes negative if the population density goes below a certain threshold density \( B \); cf. Owen and Lewis, 2001; Wang and Kot, 2001), in particular, because the deterministic population dynamics becomes more realistic in this case, excluding a possibility of population persistence once the population density has fallen to a very low value. Moreover, the Allee effect was shown to affect virtually all aspects of species interactions in space and time (Berryman, 1981; Amarasekare, 1998; Gyllenberg et al., 1999; Taylor and Hastings, 2005; Courchamp et al., 2008).

Throughout this section, we will focus on the spatiotemporal dynamics of the prey–predator system described by the following equations:

\[
\frac{\partial U(X,Y,T)}{\partial T} = D_1 \left( \frac{\partial^2 U}{\partial X^2} + \frac{\partial^2 U}{\partial Y^2} \right) + P(U) - E(U,V) ,
\]

\[
\frac{\partial V(X,Y,T)}{\partial T} = D_2 \left( \frac{\partial^2 V}{\partial X^2} + \frac{\partial^2 V}{\partial Y^2} \right) + \kappa E(U,V) - MV ,
\]

where all terms have their usual meanings.
We assume that predation is of Holling type II and use the standard functional form; see (2.5a) or (10.4). Regarding the prey growth, we now assume that it is damped by the strong Allee effect. Mathematically, the population growth under the impact of the Allee effect should be described by a function that is concave in a vicinity of \( U = 0 \) but becomes convex for larger \( U \). For mathematical analysis and simulations, different parametrization can be used; perhaps the simplest and the most common one is the cubic polynomial:

\[
P(U) = \tilde{\alpha} U ((U - B) (K - U)),
\]

where \( \tilde{\alpha} \) is a coefficient proportional to the maximum per capita growth rate; see Lewis and Kareiva (1993).

For technical reasons, here we introduce dimensionless variables differently from the two previous chapters, that is, as

\[
u = \frac{U}{K}, \quad v = \frac{V}{\kappa K}, \quad x = X \left( \frac{a}{D_1} \right)^{1/2}, \quad y = Y \left( \frac{a}{D_1} \right)^{1/2}, \quad \text{and} \quad t = aT,
\]

where \( a = A\kappa K/H \), and \( A \) and \( H \) are the parameters quantifying predation. Then, from (12.1)–(12.2), we obtain

\[
\begin{align*}
\frac{\partial u(x, y, t)}{\partial t} &= \left( \frac{\partial^2 u}{\partial x^2} + \frac{\partial^2 u}{\partial y^2} \right) + \gamma u (u - \beta) (1 - u) - \frac{uv}{1 + \alpha u}, \\
\frac{\partial v(x, y, t)}{\partial t} &= \epsilon \left( \frac{\partial^2 v}{\partial x^2} + \frac{\partial^2 u}{\partial y^2} \right) + \frac{uv}{1 + \alpha u} - \delta v,
\end{align*}
\]

where \( \alpha = K/H, \beta = B/K, \gamma = \tilde{\alpha} H K/(\kappa a), \delta = M/a, \) and \( \epsilon = D_2/D_1 \). For the most of this chapter, we fix \( \epsilon = 1 \); the effect of differential diffusivity will be briefly addressed in Section 12.4.

In this section, we will focus on the one-dimensional dynamics; correspondingly, system (12.4)–(12.5) is reduced to

\[
\begin{align*}
\frac{\partial u(x, t)}{\partial t} &= \frac{\partial^2 u}{\partial x^2} + \gamma u (u - \beta) (1 - u) - \frac{uv}{1 + \alpha u}, \\
\frac{\partial v(x, t)}{\partial t} &= \epsilon \frac{\partial^2 v}{\partial x^2} + \frac{uv}{1 + \alpha u} - \delta v.
\end{align*}
\]

Before proceeding to the spatiotemporal dynamics of the system (12.6)–(12.7), it is worth having a brief look at its nonspatial counterpart:

\[
\begin{align*}
\frac{du}{dt} &= \gamma u (u - \beta) (1 - u) - \frac{uv}{1 + \alpha u}, \\
\frac{dv}{dt} &= \frac{uv}{1 + \alpha u} - \delta v.
\end{align*}
\]

The isoclines of the system (12.8) are given by

\[
\begin{align*}
\text{(a)} \quad v &= \gamma (1 + \alpha u) (u - \beta) (1 - u) \quad \text{and} \quad \text{(b)} \quad u &= \frac{\delta}{1 - \alpha \delta}.
\end{align*}
\]
In the biologically meaningful first quadrant \((u \geq 0, v \geq 0)\) of the phase plane, the isocline (12.9a) is dome-shaped and the stability of the coexistence steady state (given by the isocline intersection) is different depending on the position of the vertical line (12.9b). Considering \(\delta\) as a controlling parameter, which will be justified later, for the values of delta large enough (but not exceeding \(1/\alpha\) when the steady state disappears from the first quadrant) the intersection point is on the right of the hump and the steady state is stable. Let us note, however, that, contrary to the case of the prey–predator system with the logistic growth for prey, the extinction steady state \((0, 0)\) is now stable for any parameter values. Whether the system trajectories go to the extinction state or to the coexistence state depends on their position with respect to the separatrix (see Figure 12.1), while the other two steady states \((\beta, 0)\) and \((1, 0)\) are the saddle-points.

As \(\delta\) becomes smaller, the intersection point passes the hump for \(\delta = \delta_{\text{Hopf}}\) and the steady state loses its stability through the Hopf bifurcation. The attractors of the system are then the stable limit cycle and the extinction state, the two fields of trajectories being separated by the separatrix; see Figure 12.2.

The limit cycle does not persist for long, though. Along with a further decrease in \(\delta\), it promptly grows in size. When \(\delta\) reaches a certain critical
value $\delta_{het}$, it touches the saddle-points $(\beta, 0)$ and $(1, 0)$ and a heteroclinic connection is created. For $\delta < \delta_{het}$, the limit cycle disappears, the coexistence state is unstable, and the only attractor in the system is the extinction state; see Figure 12.3. The system kinetics then becomes "excitable": Depending on how far the initial point (given by the initial conditions of the system (12.8)) is from the extinction state, the system either returns to $(0, 0)$ straightforwardly (cf. curve 1) or after a long excursion over the phase plane (cf. curve 2). Which of the two trajectories actually takes place apparently depends on whether the initial point lies on the left or on the right of the separatrix connecting the coexistence steady state to the saddle $(\beta, 0)$; the two situations can be interpreted as "undercritical" and "supercritical" perturbations, respectively.

Therefore, the properties of the nonspatial system (12.8) accounting for the Allee effect are significantly different from those in case of the logistic growth; cf. Figure 11.14. Correspondingly, one can expect that the properties of the spatiotemporal dynamics can be rather different as well, and probably more complicated, because the structure of the phase plane and the succession of bifurcations have now become more complicated. For convenience, below we will refer to the phase portraits shown in Figures 12.1, 12.2, and 12.3 as, respectively, kinetics of type I (stable coexistence state), type II (unstable coexistence state, stable limit cycle), and type III (excitable kinetics).

Now, we are nearly ready to proceed to an analysis of the spatiotemporal system (12.6)–(12.7). However, one point that yet needs to be clarified is
FIGURE 12.3: Phase plane of the system (12.8) for $\delta > \delta_{\text{het}}$. The thick curve shows the separatrix while curves 1 and 2, respectively, show typical system trajectories for “undercritical” and “supercritical” perturbations of the extinction steady state; see more comments in the text. Here $\delta = 0.51$, and other parameters are the same as in Figure 12.1.

the choice of the initial conditions. In order to do that properly, one has to take into account relevant biological arguments. Invasion of exotic species usually starts with the species introduction when a number of individuals of a given species are brought locally into a new ecosystem. Considering this species as a prey, it means that the initial condition for Equation (12.6) is most naturally described by a function of compact support. In particular, in numerical simulations we used the following initial population distribution:

$$u(x, 0) = u_0 \quad \text{for} \quad -\Delta_u < x < \Delta_u, \quad \text{otherwise} \quad u(x, 0) = 0,$$  \hspace{1cm} (12.10)

where $u_0$ is the initial prey density and $\Delta_u$ gives the radius of the initially infested domain.

Regarding the spread of invasive pests, a lot of attention has been paid recently to a possibility of biological control which, contrary to more traditional control measures through application of chemical pesticides, is expected to be more effective and ecologically friendly. In particular, predation has been identified as a factor that can potentially slow down or even block the invasive species spread (Fagan and Bishop, 2000; Owen and Lewis, 2001; Fagan et al., 2002; Petrovskii and Malchow, 2005). Let us note here that, in order to be fully relevant to the issue of control, the biological factor itself must be
controllable. From that perspective, the use of predation for invasive species control seems to be adequate and convenient because the magnitude of predation can be relatively easily regulated through predator mortality (e.g., by means of additional harvesting or hunting); higher mortality would correspond to lower predation. For that reason, in our insight into the patterns of species spread below, we will use the dimensionless mortality $\delta$ as the main controlling parameter.

In practice, the idea to use predation as a tool in order to affect the species spread implies that, soon enough after introduction of the given exotic species, a small population of a relevant predatory species is introduced locally into the region already inhabited by its prey. Thus, the corresponding initial condition for Equation (12.5) is as follows:

$$v(x, 0) = v_0 \quad \text{for} \quad -\Delta_v < x < \Delta_v, \quad \text{otherwise} \quad v(x, 0) = 0, \quad (12.11)$$

where $v_0$ is the initial predator density and $\Delta_v$ is the radius of the area where the predator is introduced.

It should also be mentioned that the initial conditions (12.10)–(12.11) are somewhat idealized and in reality the form of the species initial distribution can of course be much more complicated. However, results of computer simulations show that the type of the system dynamics depends much more on the radius of the initially inhabited domain and on the population density inside rather than on the details of the population density profile.

12.1.1 Patterns of species spread

The system (12.6)–(12.7) is a system of nonlinear PDEs and thus is difficult to study analytically; however, it is relatively easy to reveal its main properties by means of numerical simulations.

Before proceeding to the description of possible invasion scenarios, the issue of a system’s dynamics dependence on the initial conditions should be clarified somewhat further. In a space- and time-continuous prey–predator system (described by diffusion–reaction equations (12.1)–(12.2)) with logistic growth for prey, any infinitesimal initial distribution of prey results in its successful invasion. In case the prey growth is affected by the strong Allee effect, however, the system exhibits certain criticality due to the existence of the threshold population density: A successful invasion of prey cannot happen unless the initial density is large enough; otherwise, the species goes extinct (Lewis and Kareiva, 1993; Petrovskii and Li, 2006). Therefore, in order to exclude this somewhat trivial case, in computer experiments $u_0$ and/or $\Delta_u$ should always be chosen sufficiently large.

\footnote{For instance, the spread of airborne species is obviously affected by the direction and strength of the wind, but, since these two features are totally out of our control, we cannot use it for invasive species management.}
All regimes of the system dynamics can be classified into three groups; see Figure 12.4. These groups correspond to extinction, invasion on a geographical scale when the species keep spreading until they reach the domain boundaries, and regional persistence when the species invade locally and spread over a certain area but do not go farther.

**FIGURE 12.4:** A diagram of possible invasion scenarios in a prey–predator system with the strong Allee effect.

We begin with the regimes describing the unbounded spread of the invasive species (geographical invasion). There are three qualitatively different scenarios of the species spread.

According to the first scenario, the species is spreading over space through propagation of a traveling population front. In front of the front the species is absent, and behind the front it is present at a considerable density. Depending on parameter values, in the wake of the front there can arise either a station-
FIGURE 12.5: Solitary traveling patches of population density (solid curves for prey, dashed for predator) at $t = 350$, 600 and 850 (top to bottom, respectively) as predicted by the model (12.6)–(12.7). Parameters are $\alpha = 0.5$, $\beta = 0.28$, $\gamma = 3$, and $\delta = 0.425$. The initial conditions are given by (12.10)–(12.11) with $u_0 = 1$, $v_0 = 0.2$, $\Delta_u = 40$, and $\Delta_v = 20$. Since the problem is symmetrical with respect to the origin, only a half of the domain is shown.

ary spatially homogeneous species distribution (local kinetics of type I) or spatiotemporal population oscillations (local kinetics of either type II or III), which can be either regular or, more typically, chaotic. For some parameter values, the propagating front is linked to the domain with chaotic oscillations by means of a quasi-homogeneous plateau where the values of population
FIGURE 12.6: Species invade over space through irregular dynamics of separate patches. The snapshots of the population density (solid curves for prey, dashed for predator) is shown at $t = 600, 1000, \text{ and } 1400$ (top to bottom, respectively). Parameters are $\alpha = 0.05, \beta = 0.28, \gamma = 3, \text{ and } \delta = 0.53$, and the initial conditions are the same as in the previous figure.

density correspond to the locally unstable equilibrium; cf. the phenomenon of “dynamical stabilization” considered in Chapter 10. Apparently, the pattern of spread through the propagation of a population front is of high ecological relevance (cf. Shigesada and Kawasaki, 1997). However, as a whole, this scenario is very similar to what has already been observed and studied for the system without Allee effect; therefore, we do not go into more detail here.

According to the second scenario, the species spread over space via propa-
Patchy invasion

Invasion of a solitary moving patch, or traveling “pulse”; cf. Figure 12.5. In this case, local kinetics is of type III; indeed, traveling pulses are often regarded as a “fingerprint” of excitable kinetics in a spatiotemporal system’s dynamics (e.g., see Lindner et al., 2004, and references therein). Depending on parameter values, the traveling population pulse can be either stationary (when its shape does not change with time) or nonstationary (when its shape oscillates with time); in both cases, the pulse propagates with a constant speed.

Note that, in this case, the invasive species is absent both in front of the pulse and in its wake; the latter apparently means that invasion has failed. This fact has a curious ecological interpretation, i.e., there is species spread on a geographical scale but there is no invasion. In Sections 12.2 and 12.4, we will discuss this observation further and show that it may have important implications for biological control strategies and invasive species management.

