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Instabilities in spatially extended predator–prey systems: Spatio-temporal patterns in the neighborhood of Turing–Hopf bifurcations

Martin Baurmann[a,*], Thilo Gross[b], Ulrike Feudel[a]

[a]Institute for Chemistry and Biology of the Marine Environment, Carl von Ossietzky University, 26111 Oldenburg, Germany
[b]Department of Chemical Engineering, Princeton University, Engineering Quadrangle, Princeton, NJ 08544, USA

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Abstract

We investigate the emergence of spatio-temporal patterns in ecological systems. In particular, we study a generalized predator–prey system on a spatial domain. On this domain diffusion is considered as the principal process of motion. We derive the conditions for Hopf and Turing instabilities without specifying the predator–prey functional responses and discuss their biological implications. Furthermore, we identify the codimension-2 Turing–Hopf bifurcation and the codimension-3 Turing–Takens–Bogdanov bifurcation. These bifurcations give rise to complex pattern formation processes in their neighborhood. Our theoretical findings are illustrated with a specific model. In simulations a large variety of different types of long-term behavior, including homogenous distributions, stationary spatial patterns and complex spatio-temporal patterns, are observed.

Keywords: Predator–prey models; Turing–Hopf bifurcation; Turing–Takens–Bogdanov bifurcation; Pattern formation

1. Introduction

Ecological systems are characterized by the interaction between species and their natural environment. An important type of interaction which effects population dynamics of all species is predation. Thus predator–prey models have been in the focus of ecological science since the early days of this discipline. It has turned out eventually that predator–prey systems can show different dynamical behaviors (steady states, oscillations, chaos) depending on the value of model parameters. In the past, investigations have revealed that spatial inhomogeneities like the inhomogeneous distribution of nutrients as well as interactions on spatial scales like migration can have an important impact on the dynamics of ecological populations (MacArthur, 1958; Malchow, 2000; Medvinsky et al., 2002; Pascual et al., 2002; Petrovskii et al., 2004; Wilson and Abrams, 2005). In particular it has been shown that spatial inhomogeneities promotes the persistence of ecological populations, play an important role in speciation and stabilize population levels.

If spatial domains are considered then the question arises how the movement of individuals in this domain should be modeled. A simple approach is to assume that the individuals move randomly. On the level of population densities this leads to a diffusion equation. In this sense a spatial predator–prey system can be considered as a reaction–diffusion system—we sometimes use the term predation–diffusion system for it. Reaction–diffusion systems have been studied in chemistry for a long time (see Nicolis and Prigogine, 1977; Nicolis and Gaspard, 1990). In particular, it has been shown that such systems are capable of self-organized pattern formation (e.g. Winfree, 1991; Lengyel and Epstein, 1992; Cross and Hohenberg, 1993; Maini et al., 1997). In this process spatial patterns arise not from inhomogeneities of initial or boundary conditions, but purely from the dynamics of the system, i.e. from the interaction of nonlinear reactions or growth processes and diffusion as already shown by

*Corresponding author. Tel.: +49 441 7982092.
E-mail address: baurmann@icbm.de (M. Baurmann).

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Turing (1952). Since then the formation of stationary spatial patterns due to Turing instabilities has been studied in physics, chemistry and biology. In the last decade the focus of research has been shifted to the study of the formation of spatio-temporal patterns. Such spatio-temporal structures have been observed in chemical experiments (Perraud et al., 1993; Rüdiger et al., 2003). Theoretical studies prove their existence for the Brusselator model (Rovinsky and Menzinger, 1992), in optical systems (Tlidi et al., 1997) and in semiconductor heterostructures (Just et al., 2001).