From the point of ecological pattern formation, however, it is the third scenario that is probably the most interesting. In this case, invasion takes place through the formation and motion of separate patches and/or groups of patches; see Figure 12.6. However, the patch motion is now much more complicated than the simple locomotion in the case of traveling pulses. There is no traveling wave. The patches interact with each other, they merge and split, some of the patches or even groups of patches can disappear, new patches are formed, they can produce new groups of patches, etc. The invaded area grows and eventually the patches occupy the whole domain. Remarkably, the size of the domain occupied by the moving patches does always not grow monotonically: The boundary of the invaded domain can be set back when the leading group of patches goes extinct in the course of the system dynamics (Petrovskii et al., 2005).

Regimes of regional invasion

When predation becomes high enough (specific value of $\delta$ depends on other parameters), unbounded species spread can no longer take place. However, invasion may still take place “regionally”: For certain parameter values, evolution of the initial species distribution leads to the formation of quasistationary patches; see Figure 12.7. In this case, at early stage of the system dynamics (for $t \approx 100$), two symmetric dome-shaped patches are formed. Their position then remains fixed, although their shape can be either stationary or oscillating. Interestingly, in the latter case, in spite of the fact that the spatial structure is pretty simple and regular, temporal fluctuations of the population density can exhibit rather complicated dynamics such as $n$-periodic limit cycles and even chaos (Morozov et al., 2004).

Regimes of anomalous extinction

When the magnitude of predation is very large, simulations show that species extinction becomes inevitable, which apparently signifies the final success of the biological control strategy. Dynamically, it may happen in a rather
different manner, though. In the above, we have already mentioned that an introduced species affected by the strong Allee effect will go extinct if its initial population size is not large enough. In this case, the population size decreases exponentially while the population stays localized inside about the same domain where it had originally been introduced. This scenario of species extinction seems to agree perfectly with what is intuitively expected.

Due to the interplay between the Allee effect and predation, however, species extinction can also follow more exotic dynamical scenarios. There
can be long-term transients that mimic some of the regimes of the species’ geographical spread. In order to distinguish them from the trivial extinction scenario mentioned above, we refer to them as to “anomalous” extinctions.

Specifically, depending on parameter values, there can be two regimes when species extinction is either preceded by formation of a distinct long-living spatiotemporal pattern or by a long-distance population spread. In the first case, the initial conditions evolve into an ensemble of patches allocated over the domain. The patches interact with each other in a complicated manner similar to the patch dynamics shown in Figure 12.6. However, the regime is not self-sustainable and, finally, the species go extinct. In the second case, a moving patch is formed which propagates with approximately constant speed over distances much larger compared to the radius of the initial species distribution before the prey is caught by the predator; the pulse then decelerates, stops, and both species go extinct promptly.

We want to emphasize that, in both of these cases, the invasive population persists during a remarkably long time before the actual population decay takes place (typically, on the order of a hundred times longer than it would be in the case of the ordinary extinction) and/or it spreads over large distances. During that period, the system dynamics can be very similar to the corresponding regimes of geographical spread; see Figures 12.6 and 12.5, respectively.

Therefore, we have shown that the impact of the Allee effect (which, in a certain parameter range, makes the system kinetics excitable) brings to life two essentially new regimes of pattern formation that have not been observed in other systems. These regimes are (i) standing or traveling solitary patches of population density and (ii) the “patchy invasion,” which is not associated with any traveling wave propagation. As a whole, the spatiotemporal dynamics of the prey–predator system appears to be much richer and much more complex under the impact of the Allee effect; see Figure 12.4. For comparison, in the corresponding system without the Allee effect (cf. Equations (10.6)–(10.7)), the only scenario is pattern formation in the wake of the traveling population front.

We note that the variety of dynamical regimes shown in Figure 12.4 can be presented in a more quantitative way as maps in the parameter space of the system, with different domains corresponding to different regimes. Since biological invasion per se is largely beyond the scope of this book, we are not showing them here; an interested reader can find more information in Petrovskii et al. (2005). What is important to mention is that, although a particular succession of regimes may be somewhat different depending on other parameter values, there are features of those maps that seem to be universal. Namely, the system dynamics always leads to species extinction for small $\delta$ (high predation) and to successful invasion through the propagation of a traveling front for large $\delta$ (low predation), while less trivial scenarios such as solitary patches and patchy invasion occur for intermediate values.
By now, we have focused on species’ biological invasion and biological control in a system with one spatial dimension. This is a natural step in understanding the system spatiotemporal dynamics. Also, an advantage of an one-dimensional system is that numerical simulations are much faster than they would be in the corresponding multidimensional case; that makes it possible to study the problem thoroughly (cf. Petrovskii et al., 2005). However, in reality, biological invasion and the corresponding ecological pattern formation normally take place in two dimensions. The question thus arises to what extent the scenarios discussed in this section can be extended onto the dynamics of two-dimensional systems. This issue will be addressed below.

### 12.2 Invasion and control in the two-dimensional case

Now, we are going to make an insight into the patterns of biological invasion and corresponding species spread in the two-dimensional case, which is ecologically more realistic. As in the previous section, our main focus will be on pattern formation arising due to the interplay between the Allee effect and predation, the latter being regarded as a factor of biological control. The mathematical model is now given by the “full” system (12.4)–(12.5).

Since both the invasive species and its predator are introduced locally, relevant initial conditions for system (12.4)–(12.5) should be described by functions of compact support. Specifically, in this section we consider them in the form of rectangular patches:

\[
\begin{align*}
    u(x, y, 0) &= u_0 \quad \text{for} \quad |x - x_u| \leq \frac{\Delta_{ux}}{2} \quad \text{and} \quad |y - y_u| \leq \frac{\Delta_{uy}}{2}, \\
    v(x, y, 0) &= v_0 \quad \text{for} \quad |x - x_v| \leq \frac{\Delta_{vx}}{2} \quad \text{and} \quad |y - y_v| \leq \frac{\Delta_{vy}}{2},
\end{align*}
\]

where \((x_u, y_u)\) and \((x_v, y_v)\) give the patch centers; \(\Delta_{ux}, \Delta_{vx}, \Delta_{uy}, \Delta_{vy}\) define the patches size in the direction of \(x\) and \(y\), respectively; \(u_0\) and \(v_0\) are the initial population densities inside the patches.

Note that even the dimensionless system (12.4)–(12.5) contains a few parameters, i.e., \(\alpha, \beta, \gamma, \delta, \) and \(\epsilon\), while the system in original dimensional variables contains twice as many. Therefore, a regular simulation study of its properties over a reasonably wide parameter range would imply at least several thousands simulation runs. For a two-dimensional system, where each single simulation takes hours (if done with proper accuracy, which implies large numerical grids and small time-steps), this is hardly possible. Instead,
we use another strategy. We choose one controlling parameter and consider the changes in the invasion scenario subject to its variation, keeping all other parameters fixed at certain hypothetical values. In agreement with the idea of biological control (see the paragraph below Equation (12.10)), it seems that to use the (dimensionless) predator mortality $\delta$ as the controlling parameter is a convenient and biologically reasonable choice.

We begin with the case when $\delta$ is large – correspondingly, the predator is weak and predation is low. Intuitively, a weak predator would unlikely affect prey spread in any significant way. In the one-dimensional case, in the absence of a predator, the prey would normally spread as a stationary traveling wave (Fisher, 1937; Kolmogorov et al., 1937; Aronson and Weinberger, 1975; Fife, 1979); therefore, in a prey–predator system with a weak predator, the invasion of prey would likely take place through the traveling wave scenario as well. Although the plane waves do not directly apply to the two-dimensional case with the initial conditions of compact support, one can still expect the formation of a moving population front separating invaded and uninvaded areas.

These heuristic arguments agree very well with simulations. Having other parameters fixed, for the values of $\delta$ on the order of unity or larger, the initial conditions (12.12)–(12.13) lead to prey invasion through a radial expansion of the infested domain, with its shape being approximately circular except for a very early stage of the system’s dynamics when the specifics of the initial distribution can be essential. The domain boundary propagates as a traveling front (although its speed is not constant now but grows monotonously to a constant value in the large-time limit), behind the front the species are distributed homogeneously. The corresponding local kinetics is of type I; see Figure 12.1.

For somewhat smaller $\delta$, system’s kinetics changes to type II (stable limit cycle) and the homogeneous population distribution in the wake changes to chaotic spatiotemporal oscillations. Typical population distributions are shown in Figure 12.8. Recall that a similar pattern has been observed in a prey–predator system without Allee effect when the local dynamics is oscillatory; see Chapter 10.

A decrease in $\delta$ destroys the limit cycle and makes the system kinetics excitable; cf. Figure 12.3. In case $\delta$ is only slightly less than $\delta_{\text{het}}$, the expanding front with patterns in the wake remains to be the invasion pattern. A further decrease, however, changes it to expanding rings (see Figure 12.9), which seems to be a natural two-dimensional extension of traveling population pulses. The rings are centered around the place of original species introduction. Note that in this regime of species spread the species are absent both in front of the propagating front (i.e., outside of the ring) and behind the front (inside the ring). From the ecological standpoint, this means that the invasive species fails to establish itself in the new environment in spite of the fact that the species spread does take place on a geographical scale.

Since the growing rings scenario already means invasion failure, one might
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expect that smaller values of $\delta$ (stronger predator, higher predation) would likely lead to a fast eradication of invasive species, probably without any spread at all. Surprisingly, this is not the case. A further decrease in $\delta$ (local kinetics remains to be of type III) changes the rings to a completely different type of dynamics; see Figure 12.10. In this case, at a very early stage the species spread looks similar to the one with patterns in the wake of the expanding front; cf. Figures 12.10 (top, left) and 12.8. At a certain moment, however, the continuous front breaks to pieces and never reappears again.

**FIGURE 12.8:** Invasion fronts with chaotic spatiotemporal oscillations in the wake: Snapshots (contour lines) of the prey density shown for $t = 25$, $t = 100$, $t = 175$, and $t = 250$, (a) to (d), respectively. Thick curves correspond to large gradients in the population density. The distribution of predator density exhibits qualitatively similar properties. Parameters are $\alpha = 0.1$, $\beta = 0.22$, $\gamma = 3$, and $\delta = 0.52$. The initial conditions are given by (12.12)–(12.13) with $x_u = 145$, $y_u = 152.5$, $x_v = 140$, $y_v = 155$, $\Delta_{ux} = 20$, $\Delta_{ux} = 10$, $\Delta_{uy} = 5$, $\Delta_{uy} = 20$, $u_0 = 1$, and $v_0 = 0.2$. 
At any later time, the population spreads over space via irregular motion of separate patches. The patches move, grow, merge, split, produce new patches, etc. We emphasize that this “patchy invasion” is a self-sustained regime and it has nothing to do with the long-living transients mentioned in Section 12.1.1. Long-term simulations show that, after the population patches invade over the whole domain, the spatiotemporal dynamics of the system does not change and the spatial distribution of species at any moment is qualitatively similar (up to the position and shape of particular patches, which are changing all the time) to the one shown in Figure 12.10 (bottom, right).

FIGURE 12.9: Spread of invasive species through expanding rings: Snapshots (contour lines) of the prey density shown for $t = 25$, $t = 150$, $t = 375$, and $t = 400$, (a) to (d), respectively. Thick curves correspond to large gradients in the population density. Note that the species is absent both outside and inside the rings. The distribution of predator density exhibits qualitatively similar properties. Parameters are $\alpha = 0.1$, $\beta = 0.22$, $\gamma = 3$, and $\delta = 0.44$. The initial conditions are the same as in Figure 12.8.
For still smaller values of $\delta$, predation becomes too strong and both species go extinct. Note that, since the model (12.1)–(12.2) does not allow for an alternative food source for the predator, it cannot survive after the prey disappears. Between patchy invasion and extinction, there can be a very narrow parameter range where evolution of the initial conditions may result in stationary patches; see Morozov and Li (2007) for more details and also see Section 12.3.1 for an example of similar dynamics.

FIGURE 12.10: Patchy invasion: Snapshots (contour lines) of the prey density shown for $t = 50$, $t = 250$, $t = 450$, and $t = 750$, (a) to (d), respectively. Thick curves correspond to large gradients in the population density. Between the patches the species is absent, inside the patches the species is present at high density. Note that, except for some very early stage, there is no continuous boundary separating invaded and uninvaded areas. The distribution of predator density exhibits qualitatively similar properties. Parameters are $\alpha = 0.1$, $\beta = 0.22$, $\gamma = 3$, and $\delta = 0.42$. The initial conditions are the same as in Figure 12.8.
Thus, in a two-dimensional prey–predator system where prey growth is damped by the strong Allee effect, depending on the value of the predator mortality, the spatial spread of invasive species can follow a variety of scenarios. A decrease in mortality first changes the standard traveling wave scenario (with either homogeneous or patchy species distribution in the wake) to growing/expanding population rings (with no species in the wake) and then to patchy invasion. It should be mentioned that the last two scenarios are specific for the prey–predator system with the Allee effect, which makes system kinetics excitable, and cannot take place otherwise.

Let us recall that, in our approach, the predator mortality $\delta$ is a measure of the biological control effort. Therefore, the above succession of invasion scenarios arises in response to an increase in the control effort. One conclusion that can be made here is that these results make a strong argument in support of the biological control strategy: Indeed, a sufficiently strong predator will eradicate the invasive prey, although the actual scenario is much more complicated than the previously suggested blocking and reverse of the propagating population fronts (Owen and Lewis, 2001; Petrovskii and Malchow, 2005).

Now, one curious feature of this succession is that the success of biological control depends on the control effort in a nonmonotonic way. Increasing effort brings successful invasion (traveling front with patterns in the wake) to failure (expanding rings). In the latter case, the invasive species is spreading, but it will not do much harm because it is going to disappear anyway. However, a further increase in controlling effort restores successful invasion, although the scenario (“patchy invasion”) will be quite different.

A relevant question is how the rate of species spread changes between the different regimes. It should be mentioned here that, while in traveling wave scenarios the rate of spread apparently coincides with the wave speed and thus can be calculated straightforwardly, in case of the patchy invasion its definition is somewhat less obvious. One way to obtain it is to introduce the domain radius $R$ as the maximum distance between the center of the invaded domain and the positions in space where the prey density exceeds a certain threshold level $u_{\text{cut}}$, i.e.,

$$R(t) = \max_{u(x,y) > u_{\text{cut}}} \left[ (x - cC)^2 + (y - yC)^2 \right]^{1/2} \quad (12.14)$$

(where $u_{\text{cut}}$ should be chosen reasonably small), and then to calculate $dR/dt$. The position $(xC, yC)$ of the domain center can be defined differently; however, the results do not depend much on the exact definition (cf. Morozov et al., 2006).

The results are shown in Figure 12.11. It is readily seen that, while for the regimes associated with traveling fronts the rate of spread varies insignificantly (showing a gradual increase with an increase in $\delta$), in the regime of patchy invasion it drops to zero within a rather narrow parameter range.
FIGURE 12.11: Rate of species spread for different values of the predator mortality $\delta$ and for different invasion scenarios (dots and stars correspond to different definitions of $x_C$ and $y_C$); (A) for invasion front with patterns in the wake, (B) for expanding rings, (C) for patchy invasion. Other parameters are $\alpha = 0.1$, $\beta = 0.22$, and $\gamma = 3$. With permission from Morozov et al. (2006).

12.2.1 Properties of the patchy invasion

From the invasion scenarios described above, probably the most curious and the most congenial to the scope of this book is the patchy invasion. Therefore, now we are going to make a somewhat deeper insight into its properties.

Perhaps the first question coming into mind when looking at the irregular patterns shown in Figure 12.10 is whether this dynamics is actually chaotic. The answer is yes; numerical simulations show that the temporal fluctuations of the population densities are remarkably irregular and the solutions of the system (12.4)–(12.5) exhibit the special type of sensitivity to the initial conditions typical for chaos when the difference between the perturbed and unperturbed solutions grows promptly (exponentially) with time. A more quantitative approach to this issue gives the value of the dominant Lyapunov exponent on the order of $10^{-2}$; in particular, for the parameters of Figure 12.10, $\lambda_D \approx 0.035$ (Morozov et al., 2006). Also, the spatial properties of the pattern, with the autocorrelation function showing fast decay at short distances, appear to be typical for spatiotemporal chaos; cf. Section 11.2 for an example of a similar behavior.

It should be mentioned here that chaos in a spatially extended prey–predator system is a typical rather than exotic phenomenon. In particular, we have mentioned earlier that the irregular patterns arising in the wake of the prop-
agating front are chaotic as well. (Indeed, the patterns in the wake look qualitatively similar to the patterns arising as a result of patchy invasion; cf. the bottom of Figure 12.8 and the middle row of Figure 12.10.) Thus, chaos alone is not enough to distinguish between the two regimes. A question that arises is whether it is in both cases “the same” chaos (i.e., with essentially the same spatiotemporal properties) in spite of the fact that its onset follows rather different scenarios, or there is anything special or unusual about the pattern emerging as a result of patchy invasion.

In order to address this issue, we have to make a deeper insight into the properties of the corresponding dynamics. High complexity of the system behavior means that it is close to stochastic dynamics if considered on a timescale larger than the correlation time $\tau_{corr}$ of the system. This observation makes it possible to apply a statistical approach and to quantify the system dynamics in terms of the probability distribution of its different states.

To make use of the patch statistics, first, we need to estimate the correlation time of the system. That can be done in a standard way by means of calculating the autocorrelation function(s) and finding its/their first zero; for the parameters of Figure 12.10, $\tau_{corr} \approx 35$.

Second, we should construct a relevant “ensemble” of the system’s states. For that purpose, from a numerical solution of the system (12.4)–(12.5) obtained at a certain fixed position ($\bar{x}, \bar{y}$), we extract a time series of the population densities where any two consecutive terms are separated with a time-lag $\tau_{corr}$. Owing to the meaning of $\tau_{corr}$, any two measurements in these series can be regarded as independent, so that we can now restore the probability distribution functions (PDF) of the system states.

Since the time series obtained for prey and predator exhibit qualitatively similar properties, below we show only the results obtained for prey. Figure 12.12 shows the PDF of the local prey density obtained for the parameters of Figure 12.8, i.e., for the chaotic pattern generated by a propagating front. The properties of the obtained histogram are heuristically clear and agree very well with intuitive expectations. A “sampling” of the system would normally bring a value of the prey density somewhere inside the limit cycle (cf. the shallow maximum in the middle); recall that the system kinetics is of type II in this case. Existence of another local maximum in vicinity of $u = 0$ reflects the fact that $(0,0)$ is a stable steady state of the system.

Obviously, a similar analysis can also be applied to a time series of spatially averaged population density. Figure 12.13 shows the PDF of the spatially averaged prey density calculated for the parameters of Figure 12.8. Note that, in this case, the shape of the PDF is very close to that of the normal distribution (shown by the solid curve). Since in the regime of spatiotemporal chaos the whole domain appears to be dynamically split to an ensemble of mutually independent oscillators with the spatial size $\sim L_{corr}$ (see Section 11.2), a normal distribution arises naturally as a result of the Central Limit Theorem.

However, the PDF properties appear to be rather different in case of the
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**FIGURE 12.12:** Probability distribution function of the local prey density in case of spatiotemporal chaos generated by the propagating front; parameters are the same as in Figure 12.8 (with permission from Morozov et al., 2006).

**FIGURE 12.13:** Probability distribution function of the spatially averaged prey density in case of spatiotemporal chaos generated by the propagating front; parameters are as in Figure 12.8 (with permission from Morozov et al., 2006).
**FIGURE 12.14**: Probability distribution function of the local prey density in case of spatiotemporal chaos generated by the patchy invasion; parameters are the same as in Figure 12.10 (with permission from Morozov et al., 2006).

**FIGURE 12.15**: Probability distribution function of the spatially averaged prey density in case of spatiotemporal chaos generated by the patchy invasion; parameters are the same as in Figure 12.10 (with permission from Morozov et al., 2006).
patterns emerging as a result of the patchy invasion. Figure 12.14 shows the PDF of the local prey density calculated for the parameters of Figure 12.10. In this case, the PDF has the only distinct maximum at \( u = 0 \), which means that an expected value of the prey density is well below the survival threshold \( \beta = 0.22 \).

A similar result is obtained for the PDF of the spatially averaged density. Figure 12.15 shows the PDF of the spatially averaged prey density obtained for the parameters of Figure 12.10. Again, the PDF shape is very close to normal distribution. However, contrary to the case shown in Figure 12.13, now the PDF maximum is well below the survival threshold.

Therefore, in the case of pattern formation preceded by front propagation, high values of the prey density are more probable than low values. On the contrary, in the case of the patchy spread, low values of prey density are more probable than high ones. Note that, in both cases, the value of the survival threshold is the same, \( \beta = 0.22 \). A brief inspection of Figure 12.14 immediately leads to a rather surprising result, that, in the regime of patchy spread, the probability of detecting the prey density below the survival threshold is higher than the probability of finding it above the threshold. Moreover, as it is readily seen from comparison between Figures 12.13 and 12.15, the spatially averaged prey density appears to be well above and well below the survival threshold for the regimes of invasion with and without continuous population front, respectively.

**Impact of space dimension**

We have already shown that patchy invasion can be observed in a parameter range separating the parameter domain where the species will spread without invasion and the domain where the species will go extinct straightforwardly, i.e., without spreading at all. Therefore, patchy invasion gives a scenario of species invasion “at the edge of extinction” so that a reasonably small variation of the controlling parameter \( \delta \) will turn successful invasion into failure.

Moreover, statistical analysis of the patch dynamics shows that, in the patchy regime, the species spread takes place at an amazingly low value of the average population density, i.e., well below the Allee threshold \( \beta \). Obviously, this is essentially a spatiotemporal effect associated with the self-organized patchiness; a similar dynamics – i.e., species persistence below the survival threshold – could never be observed in a nonspatial system. That gives another example of crucial importance of spatial dimension(s) for population dynamics.

Note that a qualitatively similar succession of regimes has been observed in the corresponding one-dimensional case (see section 12.1.1), where the patchy invasion also takes place in a parameter range preceding species extinction. An interesting question is how the parameter ranges in one and two dimensions correspond to each other.

Simulations show that, for the parameter values when patchy invasion occurs in the one-dimensional system, the species spread in two spatial dimen-
A decrease in $\delta$ turns the one-dimensional patchy invasion to extinction. Surprisingly, however, for the same parameter values the system dynamics in two spatial dimensions leads not to species extinction but to patchy invasion; a detailed consideration of this issue can be found in Petrovskii et al. (2005) and Morozov and Li (2007).

A heuristic description of invasion failure in the one-dimensional case is as follows. At an early stage of species spread, the traveling front of the prey density propagates into empty space, followed by a traveling front of predator density; the dynamics is similar to the so-called prey–predator pursuit scenario (Murray, 1989). The speed of prey wave appears to be lower than that of the predator wave; as a result, prey is caught up by the predator. After some oscillations, predator decreases the prey density below the survival threshold $\beta$ at every location in space, and extinction of both species takes place.

In the two-dimensional system, however, the patch border is curvilinear; thus, prey can escape through the lateral sides and create separate patches. Each new patch then starts growing through the formation and expansion of a circular population front (cf. the top left panel of Figure 12.10) until prey is caught again by the predator, and this scenario occurs again and again resulting in a prominent patchy structure.

Note that the effect of patch border curvature is positive for prey and negative for predator. Indeed, the larger curvature (i.e., the smaller the patch radius), the higher the rate of population density decrease due to its outflux through the patch border. A decrease in the population densities inside a given patch weakens the impact of predation on the prey growth and also decreases the growth rate of predator; see Equations (12.4)–(12.5), where the term describing interspecies interaction has a different sign in the equation for prey and in the equation for predator. Thus, prey has more chances to survive in small patches where the border curvature is large than in large patches where the border shape is close to a straight line. That helps us to understand how prey can possibly survive in the two-dimensional case for the parameter values when it is brought down by predation in the one-dimensional case.

In the above, we have considered how the scenario of biological invasion in a prey–predator system with a strong Allee effect changes in response to a decrease in predator mortality. We want to mention, however, that similar changes are observed if we choose another controlling parameter, e.g., the threshold density $\beta$, and keep the other parameters fixed. In this case, an increase in $\beta$ (instead of a decrease in $\delta$) eventually leads from species invasion through propagation of continuous fronts to patchy invasion and then, for a larger $\beta$, to extinction.

It should be mentioned that, apart from restrictions on parameters, patchy
invasion is also somewhat sensitive to the choice of initial conditions. In particular, simulations show that the patchy invasion scenario will never take place in case of predators spreading into the area already inhabited by prey; even for the relevant parameter values, it will be the regime of traveling patches or growing rings instead. Thus, the finiteness of the species’ initial distribution is an essential condition.

A question important for understanding prospective ecological implications of patchy invasion is, what could be the typical size of the patches, if any, in terms of real ecosystems? Indeed, a closer look at the spatial patterns (e.g., by means of calculating the correlation length; for details see Morozov et al., 2006) emerging as a result of patchy invasion shows that there is an intrinsic spatial scale, with its value being typically between 10 and 20 in dimensionless units. According to the definition of dimensionless variables (see the lines above Equations (12.4)–(12.5)), the relation between the dimensional $R$ and dimensionless $r$ spatial scales is given as $R = r [DH/(A\kappa K)]^{1/2}$. Note that, in our simulations, parameter $\alpha$ is always fixed at a hypothetical value of 0.1, which means that the effect of saturation on predator response is insignificant. As for $D$ and $A\kappa$ (recalling here that $A\kappa$ has the meaning of predator maximum growth rate), they can be different for different species. As an example, we consider the vole-weasels interaction (cf. Sherratt et al., 2002) with $D = 0.2$ km$^2$ year$^{-1}$ and $A\kappa = 2.7$ year$^{-1}$ as typical values. We then obtain that $R$ lies between 8.5 km and 17 km, which seems to be ecologically reasonable. Note that an increase/decrease in $D$ or $A\kappa$ as much as ten times corresponds to only about three times’ increase/decrease in $R$; thus, an estimate for $R$ to be between a few kilometers and a few dozens kilometers is likely to remain valid for many other terrestrial species.

12.3 Biological control through infectious diseases

In the previous two sections we showed that the use of predation as a factor of biological control aiming to slow down or block the spatial spread of an invasive pest may result, provided the pest’s local population growth is affected by the strong Allee effect, in a new and curious scenario of spatiotemporal pattern formation. Namely, in a certain parameter range the spread can take place not via the intuitively expected traveling continuous population front but via the motion and interaction of separate patches. We then showed that the phenomenon of patchy invasion takes place “at the edge of extinction,” so that a reasonably small change of controlling parameters either brings the species to extinction or restores the standard traveling front scenario.

Another recognized factor of biological control on invasive species is the impact of infectious diseases. In that case, in order to affect the spread of a
harmful species (and, ideally, to lead to its eradication) at an early stage of invasion an infectious disease is introduced into the spreading population, i.e., a number of individuals are deliberately infected by a certain dangerous or even lethal virus (Fitzgerald and Veitch, 1985; Courchamp et al., 1995). The impact of infection on invasive species spread has been studied theoretically and a possible slowdown of invasion rates was demonstrated for one-dimensional systems; e.g., see Hilker et al. (2005, 2007) and references therein. A “strong” infection (i.e., one with high transmission rate and/or high virulence) was predicted to block species invasion by means of blocking and reversing the corresponding traveling population fronts. The two-dimensional case, however, has yet remained less studied and poorly understood. A thorough consideration of this problem would carry us away far beyond the scope of this book; instead, our goal here is much more modest. Namely, we are going to check whether infection-based biological control can change the scenario of invasive species spread in a manner similar to that observed above for the prey–predator systems. We are especially interested to know whether the impact of infection may change a continuous front scenario to patchy invasion.