Pattern formation in predator–prey systems has been studied in ecology for more than thirty years, now. Starting with the pioneering work of Segel and Jackson (1972) several researches have formulated conditions for the occurrence of Turing instabilities in specific systems (Mimura and Murray, 1978, Murray, 1993, 2003, Bartumeus et al., 2001; Alonso et al., 2002). Using an approach introduced by Gross et al. (2004) we generalize their findings in order to identify the properties of predation–diffusion systems that promote or inhibit the phenomenon of pattern formation. Beyond the classical Turing mechanisms, researchers have found that complex spatio-temporal behavior can be induced by spatially heterogeneous living conditions (e.g. varying reproduction rate of the prey (see Pascual, 1993) or by certain modeling approaches (see Morozov et al., 2006 for the impact of the Allee effect and Sherratt, 2001 for that of predator invasion)). As distinguished from these approaches we focus on a special class of spatio-temporal patterns that are very likely to be found in the neighborhood of Turing–Hopf bifurcations. In this kind of bifurcation the formation of inhomogeneous stationary patterns caused by Turing instabilities "interacts" with the appearance of oscillations due to a Hopf bifurcation. This mechanism of the emergence of spatio-temporal patterns has been studied intensively during the last years in physical and chemical system (De Wit et al., 1996; Meixner et al., 1997). We show that this interplay of stationary inhomogeneous patterns with homogeneous patterns lead to spatio-temporal patterns in predation–diffusion systems.

In this paper, we study the bifurcations in spatially extended population dynamical models from a rather general point of view. We extend the concept of generalized models (Gross and Feudel, 2006) to spatially extended systems, in which the motion can be described by a diffusion term. These general models allow us to investigate bifurcations of an equilibrium state in a rather general context, namely in systems, in which the predator–prey functional response is not a priori specified. Furthermore, generalized models depend on a set of parameters that allow a biological interpretation and that can be used to determine the models' dynamics easily. In this way we find Turing-, Hopf- and transcritical bifurcations depending on the functional forms used to describe growth and predation processes in specific models. As a result we obtain bifurcation surfaces which intersect. These intersections lead to higher codimension bifurcations. Using the concept of generalized models we can show that in the neighborhood of Turing–Hopf bifurcations complex spatio-temporal dynamics appear. To illustrate this in more detail and to show the kind of structures which emerge we investigate a specific population dynamical model, namely a modified Rosenzweig–McArthur model in space.

The paper is organized as follows: In Section 2 we employ a special normalization procedure that identifies natural parameters of the predation–diffusion system. These parameters determine the dynamics of the system and we show, considering examples taken from literature, that these parameters can be interpreted biologically. With respect to these parameters we derive the mathematical conditions for the occurrence of pattern formation processes. Based on these conditions we locate the Turing bifurcation within the generalized parameter space. In Section 3 we use findings of Gross and Feudel (2004) to compute the transcritical and Hopf bifurcations, as well. We show that the Turing instability can coincide with these bifurcations constituting a codimension-2 bifurcation. Since we are, in particular, interested in the model dynamics in the neighborhood of the Turing–Hopf bifurcation, we perform a series of simulations (Section 4) to illustrate the emergence of spatio-temporal patterns. For this purpose it is necessary to choose a specific model for our studies. We employ a Rosenzweig–McArthur model with a quadratic mortality of the predator. By this modification we take into account that predator individuals may compete for resources and that higher order predation has an impact on the predator's mortality. Depending on the parameters—we keep them in the general context—this model shows homogeneous and inhomogeneous equilibria as well as spatio-temporal dynamics which turns out to be chaotic in space and time.

2. A generalized predation–diffusion system

In this section we analyse a generalized predator–prey system on a two-dimensional domain. Diffusion is considered as the principal mechanism of motion. Such a system can be described by

$$\frac{\partial U}{\partial \tau} = S(U) - F(U,V) + D_u \nabla^2 U,$$

$$\frac{\partial V}{\partial \tau} = \eta F(U,V) - M(V) + D_v \nabla^2 V, \tag{1}$$

where τ denotes time. The variable U is the biomass density of the prey and V is the biomass density of the predator. The functions S and M determine the species intrinsic population dynamics (primary production and mortality, respectively) and F is the predation rate. The scalar factor η denotes the fraction of prey's biomass that can be converted into predator biomass. The diffusion coefficients are denoted by D_u and D_v, respectively.