We begin with one of the simplest models of mathematical epidemiology, i.e., the so-called SI model:

\[
\frac{\partial s(r,t)}{\partial t} = \left( \frac{\partial^2 s}{\partial x^2} + \frac{\partial^2 s}{\partial y^2} \right) + \gamma s(s - \beta)(1 - s) - si, \tag{12.15}
\]

\[
\frac{\partial i(r,t)}{\partial t} = \epsilon \left( \frac{\partial^2 i}{\partial x^2} + \frac{\partial^2 i}{\partial y^2} \right) + si - \delta i \tag{12.16}
\]

(e.g., see Murray, 1989), where \(s\) and \(i\) are the densities of the susceptible and infected individuals, respectively, at moment \(t\) and position \(r = (x, y)\). For the sake of brevity, in Equations (12.15)–(12.16), all variables are already scaled to dimensionless values following a standard routine. Throughout this section, we assume that the diffusivity ratio \(\epsilon = 1\). The term \(si\) describes the disease transmission rate from infected to susceptible individuals. We assume that the disease is serious enough that infected individuals cannot produce offspring and the population can grow only due to multiplication of susceptibles.

Obviously, the initial spatial distribution of \(s\) and \(i\) should be described by functions of compact support; therefore, for modeling purposes, the initial conditions (12.12)–(12.13) are still appropriate, up to the corresponding change of \(u\) to \(s\) and \(v\) to \(i\).

The system (12.15)–(12.16) has been studied by means of numerical simulations. We obtain that the succession of the regimes in response to a change in the controlling parameter, e.g., in either \(\delta\) or \(\beta\) (cf. the remark at the end of Section 12.2), is similar to the one that has been observed for the prey–predator system. A decrease in \(\delta\) eventually changes the invasion scenario via propagation of traveling fronts with pattern formation in the wake (see Figure 12.16) to patchy invasion (see Figure 12.17). For smaller \(\delta\), the species
FIGURE 12.16: (See color insert.) Snapshots (contour lines) of the two-dimensional spatial distribution of the density of susceptibles, as given by the SI model (12.15)–(12.16), calculated at the moments $t = 0$, $t = 40$, $t = 80$, and $t = 160$ (left to right, top to bottom). The density of infected exhibits similar properties. Parameters are $\gamma = 2$, $\beta = 0.2$, and $\delta = 0.44$.

... goes extinct. Thus, the patchy spread of invasive species can arise in response to controlling efforts based on the introduction of certain infectious diseases in the same way as it arises in response to predation. The patchy invasion in this case has exactly the same implication as in the prey–predator system: It describes the scenario of spatial spread at the edge of extinction so that a further small (but finite) change of the controlling parameter leads to species extinction.

Note that, from a mathematical aspect, the system (12.15)–(12.16) is not just a particular case of Equations (12.4)–(12.5) corresponding to $\alpha = 0$. The decrease from $\alpha > 0$ to $\alpha = 0$ means a certain structural change: Instead of strong nonlinearity $uv/(1+\alpha u)$, we now have a bilinear term that corresponds to the classical Lotka–Volterra model. It is well known (cf. Murray, 1989) that the dynamics of the model with a bilinear interaction term and the one with Holling type II can differ in many aspects, in particular, with regards to steady states/limit cycle(s) existence and stability. Thus, extension of the main results onto the SI model is nontrivial, although it might be intuitively expected.
It should also be mentioned that, although the succession of dynamical regimes described above when a decrease in $\delta$ eventually leads to species extinction may seem to be somewhat counter-intuitive, it is in full agreement with biological arguments. Indeed, the dimensionless parameter $\delta$ gives, up to a certain factor, a ratio of the infected mortality rate and the transmission rate. A decrease in $\delta$ thus corresponds to an increase in the transmission rate, which tends to make disease more dangerous.

### 12.3.1 Patchy spread in SIR model

The results of the previous section inspire us to look now at the dynamics of a more complicated epidemiological model. Namely, now we are going to consider the spatiotemporal dynamics of an infectious disease described by
the following equations:

\[
\frac{\partial s(r, t)}{\partial t} = \left( \frac{\partial^2 s}{\partial x^2} + \frac{\partial^2 s}{\partial y^2} \right) + \gamma s(s - \beta)(1 - s) - si + \alpha i + \eta \rho ,
\]

\( (12.17) \)

\[
\frac{\partial i(r, t)}{\partial t} = \left( \frac{\partial^2 i}{\partial x^2} + \frac{\partial^2 i}{\partial y^2} \right) + si - \delta i - \alpha i - \sigma i ,
\]

\( (12.18) \)

\[
\frac{\partial \rho(r, t)}{\partial t} = \epsilon \left( \frac{\partial^2 \rho}{\partial x^2} + \frac{\partial^2 \rho}{\partial y^2} \right) + \sigma i - \eta \rho - \omega \rho
\]

\( (12.19) \)

(the so-called SIR model), where \( s \) is the density of the susceptible individuals of a given population, \( i \) is the density of infected, and \( \rho \) is the density of removed at the position \( r = (x, y) \) and time \( t \). As above, we assume that Equations (12.17)–(12.19) are already scaled to dimensionless values. As in the case of the SI model, we assume that only the susceptibles can produce offsprings.

Apparently, the SIR model is more complicated than the SI model and contains more mechanisms and scenarios of disease development; in particular, it includes a possibility for the infected and removed individuals to become susceptible again (with the rates \( \alpha \) and \( \eta \), respectively). Also, it allows a somewhat different biological interpretation. For instance, \( \rho \) can be treated as the density of individuals who recovered from the disease but cannot become susceptible again, e.g., because they get immunized. In this case, \( i \) gives the density of sick individuals. Alternatively, however, \( \rho \) can be treated as the density of sick individuals; in this case, \( i \) gives the density of the individuals who have the disease in the latent stage. More details and further references can be found in Hethcote (2000) and Diekmann and Heesterbeek (2000).

Some typical simulation results are shown in Figures 12.18 to 12.20. The snapshots were obtained for the same initial conditions as for the SI model (cf. (12.12)–(12.13) with apparent change of notations), and assuming that at the beginning of the disease spread the removed subpopulation is absent, \( \rho(x, y, 0) = 0 \).

Since the SIR model has additional feedbacks, e.g., through possible recovering of the removed individuals, the system response to variation of the mortality \( \delta \) of infected is more complicated than it is in the SI model. For that reason, in our search for the regime of patchy spread, it appears more convenient to vary \( \beta \), not \( \delta \), and to keep all other parameters fixed. Specifically, the simulation results shown here were obtained for \( \epsilon = 0.5 \), \( \alpha = 0 \), \( \gamma = 4 \), \( \delta = 0 \), \( \eta = 0.1 \), \( \omega = 0.8 \), and \( \sigma = 0.5 \). Note that, in our choice of parameter values, we are more inclined to consider \( \rho \) as the density of the sick subpopulation and \( i \) as the latent subpopulation; that is why we neglect the mortality rate of infected (\( \delta = 0 \)) and choose \( \epsilon < 1 \).

For the corresponding one-dimensional case, the species spread from the place of original introduction would occur (for sufficiently small values of \( \beta \)) through propagation of a traveling population front, in exactly the same
FIGURE 12.18: (See color insert.) The two-dimensional density of infected individuals calculated in the SIR model (12.17)–(12.19) at the moments $t = 0$, $t = 100$, $t = 200$, and $t = 400$ (left to right, top to bottom) for $\beta = 0.26$; other parameters are given in the text. The density of susceptibles and removed exhibits essentially the same properties except for very early stage of the system dynamics.

way as it takes place for the prey–predator and the SI models considered above. Depending on $\beta$, the species distribution behind the front can be either homogeneous and stationary or patchy and transient. In all those cases, the species spread in the corresponding two-dimensional system takes place through an expanding population front of circular shape.

For somewhat larger $\beta$, the regime of spread in the one-dimensional system turns to the propagation of separate patches, or groups of patches, qualitatively similar to the pattern shown in Figure 12.6; see Petrovskii et al. (2005) for further details. Remarkably, for the same value of $\beta$, the species spread in the two-dimensional system still takes place through an expanding circular front; see Figure 12.18.

A further increase in $\beta$ leads to species extinction in the one-dimensional system. In the two-dimensional system, however, for the same parameter when species goes extinct in the one-dimensional case, the evolution of the initial population distribution does not lead to species extinction but to its patchy spread; see Figure 12.19. Larger values of $\beta$ make the patchiness of
FIGURE 12.19: (See color insert.) The two-dimensional density of infected individuals calculated in the SIR model at the moments $t = 0$, $t = 200$, $t = 700$, and $t = 1300$ (left to right, top to bottom) for $\beta = 0.273$; other parameters are the same as in Figure 12.18. The density of susceptibles and removed exhibits essentially the same properties.

the spatial pattern even more distinct and the rate of spread notably lower. These results confirm our earlier observation that the regime of patchy spread provides a mechanism of species invasion at the edge of extinction.

A further increase in $\beta$ does not immediately lead to species extinction in two dimensions but first to formation, in the large-time asymptotic, of stationary patchy distribution of the species; see Figure 12.20. After the transient stage, which takes a considerable time, $t \simeq 1500$, a few stationary patches of the population density appear, see the bottom of the figure. Large-time simulations confirm that the patches remain stationary. The number of patches depends on the parameter values while the initial conditions may affect both the number of patches and their position. Recall that the formation of stationary patches was also observed in the two-dimensional prey–predator system in a narrow parameter range between patchy invasion and extinction.

Therefore, in this section we have shown that the “response” of the invasive species to an effort of biological control based on intentional introduction of an infection disease is similar to that observed in the case of biological control through predation. In particular, by means of numerical simulations
we showed that an increase in the disease “strength” may change the standard invasion scenario by a traveling population front to patchy invasion, provided the local population growth of the alien species is damped by the strong Allee effect.

As previously for the prey–predator model, we have shown that patchy invasion is a mechanism of species spread “at the edge of extinction” so that a small change of controlling parameters can bring a given invasive species to extinction. For those parameter values when the patchy invasion takes place in a two-dimensional system, in the corresponding one-dimensional system the species go extinct.
12.4 Concluding remarks

We have already shown earlier in this book (see Chapter 10) that biological invasion may trigger spatiotemporal pattern formation in the wake of the propagating population front. In this chapter, we have extended those results onto the case when the growth rate of an invasive species is affected by the strong Allee effect. We have specifically focused on the situation when the invasive species are subjected to biological control. The agents of biological control were considered to be either predation or infectious disease, which means that, respectively, either a relevant predatory species or some lethal virus are deliberately introduced in the wake of the spreading pest population. In both cases, we have shown that the interplay between the Allee effect and predation or infection then makes the system's spatiotemporal dynamics much richer, resulting in invasion scenarios that have not been observed otherwise. In particular, we have shown that invasive species spread may take the form of “patchy invasion” when formation of a distinct irregular patchy spatial population structure (where patches of high population density are separated by large areas of virtually empty space; see Figure 12.10) is not preceded by a propagation of a continuous population front.

Patchy invasion is shown to be a scenario of species invasion “at the edge of extinction,” so that a reasonably small change in the value of a controlling parameter would turn the population to extinction. Moreover, it appears to possess some rather unusual and counter-intuitive properties: A closer look at the statistics of the patch dynamics shows that the corresponding species spread takes place with the typical values of population density well below the survival threshold.

We mention here that, as in the case of patterns in the wake, formation of the spatiotemporal pattern in the course of patchy invasion was observed when diffusivity was the same for both species. Therefore, the pattern formation is obviously not the result of the Turing mechanism (which requires sufficiently different diffusion coefficients; see Chapter 9) and has a completely different origin. However, we also want to emphasize that existence of patchy spread is not restricted to the case of identical diffusivity. Simulations show that all invasion scenarios considered in this chapter remain qualitatively the same when $D_2/D_1$ is not equal to unity but remains on the order of unity. In particular, patchy invasion with the properties as described above can certainly be observed in the range $0.7 < D_2/D_1 < 1.5$, although other parameters might have to be chosen slightly differently.

Also, patchy invasion appears to be robust with respect to external heterogeneity when some of the system parameters may become a function of the position in space. (Obviously, such position dependence would mimic the properties of real nature where species spread often takes place in a heterogeneous environment or fragmented landscape.) Namely, Morozov et al. (2006)
showed that, in a relevant parameter range, invasive species spread still follows the patchy invasion scenario over the space with a checkboard-type heterogeneity, where “good” patches alternate with “bad” patches on a rectangular grid, even when the environmental conditions inside the bad patches are very unfavorable so that the corresponding spatially homogeneous population would go extinct.

Another curious regime of the dynamics of the systems with a strong Allee effect is the formation of stationary patches, e.g., see Figure 12.20. Remarkably, field studies give many examples when, after introduction, invasive species remain localized inside a certain area for a long time, so that their local invasion and subsequent regional persistence is not followed by geographical spread. There exist a number of different explanations of this phenomenon such as the impact of environmental borders, the existence of time-lags related to mutations and evolutionary changes caused by adaptation in the new environment, etc. The results of this chapter provide another plausible explanation showing that invasive species can be held localized due to purely dynamical mechanisms, i.e., due to the interplay between the Allee effect and predation.

Now, an important question is whether a scenario qualitatively similar to patchy invasion can ever be observed in real nature. A traditional theoretical approach to species spread during biological invasion, which ascends to seminal papers by Kolmogorov et al. (1937) and Fisher (1937), predicts invasion through the propagation of a population front separating invaded areas (behind the front) with high population density of the alien species from uninvaded areas (in front of the front) where the species is yet absent. For a two-dimensional case, this approach has been extended to predict a continuous propagating front (Skellam, 1951; Andow et al., 1990; Okubo et al., 1989). However, there has been a growing amount of empirical evidence recently, both in published literature (Shigesada and Kawasaki, 1997; Davis et al., 1998; Kolb et al., 2004; Swope et al., 2004) and on the Web, showing that, in some cases, invasion of exotic species takes place through dynamics of separate population patches not preceded by propagation of any continuous population front at all. By way of example, Figure 12.21 shows maps of the gypsy moth invasion in the United States. It is readily seen that the infested area has a distinct patchy structure and there does not exist anything that, even with a large bit of imagination, might be regarded as a continuous boundary. Note that, if we exclude a few heavily infested areas, the typical size of the remaining patches is estimated to be on the order of a few dozen kilometers, which is in an encouraging agreement with the theoretical prediction; see the last paragraph in Section 12.2.1.

It would be rather frivolous, though, to try to relate all cases of patchy invasion to a single reason. Indeed, there have been suggested several different mechanisms for patchy invasion such as impact of environmental heterogene-
FIGURE 12.21: (See color insert.) Maps of gypsy moth invasion in the Northeast of the United States: top for 1977, bottom for 1981. Red color shows infested areas (By courtesy of Andrew Liebhold; from www.fs.fed.us/ne/morgantown/4557/gmoth/atlas/).

ity (Murray, 1989), “stratified diffusion” (Shigesada et al., 1995), transport with either humans or vector species (Shigesada and Kawasaki, 1997), impact of stochastic factors (Lewis, 2000; Lewis and Pacala, 2000), etc. It is not always easy to distinguish between the impact of these factors in field data, even though they may sometimes act on a different spatial scale. In most cases, the information currently available does not make it possible to establish an unambiguous relation between the pattern and the underlying process or processes, and additional studies are required. Thinking about a specific example, the first idea that may come to mind when looking at the maps

A behavioral population-level response to an overcritical increase in population density when flocks/swarms of a given species migrate out of population range in order to establish new colonies.
of gypsy moth invasion (cf. Figure 12.21) is that it is probably habitat frag-
mentation that results in the patchy spread. However, comparison with the

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corresponding vegetation maps readily reveals that the correlation is actually
not that strong. On the other hand, in order to link it to the patchy invasion
due to the interplay between the Allee effect and predation, one should first
prove the existence of the strong Allee effect in the gypsy moth population
growth and identify the relevant predatory species. Therefore, it would be
premature to name the dynamical mechanism of patchy invasion considered
in this chapter as an explanation of these or other similar data. Nevertheless,
we think that this mechanism is important in a wider theoretical perspective
because it shows that a basic prey–predator interaction in space and time is
intrinsically capable of generating a patchy invasion without any additional
assumptions. In that sense, it gives a “minimum model” of the phenomenon.
Part IV

Spatiotemporal patterns and noise
Chapter 13

Generic model of stochastic population dynamics

Mother nature is noisy, therefore, it is time to deal with stochasticity. The evolution of deterministic systems is fixed by the initial and boundary conditions, though a forecast is impossible for chaotic dynamics on large time scales. In stochastic systems, the noise leads to different realizations for the same initial conditions. The statistical ensemble of infinitely many realizations defines a stochastic process. For simplicity, only Markov processes will be considered here, where the present state determines the further evolution. The interplay of stochasticity and determinism can be modeled by stochastic (partial) differential equations [S(P)DE’s] on the level of state variables (Langevin equations) or by dynamical equations for different probability densities (diffusion–reaction master and Fokker–Planck equations). Nice introductions to the theory of stochastic processes and its applications have been written by Gardiner (1985); Allen (2003); Anishenko et al. (2003), and with regard to spatial processes by García-Ojalvo and Sancho (1999).

On average, the system dynamics might remain unchanged. However, the noise generates a corridor in phase space for the dynamics rather than a single trajectory and only certain probabilities for steady states or oscillations and waves. But this is not the only effect of noise. It will be shown by example that it can induce transitions between steady states in systems with steady-state multiplicity. Furthermore, it can generate unexpected new structures in space and time. A side effect is that the noise blurs unrealistic distinct and symmetric spatial and spatiotemporal patterns.

Population-dynamical systems are subjected to internal demographic and external environmental noise. Demographic noise describes stochastic fluctuations caused by the random and discrete nature of individual growth, interaction, and motion. It is especially important at small population sizes, i.e., low species numbers, because any fluctuation can become dangerous and finally cause extinction. The impact of demographic noise on the system dynamics can be described by (diffusion–reaction) master equations. The latter concept originated from mathematics and physics; cf. Wax (1954); Bharucha-Reid (1960); Arnold (1974); Kuramoto (1974); and Karlin and Taylor (1975) with early and then most applications in physical chemistry, especially chemical kinetics (Bartholomay, 1958b; McQuarrie, 1967; Janssen, 1974; Haken, 1975; Matheson et al., 1975; van den Broeck et al., 1977; Feistel and Ebeling,
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For larger population sizes, the demographic noise is masked by the environmental (extrinsic, external) random variability that induces parameter fluctuations (Bonsall and Hastings, 2004). The latter can be incorporated in a straightforward manner by applying the white noise forcing directly to one or more selected parameters. The values of these parameters are then chosen randomly at each space point and each unit time step from a truncated normal distribution between a minimum and maximum fraction of their spatiotemporal mean. An example with an application to plankton dynamics can be found in Steele and Henderson (1992b). A corresponding problem will be presented in Ch. 14.

Another way to account for noise is the use of stochastic partial differential (Langevin) equations of the form

$$\frac{\partial \phi(\vec{r}, t)}{\partial t} = d \nabla^2 \phi(\vec{r}, t) + f(\phi(\vec{r}, t), \psi) + \omega[\phi(\vec{r}, t)] \xi(\vec{r}, t), \quad (13.1)$$

where $\phi(\vec{r}, t)$ and $f(\phi(\vec{r}, t), \psi)$ are the usual vectors or scalar fields of population densities and growth and interaction functions, respectively. $\psi$ stands for the system parameters. The last term on the right-hand side represents the stochastic force $\xi(\vec{r}, t)$ that disturbs the system with intensity $\omega[\phi(\vec{r}, t)]$. These equations have a long history in mathematical modeling of population dynamics (Levins, 1969; May, 1973; Capocelli and Ricciardi, 1974; Tuckwell, 1974; May et al., 1978; Braumann, 1979, 1983; Kliemann, 1983; Dennis and Patil, 1984). Throughout the chapter, it is assumed that $\xi(\vec{r}, t)$ is a Gaussian white noise with zero mean and delta correlation

$$\langle \xi(\vec{r}, t) \rangle = 0, \langle \xi(\vec{r}_1, t_1) \xi(\vec{r}_2, t_2) \rangle = \delta(\vec{r}_1 - \vec{r}_2) \delta(t_1 - t_2). \quad (13.1a)$$

$\omega[\phi(\vec{r}, t)]$ is the density-dependent noise intensity. The postulate of parenthood (Hutchinson, 1978) in population dynamics requires this density dependence, i.e., multiplicative noise. Furthermore, it is chosen as

$$\omega[\phi(\vec{r}, t)] = \omega \phi(\vec{r}, t); \quad \omega = \text{const} \quad (13.1b)$$

which reflects an increase of noise with growing species numbers. In particular, such noise is originated by fluctuating mortalities. But it is not intended to specify the origin of the noise. Colored noise (Kaitala et al., 1997), i.e., different shapes of Equations (13.1a) and (13.1b) would result in similar effects.
In this respect, the action of the considered fluctuations in (13.1a) and (13.1b) is structurally robust and a good approximation of environmental noise. One should keep in mind that the numerical treatment of spatially two-dimensional white noise problems introduces a spatial correlation length that is the grid spacing (Walsh, 1986; Lythe and Habib, 2001; Milstein and Tretyakov, 2004). General results state that the continuum solutions of the presented stochastic partial differential equations driven by space-time white noise in two spatial dimensions are not continuous functions but only distributions. Nevertheless, a discrete lattice can be used to resemble a spatially two-dimensional setting.

Further on, Stratonovich calculus (Stratonovich, 1967; Anishenko et al., 2003) will be applied for the interpretation of the multiplicative white noise during simulations. There has been and possibly still is a controversy on using Stratonovich or Itō (1961) calculus, however, Braumann (1999, 2007) has shown that this issue is merely semantic.

A quite general form of a model of the interplay determinism and stochasticity in population dynamics is with restriction to two populations $U, V$ at location $\vec{R}$ and time $T$:

$$\frac{\partial U(\vec{R}, T)}{\partial T} = D_U \nabla^2 U + \omega U \Xi(\vec{R}, T) + \Phi_U(\vec{R}, T) + P(U) - E(U, V) - M_{IU} I_U, \quad (13.2)$$

$$\frac{\partial V(\vec{R}, T)}{\partial T} = D_V \nabla^2 V + \omega V \Xi(\vec{R}, T) + \Phi_V(\vec{R}, T) + Q(V) - \kappa E(U, V) - M_{IV} I_V. \quad (13.3)$$

The function $P(U)$ describes the intrinsic growth of the prey population, usually logistic growth here. $E(U, V)$ stands for predation usually of Holling type II or III. Compared to previous chapters, the stochastic force $\Xi(\vec{R}, T)$, in- and outflows $\Phi_U(\vec{R}, T), \Phi_V(\vec{R}, T)$, and additional mortality rates $M_{IU}, M_{IV}$ due to a possible disease as well as intrinsic growth $Q(V)$ of population $V$ have been added. Later on, an infectious disease of $U$ will be considered; then, the total population of $U$ will be split into a susceptible part $S$ and an infected $I$ with $U = S + I$. Equation (13.2) then splits into

$$\frac{\partial S(\vec{R}, T)}{\partial T} = D_S \nabla^2 S + \omega S \Xi(\vec{R}, T) + \Phi_S(\vec{R}, T) + P(S, I) - E(S, I, V) - \Sigma(S, I), \quad (13.2a)$$

$$\frac{\partial I(\vec{R}, T)}{\partial T} = D_I \nabla^2 I + \omega I \Xi(\vec{R}, T) + \Phi_I(\vec{R}, T) + P(S, I) - \kappa E(S, I, V) + \Sigma(S, I) - M_I I; \quad (13.2b)$$
whereas Equation (13.3) reduces to

$$\frac{\partial V(\vec{R}, T)}{\partial T} = D_V \nabla^2 V + \omega V \Xi(\vec{R}, T) + \Phi_V(\vec{R}, T) + Q(V) + \kappa E(S, I, V).$$

(13.3a)

The function \(\Sigma(S, I)\) describes the mechanism of disease transmission (Nold, 1980; de Jong et al., 1995; Hethcote, 2000; McCallum et al., 2001). Here, only two mechanisms will be considered. One is the mass-action type

$$\Sigma(S, I) = \sigma SI,$$

(13.4)

the other the frequency-dependent transmission

$$\Sigma(S, I) = \sigma SI/U.$$

(13.5)

Following McCallum et al. (2001), for a directly transmitted pathogen, the infection rate is the product of three factors: first, the contact rate, second, the fraction of these contacts that take place with susceptibles, and third, the fraction of contacts that finally lead to infections. The mass-action type suggests that the contact rate of susceptibles and infected that leads to infection is directly proportional to density. On the other hand, the frequency-dependent type is independent of the host density. For randomly mixed susceptibles and infected, the transmission goes with \(\sigma SI/U\). On average, each susceptible \(S\) will make the same number of contacts independent of the host density, and a fraction \(I/U\) of these contacts will be with infected. This frequency-dependent type is often assumed for sexually transmitted diseases (May and Anderson, 1987, 1988; Barlow, 1994). However, Beltrami and Carroll (1994) have applied this approach to the modeling of viral diseases in phytoplankton. They have assumed that the number of infected \(I\) is much smaller than the number of susceptibles \(S\). Therefore, the term \(I\) can be neglected in the denominator and the transmission becomes proportional to the number of infected. This assumption will be applied further on to models with virally infected phytoplankton.
Chapter 14

Noise-induced pattern transitions

14.1 Transitions in a patchy environment

To begin with, the influence of parametric noise on the pattern formation in the Rosenzweig-MacArthur (1963) prey-predator model is investigated. The latter model has been used by Scheffer (1991a) for specifying the phytoplankton–zooplankton dynamics in a shallow lake under the control of nutrient density and of planktivorous fish stock. Structures in a deterministic environment have been presented by Malchow (1993, 2000a). The model reads in dimensionless quantities

\[
\begin{align*}
\frac{\partial u}{\partial t} &= ru(1 - u) - \frac{au}{1 + bu} v + du \Delta u, \\
\frac{\partial v}{\partial t} &= au \frac{v}{1 + bu} v - \frac{g^2 v^2}{1 + h^2 v^2} f + dv \Delta v.
\end{align*}
\]

(14.1)

(14.2)

The environmental heterogeneity is described through a simple approach: the considered \(L \times 2L\) model area is divided into three habitats of sizes \(L \times L/2\), \(L \times L\) and \(L \times L/2\) respectively; cf. Figure 14.1. The following model parameters have been chosen for the simulations, cf. Pascual (1993); Malchow et al. (2000, 2002):

\[
\langle r \rangle = 1, \quad a = b = 5, \quad g = h = 10, \quad \bar{m} = 0.6, \quad f = 0, \quad (14.3)
\]

\[
L = 100, \quad x \in [0, L], \quad y \in [0, 2L], \quad d_u = d_v = 5 \times 10^{-2}. \quad (14.4)
\]

\(\langle r \rangle\) is the spatially averaged prey growth rate \(r(x, y)\) whereas \(\bar{m}\) stands for the spatiotemporal mean of the noisy predator mortality rate \(m_v(x, y, t)\). The value of \(m_v\) is randomly chosen at each space point and each unit time step from a truncated normal distribution between \(I = 0\) and 15% of \(\bar{m}\), i.e. \(m(x, y, t) = \bar{m}[1 + I - \text{rndm}(2I)]\) with \(\text{rndm}(z)\) as a random number between 0 and \(z\). The upper layer has double mean phytoplankton productivity \(2\langle r \rangle\); the bottom layer only 60% of \(\langle r \rangle\). Both are coupled by the middle habitat with linearly decreasing productivity. The latter gradient reflects assumptions by Pascual (1993). The chosen model parameters generate limit cycles at each space point, i.e., one has a kind of continuous chain of diffusively
coupled nonlinear oscillators. The spatial setting yields a fast spatially uniform prey–predator limit cycle in the top habitat, continuously changing into quasiperiodic and chaotic oscillations and waves along the productivity gradient in the middle, coupled to slow spatially uniform limit cycle oscillations in the lower layer.

FIGURE 14.1: Left: Gradual change of mean prey growth rate in space. Neumann boundary conditions at bottom and top boundaries. Periodic boundary conditions at left- and right-hand sides. Right: Homogeneous initial condition.

14.1.1 No noise
At first, the pattern formation due to the layered spatial structure of the environment is studied. Fish and environmental noise are ignored. Five snapshots of a long-term simulation of the spatial and temporal dynamics have been taken and are presented in Figure 14.2.

The densities in the upper layer oscillate rather quickly throughout the simulation. The diffusively coupled limit cycles along the gradient in the middle layer induce a transition from periodic oscillations near the upper border to quasiperiodic in the middle part and to chaotic oscillations near the lower border (Pascual, 1993) which couple to the slowly oscillating bottom layer. The latter slow oscillator is not strong enough to fight the chaotic forcing from above. Finally, chaos prevails in the underpart of the model area.

14.1.2 Noise-induced pattern transition
For a weak 10% noise intensity, patterns are formed that are very similar to those in section 14.1.1 without noise. The spatial structures remain qual-
FIGURE 14.2: No noise. Rapid oscillations in the upper layer due to high prey growth rate. Formation of plane waves with spatiotemporal quasiperiodicity and chaos with decreasing prey growth rate. These plane waves compete with the rapid oscillations in the top and the slower in the bottom layer but can invade only the latter.

Itatively the same, however, the noise enhances the spread of the wavy and chaotic part towards the upper layer. Furthermore, the boundaries of the layers become blurred. A slightly higher noise intensity of 15% shakes the results up.

FIGURE 14.3: Fifteen percent noise. The noise lets the plane waves break up to spirals that first occupy the bottom layer again. However, due to the noisy forcing, the spirals are able to also invade the top layer, and the system undergoes a pattern transition.
The wavy and chaotic region on the lower side “wins the fight” against the upper regular structures and invades the whole space. This corresponds to a pronounced noise-induced transition (Horsthemke and Lefever, 1984) from one spatiotemporally structured dynamical state to another. This transition can also be seen in the local power spectra, which have been computed for the upper layer close to the upper reflecting boundary (Malchow et al., 2002).

**FIGURE 14.4:** Power spectra for increasing noise intensities at $x = 50$ and $y = 5$.

The increase to 15% lets the periodicity disappear and a nonperiodic system dynamics remains. This is another proof of the noise-induced transition from periodical to aperiodical local behavior in the upper part of the model area after crossing a critical value of the external noise intensity.

### 14.2 Transitions in a uniform environment

Now, the stochastic variability is modeled through Langevin equations 13.1. At first, the influence of external noise on the formation of Turing patterns in model (14.1)–(14.2) is investigated.
14.2.1 Standing waves driven by noise

Parameter intervals for the occurrence of diffusive and/or advective instabilities of a spatially homogeneous stationary solution against supercritical wave perturbations have been assessed and given in detail, cf. Malchow (1995, 2000a); Satnoianu and Menzinger (2000) and Satnoianu et al. (2000).

The following set of model parameters has been chosen for the simulations described in this section, cf. Malchow (2000a):

\[
\begin{align*}
    r &= 1, \ a = 8.0, \ b = 11.905, \ g = 1.434, \ h = 0.857, \ m_v = 0.49, \ f = 0.093, \\
    L &= 100, \ x, y \in [0, L], \ d_u = 2.8 \times 10^{-5}, \ d_v = 5.6 \times 10^{-3}.
\end{align*}
\]

\[FIGURE 14.5: \text{Formation of a symmetric Turing structure after perturbation of the homogeneous solution with a central circular wave of supercritical wave number; cf. Malchow (2000a). Neumann boundary conditions. No noise.}\]

The system is integrated using the explicit Euler-Maruyama scheme (Maruyama, 1955; Kloeden and Platen, 1999) for the stochastic interaction part and a Peaceman-Rachford alternating-direction implicit (ADI) scheme for diffusion (Peaceman and Rachford Jr., 1955; Thomas, 1995). For the noise term, random numbers are generated using the Mersenne Twister (Matsumoto and Nishimura, 1998), normally distributed by the Box-Muller method (Box and Muller, 1958).

The simulations start with a wave of supercritical wave number in the center that destabilizes the spatially homogeneous species distribution, resulting in that funny fully symmetric 5-eyes pattern; cf. Figure 14.5. Here and further on, the prey pattern is displayed on a greyscale from black \((u = 1)\) to white \((u = 0)\).

Besides the strong conditions on the difference of the diffusion coefficients, the symmetry and polarity of Turing patterns have long been a reason to question the role of those instabilities and structures in biodynamics because nature is not that symmetric in a mathematical sense. We will see now how a little noise changes the patterns completely. The same initial perturbation
wave is used, however, the system is subject to increasing noise. The result is seen in Figure 14.6.

The pattern symmetry is immediately disturbed and the 5-eyes pattern disappears. Finally, we have an asymmetric Turing pattern even with varying sizes of the empty (white) patches. The disturbing symmetry of diffusion-induced Turing patterns has been overcome by some noise that is present everywhere in natural systems. Furthermore, noise has induced transitions between different structures that has also been shown in Section 14.1.2.
Chapter 15

**Epidemic spread in a stochastic environment: plankton as a paradigm**

There are many publications on pattern formation and chaos in minimal prey–predator models, also for phytoplankton-zooplankton dynamics with patchiness and blooming that will be in the focus of this chapter. In contrast, not so much is known about marine viruses and their role in aquatic ecosystems and the species that they infect; for reviews cf. Fuhrman (1999); Wommack and Colwell (2000) as well as Suttle (2000, 2005). Jacquet et al. (2002) have found that viral infection might accelerate the termination of phytoplankton blooms.

There are two types of viral replication cycles. Contrary to lytic infections with destruction and without reproduction of the host cell, lysogenic infections are a strategy whereby viruses integrate their genome into the host’s genome. As the host reproduces and duplicates its genome, the viral genome reproduces, too. The understanding of the importance of lysogeny is just at the beginning (Wilcox and Fuhrman, 1994; Jiang and Paul, 1998; McDaniel et al., 2002; Ortmann et al., 2002). Cochran et al. (1998) report that many environmentally important pollutants may be inducing agents for natural lysogenic viral production in the marine environment.

Mathematical models of the ecoepidemiology of virally infected phytoplankton populations are rare as well. The classical publication is by Beltrami and Carroll (1994). Recent work is of Chattopadhyay et al. (2002; 2002; 2003; 2004). The latter deals with lytic infections and mass action incidence functions. Hilker and Malchow et al. have observed oscillations and waves in a phytoplankton-zooplankton system with Holling type II (Malchow et al., 2004; Hilker and Malchow, 2006; Hilker et al., 2006) and III (Malchow et al., 2005) grazing under lysogenic viral infection and proportionate mixing incidence function (frequency-dependent transmission) (Nold, 1980; Hethcote, 2000; McCallum et al., 2001).

In this chapter, the focus is on modeling lysogenic and lytic infections and frequency-dependent transmission under multiplicative noise and its impact on the local and spatiotemporal dynamics of interacting phytoplankton and zooplankton. The Rosenzweig-MacArthur prey–predator model is used again. Starting with the local stationary behavior and for lytic infections, the local...
coexistence on a strange periodic attractor is shown to be possible even under nonstationary conditions. Then, lysogenic infections are considered to remarkably simplify the studies. The final section deals with noise-induced pattern formation in a slow-fast, excitable, prey–predator dynamics with structures like local coherence resonance, global synchronization, and the generation of stationary spatial patterns.

15.1 Model

Starting with model (14.1)–(14.2), the phytoplankton population $u$ is split into a susceptible part $s$ and an infected portion $i$. Zooplankton $v$ grazes on both susceptible and infected phytoplankton. Then, the model system reads for symmetric inter- and intra-specific competition of susceptibles and infected

$$
\frac{ds}{dt} = r_s s (1 - u) - \frac{a s}{1 + b u} v - \sigma \frac{s i}{u}, \quad (15.1a)
$$

$$
\frac{di}{dt} = r_i i (1 - u) - \frac{a i}{1 + b u} v + \sigma \frac{s i}{u} - m_i i, \quad (15.1b)
$$

$$
\frac{dv}{dt} = a u \frac{1}{1 + b u} v - m_v v. \quad (15.1c)
$$

A frequency-dependent transmission rate $\sigma$ as well as an additional disease-induced mortality of infected (virulence) with rate $m_i$ are assumed. The intrinsic growth rates of susceptibles and infected are $r_s$ and $r_i$, respectively. In the case of lysogenic infection, it holds that $0 \leq r_i \leq r_s$, whereas in the case of lytic infection, $r_i \leq 0 \leq r_s$. Then, the first term on the right-hand side of (15.1b) describes the losses due to natural mortality and competition.

Now, it is searched for stationary and oscillatory solutions of the system (15.1a)–(15.1c) which is simplified for that through a convenient transformation, then describing the dynamics of the total phytoplankton population $u = s + i$ and the prevalence $p = i/u$. The vector of population densities and prevalence is $X = \{u, p, v\}$, and the model equations read

$$
\frac{du}{dt} = \left[ r_s (1 - p) + r_p (1 - u) u - \frac{a u}{1 + b u} v - m_i p u, \quad (15.2a)
$$

$$
\frac{dp}{dt} = \left[ (r_i - r_s)(1 - u) + (\sigma - m_i)(1 - p) p, \quad (15.2b)
$$

$$
\frac{dv}{dt} = a u \frac{1}{1 + b u} v - m_v v. \quad (15.2c)
$$

System (15.2a)–(15.2c) has the following trivial and semitrivial equilibria
Epidemic spread in a stochastic environment

$E = \{u^S, p^S, v^S\}$ with

$$
\left. \frac{du}{dt} \right|_{X=E} = \left. \frac{dp}{dt} \right|_{X=E} = \left. \frac{dv}{dt} \right|_{X=E} = 0 :
$$

1) $E_{00} = \{0, 0, 0\}$.
   The trivial state is always unstable.

2) $E_{01} = \{0, p_{01}^S, 0\}$ with $p_{01}^S = 1$.
   This disease-induced extinction of the total prey population is possible for $r_i < m_i < \sigma + r_i - r_s$.

3) $E_1 = \{u_1^S, 0, 0\}$ with $u_1^S = s = 1$.
   Only the susceptible prey species survive at their carrying capacity for $\sigma < m_i$ and $m_v > a/(1 + b)$.

4) $E_2 = \{u_2^S, p_2^S, 0\}$ with $u_2^S > 0$, $p_2^S > 0$.
   a) $E_{21} = \{u_{21}^S, p_{21}^S, 0\}$ with $u_{21}^S = i = 1 - m_i/r_i$, $p_{21}^S = 1$.
      For $m_i < r_i$, $m_i < (r_i/r_s)\sigma$ and $au_{21}^S/(1 + bu_{21}^S) < m_v$, only the infected can survive.
   b) $E_{22} = \{u_{22}^S, p_{22}^S, 0\}$ with

   $$
u_{22}^S = 1 - \frac{\sigma - m_i}{r_s - r_i}, \quad p_{22}^S = \frac{r_s \sigma - m_i}{\sigma (r_s - r_i)}.
$$
   The computer-aided stability analysis of this solution yields some the lengthy expressions for the stability intervals that do not provide further insight and are omitted here.

5) $E_3 = \{u_3^S, 0, v_3^S\}$ with

   $$
u_3^S = \frac{m_v}{a - m_v b}, \quad v_3^S = \frac{r_s}{a} (1 + bu_3^S)(1 - u_3^S).
$$
   The infected go extinct for a too low transmission rate or a too high virulence. The remaining $u-v$ prey–predator model is a well studied textbook example. The solution can be a stable node or focus. An unstable focus bound by a stable limit cycle may appear after a Hopf bifurcation.

Nontrivial equilibria, i.e., the coexistence of all species, only exist for a single combination of parameters. Then, there is a continuum of stationary states:
6) \( E^S_\alpha(p) = \{ u^S_\alpha, v^S_\alpha(u^S_\alpha, p) \} \) with \( u^S_\alpha > 0, \ 0 < p < 1, \ u^S_\alpha(u^S_\alpha, p) > 0 \).

From Equation (15.2b) and (15.2c) one finds

\[
\begin{align*}
u^S_{41} &= 1 - \frac{\sigma - m_i}{r_s - r_i} \quad \text{and} \quad (15.3a) \\
u^S_{42} &= \frac{m_v}{a - m_v b}, \quad (15.3b)
\end{align*}
\]

which define two parallel planes independent of \( p \) and \( v \) in \((u\text{-}p\text{-}v)\) phase space. These planes are orthogonal to the \((u\text{-}v)\) and parallel to the \((p\text{-}v)\) plane. Both must coincide, i.e., the system parameters have to strictly obey the relationship

\[
1 - \frac{\sigma - m_i}{r_s - r_i} = \frac{m_v}{a - m_v b} = u^S_4. \quad (15.4)
\]

From Equation (15.2a), one obtains the plane

\[
v = v(u, p) = \frac{1 + bu}{a} \{ r_s(1 - u) + [(r_i - r_s)(1 - u) - m_i] p \} \quad (15.5)
\]

All points \( E^S_\alpha(p) \) on the straight intersection line of planes (15.4) and (15.5),

\[
v^S_4(u^S_\alpha, p) = v(u^S_\alpha, p) \quad \text{with} \quad u^S_\alpha \quad \text{as in} \quad (15.4), \quad (15.6)
\]

are stationary states independent of \( p \) for \( 0 < p < 1 \). This line is a heteroclinic connection between the semitrivial equilibria \( E_2 \) and \( E_3 \).

For \( m_v < a(b - 1)/(b(b + 1)) \), the \( u\text{-}v \) subsystem exhibits an unstable focus bound by a stable limit cycle. In the stationary case (15.4), a numerical stability analysis shows that all equilibria \( E^S_\alpha(p) \) on line (15.6), including \( E_2 \) and \( E_3 \), are degenerated, i.e., their third eigenvalue is zero.

The upper part of line (15.6) consists of degenerated unstable foci. A fold-Hopf (zero-pair) bifurcation point (Kuznetsov, 1995; Nicolis, 1995) separates them from the lower part closer to the \((u\text{-}p)\) plane with degenerated stable foci. The result of a corresponding numerical simulation, starting on the unstable upper branch, is shown in Figure 15.1. Such a bifurcation has also been called a zip bifurcation (Farkas, 1984), because a singular curve folds into periodic solutions when a parameter is varied. This parameter is the decreasing prevalence here.

The closer the initial condition to the \((u\text{-}v)\) plane, the longer is the journey along the trajectory through phase space. The final position of the stationary state on the line strongly depends on the initial conditions. Therefore, the final positions are only neutrally stable. This remains
true in the case when the intersection point of (15.6) in the \( u-v \) sub-
system is a stable focus or stable node, and the line (15.6) becomes a
continuum of stable solutions.

The growth rate of infected \( r_i \) has been set to zero. This choice de-
scribes the cell lysis of infected phytoplankton cells and nonsymmetric
competition of infected and susceptibles; the infected still have an im-
 pact on the growth of susceptibles by shading and space demand, but
not vice versa. Furthermore, \( m_i \) now stands for an effective mortality,
i.e., the additional disease-induced mortality (virulence) plus the natural
mortality of the infected.

15.2 Strange periodic attractors in the lytic regime

The strong parameter relationship (15.4) is surely not realistic. The prob-
ability to find such a fixed combination in nature is almost zero. Therefore,
nonstationary situations will be computed now, with parameter settings when
the planes (15.3a) and (15.3b) do not coincide and the intersection lines with
plane (15.5) are no longer stationary.

First, the virulence is increased. A computer-assisted bifurcation and sta-
bility analysis shows that under these nonstationary conditions \( E_2 \) is a saddle-
focus with a stable two-dimensional manifold and an unstable one-dimensional
manifold. In the \( (u-v) \) plane, \( E_3 \) is also a saddle-focus, but with an unstable
two-dimensional manifold and a stable one-dimensional manifold. In Fig-
ure 15.2, the trajectory starts in the upper corner and approaches the lower
end point of the right-hand line (15.3a) in the \( (u-p) \) plane which is the semi-
trivial stationary state \( E_2 \). This is the mentioned saddle-focus with stable
oscillation but unstable in the direction of \( v \). Therefore, the trajectory is
shot along the heteroclinic connection to the \( (u-v) \) plane and gets into the
sphere of influence of the end point of the left-hand line (15.3b). This is also
a semitrivial saddle-focus, namely \( E_3 \) with unstable oscillation and stable in
\( v \) direction. Thus, the trajectory bounces back, spirals down the lines and
“tube-rides” up again and again. In other terms, on the way up, it is “rein-
jected” (Nicolis, 1995) and tunnels through the two formed funnels. This is
illustrated in Figure 15.2a. It resembles the movement on a torus, where the
centre hole of the torus is shrunk to a thin tube. However, the precessing
trajectory gets “phase-locked” and finally, for long times, approaches a peri-
odic attractor (Langford, 1983, pp. 233). This is shown in Figure 15.2b. The
oscillation takes place in a plane that is orthogonal to the \( (p-v) \) plane. The at-
tractor surrounds the two intersection points of the two lines of nonstationary
points and the plane of oscillation.

The attractor is called a strange periodic attractor (Hilker and Malchow,
because of the long-lasting very peculiar approach towards the asymptotic oscillations. Obviously, the attractor is not chaotic which is sometimes called “strange.” But in order to underline the impressive long-term transient dynamics, it is named “strangely periodic.”

As for a limit cycle, the position of the asymptotic periodic attractor is independent of the initial conditions. For further increasing values of $m_i$, the behavior of the system becomes simpler. The distinct funnel formation disappears and the periodic attractor is stabilizing faster and faster. For too high virulence, the infected go extinct and the system oscillates in the $u$-$v$ subsystem. This is illustrated in Figure 15.3.

For virulences below the stationary value given in Figure 15.1, $E_2$ becomes a stable and $E_3$ an unstable focus, respectively. Numerical simulations yield that zooplankton dies out and the dynamics relaxes to $E_2$ in the $u$-$p$ subsystem; cf. Figure 15.4.

Summarizing, $E_2$ has undergone a bifurcation from an unstable saddle-focus to a stable focus. At this bifurcation point, the continuum of degenerated non-trivial equilibria $E_{vS}^c(p)$ appeared simultaneously. A zero-pair Hopf bifurcation took place along this continuum line.

The strange periodic attractor allows for the coexistence of all three species

---

**FIGURE 15.1:** (See color insert.) Stationary dynamics of system (15.2a)–(15.2c) with coexistence of all three populations. The trajectory starts at the upper unstable part of the line of stationary points, passes the fold-Hopf bifurcation point, and finally relaxes on the neutrally stable lower part. Parameters: $r_s = 1$, $r_i = 0$, $a = b = 5$, $\sigma = 4/5$ from Equation (15.4), $m_i = 2/15$, $m_v = 5/8$. Initial conditions: $u_0 = 1/3$, $p_0 = 3/40$, $v_0$ from Equation (15.6). The straight line is the continuum of equilibria $E_{vS}^c(p)$, lying on the shaded plane (15.5). With permission from Hilker and Malchow (2006), http://www.informaworld.com.
positive in a substantial range of parameters. This makes the model much more realistic and biologically interesting, because the special condition (15.4) unlikely holds exactly in reality. The nontrivial oscillations in the interior of the first octant are remarkable, because there does not exist a nontrivial stationary state.
FIGURE 15.3: (See color insert.) Dynamics of system (15.2a)–(15.2c), see details in the text. Parameters like in Figure 15.1 except for $m_i$. Initial condition: $u_0 = 1.0$, $p_0 = 0.125$, $v_0 = 0.0015$. With permission from Hilker and Malchow (2006), http://www.informaworld.com.

FIGURE 15.4: (See color insert.) Dynamics of system (15.2a)–(15.2c), see details in the text. Parameters: $m_i = 0.1$, all others like in Figure 15.1. Initial condition: $u_0 = 1.01$, $p_0 = 0.1$, $v_0 = 0.001$. With permission from Hilker and Malchow (2006), http://www.informaworld.com.

15.3 Local dynamics in the lysogenic regime

The richness of the system dynamics is reduced when considering lysogenic infections with $r_s = r_i = r$. For $m_i > \sigma$, the infected go extinct, and for $m_i < \sigma$, the susceptibles do. In the case of $m_i = \sigma$, susceptibles and infected coexist. Because of the symmetry of the growth terms of susceptibles and
infected, the initial conditions determine their final dominance in the endemic state, i.e., if $s(t = 0) > i(t = 0)$ then $s(t) > i(t) \ \forall \ t$. A corresponding example is presented in Figure 15.5 for $r = 1$ and $a = b = 5$. These three parameter values will be kept for all simulations.

It is readily seen that the transformation of model (15.1a)–(15.1c) to system (15.2a)–(15.2c) with $r_s = r_i = r$ reduces the considerations of stationarity and stability to a pseudo-two-dimensional problem because the prevalence can take only three values, i.e., zero for $\sigma < m_i$, unity for $\sigma > m_i$, or its initial value for $\sigma = m_i$. The computations are remarkably simplified because the $u$-$v$ dynamics only proceeds in the plane $p = \text{const}$.

For $\sigma = m_i$, the initial value of the prevalence is an additional control parameter that might drive the system to different dynamic behavior. An example for bistability is shown in Figure 15.6.

After the deterministic local behavior, the spatial dynamics is studied now.

15.4 Deterministic and stochastic spatial dynamics

There is a vast number of publications on the spatiotemporal self-organization in prey–predator communities, modeled by (advection–)diffusion–reaction equations; cf. the references in the introduction. Much less is known about equation-based modeling of the spatial spread of epidemics, a small collection of pa-
In this section, the space-time dynamics of the plankton model (15.1a)–(15.1c) is considered, i.e., zooplankton, grazing on susceptible and virally infected phytoplankton, both diffusing in horizontally two-dimensional space, under the influence of environmental noise. The diffusion terms have been integrated using the Peaceman-Rachford alternating direction implicit (ADI) scheme, cf. Peaceman and Rachford Jr. (1955); Thomas (1995). For the source and noise terms, the explicit Euler-Maruyama scheme has been applied (Maruyama, 1955; Kloeden and Platen, 1999; Higham, 2001). The spatial grid is a 99 × 99 point square with spacing $\Delta x = \Delta y = 1$.

The following series of Figures 15.7–15.10 shows the results of computations of the spatiotemporal dynamics of growth and interaction with parameters from section 15.3, but now including diffusion and noise.

In order to avoid boundary effects, periodic boundary conditions have been chosen for all simulations. Localized patches in empty space have been taken as initial conditions. They are the same for deterministic and stochastic simulations. The first two rows of figures show the dynamics of the susceptibles for deterministic and stochastic conditions, the two middle rows show the infected and the two lower rows the zooplankton.

For Figures 15.7, 15.8, and 15.9, there are two initial patches, one with zooplankton surrounded by susceptible phytoplankton in the upper part of the model area, and one with zooplankton surrounded by infected on the right-hand side of the model area. For Figure 15.10, there are initial central patches of all three species. Susceptibles are ahead of infected which are ahead of zoo-
plankton. This special initial configuration leads at first to the propagation of concentric waves for the deterministic case in rows 1, 3 and 5. These naturally unrealistic waves are immediately blurred and only a leading diffusive front remains for the stochastic case in rows 2, 4 and 6. A stochastic theory of diffusive waves has been developed by Schimansky-Geier et al. (1983); Schimansky-Geier and Züllicke (1991); and van Saarloos (2003).

In Figure 15.7, the final spatial coexistence of all three species for $m_i = \sigma$ is presented. The localized initial patches generate concentric waves that break up after collision and form spiral waves in a deterministic environment. The noise only blurs these unrealistic patterns. The greyscale changes from high population densities in black color to vanishing densities in white.

This changes for $m_i > \sigma$ and $m_i < \sigma$ in Figures 15.8 and 15.9, respectively. While infected or susceptibles die out in the deterministic case, the noise supports their survival and spread under unfavourable conditions.

In Figure 15.10, one can readily see the so-called dynamic stabilization of the locally unstable focus in space for a deterministic environment. A long plateau is formed with a leading diffusive front ahead; cf. Petrovskii and Malchow (2000); Malchow and Petrovskii (2002). Furthermore, the infected seem to be trapped in the center and become almost extinct. The noise fosters the escape, spread and survival of the infected.

The equal growth rates of susceptibles and infected have led to the situation where, in a constant environment, the ratio of the mortality of the infected and the transmission rate of the infection determines coexistence, survival, or extinction of susceptibles and infected. A fluctuating environment supports the survival and the spatial spread of the “endangered” species. Furthermore, noise has not only enhanced the spatiotemporal coexistence of susceptibles and infected but it has been necessary to blur distinct artificial population structures like concentric or spiral waves and to generate more realistic fuzzy patterns.

15.5 Local dynamics with deterministic switch from lysogeny to lysis

Lysogenic viral replication is very sensitive to environmental variability. First, the switch from lysogeny to lysis in the prey is studied for a constant environment. Only one local example for such a switch is drawn in Figure 15.11. After the transition, there is no further replication of infected. $r_i$ is simply set to zero when the switch occurs; cf. Section 15.1. A more technical assumption for the simulation is that the remaining natural mortality of the infected is added to the virulence, leading to a higher effective mortality of the infected, i.e., the parameter $m_i$ increases. Furthermore, the lytic cycle
Spatiotemporal patterns in ecology and epidemiology

FIGURE 15.7: Spatial coexistence of susceptibles (two upper rows), infected (two middle rows), and zooplankton (two lower rows) for $m_i = \sigma = 0.2$, $m_o = 0.5$, $d = 0.05$. No noise $\omega = 0$ and 0.25 noise intensity, respectively. Periodic boundary conditions.

generates many more viruses, i.e., the transmission rate $\sigma$ increases as well. And, finally, the intra-specific competition of the dying infected phytoplankton cells vanishes whereas the interspecific competition of susceptibles and infected becomes nonsymmetric, i.e., the dead and dying infected still influ-
FIGURE 15.8: Spatial coexistence of susceptibles (two upper rows) and zooplankton (two lower rows). Extinction of infected (third row) for $m_i = 0.2 > \sigma = 0.19$, $m_v = 0.5$, $d = 0.05$, and no noise. Survival of infected for $\omega = 0.25$ noise intensity (fourth row).

ence the growth of the susceptibles and contribute to the carrying capacity, but not vice versa.

The switch from lysogenic to lytic virus replication results in a much lower
FIGURE 15.9: Spatial coexistence of infected (two middle rows) and zooplankton (two lower rows). Extinction of susceptibles (first row) for \( m_i = 0.2 < \sigma = 0.21, m_v = 0.5, d = 0.05, \) and no noise. Survival of susceptibles for \( \omega = 0.25 \) noise intensity (second row).

mean abundance of infected, though endemcity is still stable. However, the system responds rather sensitively to parameter changes, especially to variations of virulence and transmission rate, and the infected can easily become
Epidemic spread in a stochastic environment

FIGURE 15.10: Spatial coexistence of susceptibles (two upper rows), infected (two middle rows), and zooplankton (two lower rows) for $m_i = \sigma = 0.2$, $m_v = 0.625$, $d = 0.05$. Without noise, trapping and almost extinction of infected in the center (third row). With $\omega = 0.25$ noise intensity, noise-enhanced survival, and escape of infected (fourth row). Phenomenon of dynamic stabilization of a locally unstable equilibrium (first and fifth rows).

extinct. As in the preceding Section, multiplicative noise supports the survival
FIGURE 15.11: The nonoscillating endemic state with lysogenic infection, switching at $t=100$ to an oscillating endemic state with lytic infection. The growth rate of infected $r_i$ is set from $r_i^{\text{max}} = 0.4$ to zero, the virulence $m_i$ from $m_i^{\text{min}} = 0.2$ to $m_i^{\text{max}} = 0.3$, and the transmission rate $\sigma$ from $\sigma^{\text{min}} = 0.6$ to $\sigma^{\text{max}} = 0.9$; see details in the text. Other parameter values: $r_s = 1$, $a = b = 5$, $m_v = 0.625$. Susceptibles are plotted with a solid line, infected with a dashed line, and zooplankton with dots. With permission from Hilker et al. (2006).

of the endangered species, i.e., there is always some probability to survive in a noisy environment while the deterministic setting inevitably leads to extinction.

15.6 Spatiotemporal dynamics with switches from lysogeny to lysis

Now, the switch dynamics of the plankton model (15.1a)–(15.1c) with horizontal diffusion in two-dimensional space in a constant and a fluctuating environment is considered.

Periodic boundary conditions have been chosen again for all simulations in order to avoid boundary effects. The initial conditions are as follows: The space is filled with the non-oscillating endemic state $(u^S, i^S, v^S) = (0.109, 0.224, 0.141)$; cf. Figure 15.11. Furthermore, there are two localized patches in space. They can be seen in Figure 15.12. The greyscale changes again from high population densities in black color to vanishing densities in white.

As in Section 15.4, one patch is located in the upper middle of the model area with susceptibles $u = 0.550$, four grid points further away from zooplank-
Epidemic spread in a stochastic environment

FIGURE 15.12: Initial conditions for further spatiotemporal computations. With permission from Hilker et al. (2006).

ton $v = 0.450$ in each direction. The infected are at $i^S$. In the other patch at
the right, the infected $i = 0.333$ are further away from zooplankton $v = 0.036,$
whereas the susceptibles are at $u^S$. These initial conditions are the same for
deterministic and stochastic computations.

The chosen system parameters generate oscillations in the center of the
patches. The latter act as leading centers for concentric waves that collide
and break up to spirals. Increasing noise again blurs this naturally unrealistic
patterning; see also Malchow et al. (2004).

15.6.1 Deterministic switching from lysogeny to lysis

At first, switching begins in the area with the highest initial density of
infected, i.e., in the right-hand patch. The growth rate of infected $r_i$
vanishes whereas virulence and natural mortality of infected increase to a higher
effective virulence $m_i$. The transmission rate $\sigma$ also rises as described in Sec-
tion 15.3. It is assumed that these parameter changes diffuse through space
like a Fisher wave (1937). If a subsidiary quantity $r_a$ with Fisher dynamics is
introduced,

$$
\frac{\partial r_a(\vec{r}, t)}{\partial t} = r_a(1 - r_a) + d\Delta r_a ,
$$

then

$$
r_i(\vec{r}, t) = r_i^{max}(1 - r_a) ,
$$

$$
m_i(\vec{r}, t) = m_i^{min} + (m_i^{max} - m_i^{min})r_a ,
$$

$$
\sigma(\vec{r}, t) = \sigma^{min} + (\sigma^{max} - \sigma^{min})r_a .
$$

The initial conditions are $r_a = 1$ in the right patch and zero elsewhere. For
simplicity, the diffusivity $d$ is assumed to be the same as for all the populations.
Its value of $d = 0.05$ has been chosen from Okubo’s (1971) diffusion diagrams
in order to model processes on a kilometer scale. The spatial propagation of
$r_i = 0$ is displayed in white in Figure 15.13.

The arising dynamics of susceptibles is presented in Figure 15.14. The latter
graphics has been chosen because of richer contrast, however, the patterns of
infected are similar.
FIGURE 15.13: Spatial propagation of zero replication rate of the infected. With permission from Hilker et al. (2006).


The breakup of concentric waves to a rather complex structure with spirals is nicely seen. In the long run, a pinning-like behavior of pairs of spirals is found. This effect is well-known from excitation waves in cardiac muscles; cf. the classical publications by Davidenko et al. (1992) and Pertsov et al. (1993). Here, the biological meaning remains unclear. The almost fixed pair-forming and rapidly rotating spirals approach each other extremely slowly, collide, and burst. A weak multiplicative noise accelerates this process, a fact shown in Figure 15.15. Stronger noise, i.e., higher environmental variability, suppresses the generation of pins. For the long run, the homogeneously oscillating endemic state prevails.

The system parameters have been selected in order to guarantee the survival of all three populations under deterministic conditions. The simulation for Figure 15.15 with 5% noise also yields this final endemic coexistence. However,
after the switch to cell lysis, it should be noted that there is only a certain survival probability for all three populations, the lowest for the infected.

15.6.2 Stochastic switching

The deterministic once-for-ever switching mechanism is very unrealistic. Furthermore, lytic infections might become lysogenic again (Herskowitz and Hagen, 1980; Moebus, 1996; Wilson et al., 1996; Oppenheim et al., 2005). Therefore, it is considered that only a certain fraction of viruses locally begins with the lysogenic replication and then switches. In order to model this, the subsidiary quantity \( r_a \) is redefined to obey bistable kinetics and multiplicative noise, i.e.,

\[
\frac{\partial r_a(r, t)}{\partial t} = (r_a - r_a^{\min})(r_a - r_a^{\text{crit}})(r_a^{\max} - r_a) + \omega_a r_a \cdot \xi(r, t). \tag{15.8}
\]

The noise stimulates system (15.8) to jump between its stable stationary states \( r_a^{\min} \) and \( r_a^{\max} \) (Nitzan et al., 1974; Ebeling and Schimansky-Geier, 1980; Malchow and Schimansky-Geier, 1985). It is assumed that the replication rate of the infected changes accordingly, i.e.,

\[
\begin{align*}
& \text{if } r_a > r_a^{\text{crit}} \quad \text{then } m_i = m_i^{\min}, \sigma = \sigma_i^{\min}, r_i = r_i^{\max} \text{ (lysogeny),} \\
& \text{if } r_a \leq r_a^{\text{crit}} \quad \text{then } m_i = m_i^{\max}, \sigma = \sigma_i^{\max}, r_i = 0 \text{ (lysis).} 
\end{align*}
\tag{15.9, 15.10}
\]

This noise-induced dynamics of \( r_a \) and \( r_2 \) as well as the temporal development of the spatial mean and the spatiotemporal pattern of \( r_2 \) are drawn.
FIGURE 15.16: Noisy bistable dynamics of $r_a$ and resulting local switches of $r_i$ for $r_{a\text{max}} = 2$, $r_{a\text{crit}} = 1.5$, $r_{a\text{min}} = 1$, and $\omega_a = 0.1$. The spatial mean of $r_i$ decreases from the maximum as a homogeneous initial condition to a value of approximately 0.15. The growth rate of infected $r_i$ switches from $r_{i\text{max}} = 0.4$ to zero, the virulence $m_i$ from 0.2 to 0.3, and the transmission rate $\sigma$ from 0.6 to 0.9; see details in the text. Other parameter values: $r_1 = 1$, $a = b = 5$, $m_3 = 0.625$. With permission from Hilker et al. (2006).

in Figures 15.16 and 15.17, respectively. Initially, the whole system is in the lysogenic state (15.9).
FIGURE 15.17: Spatiotemporal pattern of $r_i$. With permission from Hilker et al. (2006).

FIGURE 15.18: Deterministic dynamics of susceptibles with noisy switch. $\omega_i = \omega_j = \omega_k = 0$, $\omega_l = 0.1$. The almost stationary spiral pairs do still exist. With permission from Hilker et al. (2006).
At first, the simulation is run with noisy switches of \( r_i, \sigma \) and \( m_i \) but deterministic population dynamics. In this unrealistic setting, the pins can still be seen; cf. Figure 15.18.

If also the population dynamics is subject to noise, the result becomes more realistic. The pins are suppressed and the plankton forms a rather complex noise-induced patchy structure, cf. Figure 15.19.

\[
\omega_j = 0.1, \quad j = u, i, v, a.
\]

The “pins” can no longer be found. With permission from Hilker et al. (2006).

Noisy jumps and population dynamics generate a complex patchy spatiotemporal structure that is typical for natural plankton populations. The presented sample results have led to a final endemic state with the coexistence of susceptibles, infected and zooplankton like in the deterministic case. However, one should be aware that the survival probability of the system with all nonzero populations is smaller than 1. Thus, there are good chances that the three-component system will switch to one of its subsystems. Again, noise does not only influence the spatiotemporal coexistence of the populations but also blurs distinct artificial population structures.
Chapter 16

Slow-fast dynamics and noise-induced pattern formation in an excitable prey–predator plankton system with infected prey

In this chapter, we continue considering the lysogenic case but focus on modeling the pattern-generating impact of multiplicative noise (Spagnolo et al., 2002, 2004; Allen, 2003; Anishenko et al., 2003; Valenti et al., 2004, 2006; Sieber et al., 2007) on excitable dynamics, i.e., noise-induced effects on interacting phytoplankton and zooplankton with Holling type III grazing, in the excitable parameter interval, close to a Hopf bifurcation, in time and space. A review on processes and models of spatiotemporal ordering out of noise has been provided by Sagüés et al. (2007).

The local excitable prey–predator model with virally infected prey and lysogeny reads

\[ \frac{\epsilon}{dt} = \epsilon \frac{ds}{dt} = r_s s(1 - u) - \frac{a^2 s u}{1 + b^2 u^2} v - \frac{s i}{u} , \] (16.1)
\[ \frac{di}{dt} = r_i i(1 - u) - \frac{a^2 i u}{1 + b^2 u^2} v + \frac{s i}{u} - m_i i , \] (16.2)
\[ \frac{dv}{dt} = \frac{a^2 u^2}{1 + b^2 u^2} v - m_v v . \] (16.3)

Slow-fast prey–predator cycles in this model have been studied by Fernández et al. (2002). Such processes with longer and shorter turnover times are wellknown in ecosystem dynamics, e.g., forest-pest interactions with periodic massive outbreaks of insect pests (Ludwig et al., 1978; Rinaldi and Muratori, 1992a,b) or cyclic grazing systems with periodic collapses and recoveries of the vegetation (Noy-Meir, 1975; Rietkerk, 1998). Other sudden catastrophic regime shifts in ecosystems with long return times have been reviewed by Scheffer et al. (2001), Scheffer and Carpenter (2003), and Rietkerk et al. (2004); see also Carpenter and Turner (2000) and the whole Ecosystems issue including the work by Rinaldi and Scheffer (2000) on prey–predator food chain models.

The small parameter \( \epsilon \ll 1 \) even pushes the fast prey dynamics modeling the high sensitivity and much faster response of the phytoplankton population...
Spatiotemporal patterns in ecology and epidemiology to environmental changes like fluctuations of nutrient supply, radiation, or temperature.

![Population Densities vs Time for Different Parameters](image)

**FIGURE 16.1:** Noise-driven regimes for $\omega = 0.006$ and $0.0475$ (upper and lower figure, resp.), as well as coherence resonance for $\omega = 0.02$ (middle figure). Parameters: $r_s = r_i = 1.0$, $a = 4.0$, $b = 12.0$, $\sigma = m_i = 0.01$, $m_v = 0.0525$, $\epsilon = 10^{-3}$.

One of the remarkable effects discovered in stochastic excitable dynamics is
the generation of oscillatory behavior by noisy perturbations (Treutlein and Schulten, 1986; Ebeling et al., 1986; Sakaguchi et al., 1988; Sigeti and Horsthemke, 1989; Hillebrand, 2002; Zaks et al., 2003) and without an external periodic force. Excitable systems driven by noise have a noise-induced eigenfrequency, and, hence, they are able to show a stochastic limit cycle in phase space. These noise-sustained oscillations are often accompanied by coherence resonance (Gang et al., 1993; Longtin, 1997; Neiman et al., 1997; Pikovsky and Kurths, 1997; Pradines et al., 1999), which corresponds to the existence of an optimal noise intensity at which noise-induced oscillations are most coherent. Examples for coherence resonance have been found in neural models, laser models, models of excitable biomembranes, and even in climate models; cf. Lindner et al. (2004) for a comprehensive review and the references therein. Kuske et al. (2007) report on sustained oscillations through coherence resonance in a non-excitable SIR model. In system (16.1)–(16.3) one finds the wanted coherence resonance, i.e., noise-induced prey–predator oscillations, for medium noise intensities (Sieber et al., 2007); see Figure 16.1.

In space, one finds correspondingly interesting scenarios: For low noise intensities, the system is biologically controlled; for high noise intensities it is noise-driven. However, again for medium noise intensities, there is global synchronization of all local oscillators. This is demonstrated in Figures 16.2 and 16.3.

![Figure 16.2](image-url)

**FIGURE 16.2:** Formation of globally synchronized oscillations. Only the density distribution of susceptibles is drawn. Parameters: $r_s = r_i = 1.0, \ a = 4.0, \ b = 12.0, \ \sigma = m_i = 0.01, \ m_v = 0.0525, \ \epsilon = 10^{-3}, \ \omega = 0.015$ (upper row) and $0.04$ (lower row). Spatially uniform initial condition. Periodic boundary conditions.
FIGURE 16.3: Corresponding dynamics of the spatial mean population densities for $\omega = 0.015$ (upper figure) and $0.04$ (lower figure). Dashed line for susceptibles, solid line for infected, dotted line for zooplankton.

This is a first example of noise-induced prey–predator oscillations in an eco-epidemiological model system (Malchow and Schimansky-Geier, 2006). An even more striking noise-induced effect is the formation of stationary patterns in a small window of noise intensities between global synchronization and noise control. The process of pattern formation starts with local patches of higher population densities surviving after a global excitation due to combined effects of slow-fast prey–predator interaction, strong noise, and diffusion. The densities of the susceptibles and infected surrounding the patch do not relax back to their actual rest states, but are pushed below these due to the higher zooplankton density in the vicinity of those patches. Since the phytoplankton densities in this area are now too low to become excited again by noisy perturbations, a neighbourhood of an initial patch does not take part in the next global excitation. This leads to an interspace with low population densities. The phytoplankton densities are highest at the edges of the excited parts of the domain and once these parts drop back to the rest state, the edges also remain locked in the excited state. This phenomenon of population densities
being highest at the edge of a sharply bounded excited patch has already been investigated by Hastings et al. (1997) in order to explain field observations of outbreaks in an insect host-parasitoid system (Maron and Harrison, 1997; Brodmann et al., 1997). Two examples of this pattern formation are shown in Figure 16.4 and 16.5.

**FIGURE 16.4:** Formation of stationary spatial structures. Parameters: \( \omega = 0.06 \) (upper row) and 0.1 (lower row); all others parameters and conditions as in Figure 16.2.

**FIGURE 16.5:** Corresponding dynamics of the spatial mean population densities for \( \omega = 0.06 \) (upper figure) and 0.1 (lower figure). Lines as in Figure 16.3.
Environmental stress is obviously capable of stimulating catastrophic shifts in the dynamics of ecosystems, here, the recurrent generation of phytoplankton blooms. This is also seen in space. The spatiotemporal lattice dynamics of the presented model exhibits several interesting features at different ranges of noise intensity. This includes spatially synchronized oscillations, enhanced coherence resonance, and the formation of stationary spatial patterns.
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