The predator–prey dynamics occurs locally in each point of space. Thus we call this part of the dynamics the *local*

model. In a local model all terms of Eq. (1) describing the population dynamics in a point of the domain are taken into account whereas the terms of motion (i.e. the diffusion) are neglected. In the following we assume that the local model has an equilibrium with positive values for prey and predator at (U_s, V_s). If for Eq. (1) appropriate boundary conditions (e.g. periodic ones) are considered the spatially constant functions $U(\vec{z}) = U_s$ and $V(\vec{z}) = V_s$ constitute an equilibrium for this system. Using this steady state we can introduce the normalized variables

$$u(\vec{z},\tau) = \frac{U(\vec{z},\tau)}{U_s}, \quad v(\vec{z},\tau) = \frac{V(\vec{z},\tau)}{V_s} \tag{2}$$

and the normalized functions

$$f(u,v) = \frac{F(U_s u, V_s v)}{F(U_s, V_s)}, \quad s(u) = \frac{S(U_s u)}{S(U_s)}, \quad m(v) = \frac{M(V_s v)}{M(V_s)}. \tag{3}$$

We substitute these formulae into Eq. (1) and make use of $S(U_s) = F(U_s, V_s)$ and $\eta F(U_s, V_s) = M(V_s)$, conditions which hold in the spatially homogeneous stationary state. This yields

$$\frac{\partial u}{\partial \tau} = \alpha_u(s(u) - f(u,v)) + D_u \nabla^2 u,$$

$$\frac{\partial v}{\partial \tau} = \alpha_v(f(u,v) - m(v)) + D_v \nabla^2 v, \tag{4}$$

where

$$\alpha_u = \frac{S(U_s)}{U_s} = \frac{F(U_s, V_s)}{U_s}, \quad \alpha_v = \frac{M(V_s)}{V_s} = \eta \frac{F(U_s, V_s)}{V_s}. \tag{5}$$

From the way in which α_u and α_v appear in the equations of motion it can be seen that they correspond to characteristic time-scales of the system. Indeed, closer inspection reveals that α_u denotes the per-capita production rate of the prey in the steady state. This rate has to be equal to the per-capita death rate in the steady state. We can therefore think of α_u as the inverse of the life expectancy of individuals of the prey population in the steady state. In the same way α_v indicates the inverse of the life expectancy of the predators.

By means of time and space normalization $((\tau, \vec{z}) \to (t, \vec{x}))$ we obtain

$$\frac{\partial u}{\partial t} = \rho_r(s(u) - f(u,v)) + \rho_d \nabla^2 u,$$

$$\frac{\partial v}{\partial t} = f(u,v) - m(v) + \nabla^2 v, \tag{6}$$

where $t = \alpha_v \tau$, $\vec{x} = \sqrt{\alpha_v / D_v}\vec{z}$, $\rho_r = \alpha_u/\alpha_v = V_s/(\eta U_s)$ and $\rho_d = D_u/D_v$.

In order to study the stability of homogeneous equilibrium $u_s(x,y) = 1, v_s(x,y) = 1$ with respect to perturbations $(\sim \exp(\mathrm{i}\vec{\kappa}\vec{x}))$ with wave numbers κ_x and κ_y, we consider a linearized form of Eq. (6). The stability of the homogeneous equilibrium is then determined by the eigenvalues of the matrix $\mathbf{J}_d(\kappa_x, \kappa_y)$ (see e.g. Murray, 2003, p 82 for detailed description of the approach). In our predation–diffusion system we obtain

$$\mathbf{J}_d(\kappa_x, \kappa_y) = \begin{bmatrix} \rho_r(\phi - \gamma) - \rho_d(\kappa_x^2 + \kappa_y^2) & -\rho_r \psi \\ \gamma & \psi - p - (\kappa_x^2 + \kappa_y^2) \end{bmatrix}, \tag{7}$$

where

$$\phi := \left.\frac{\mathrm{d}s(u)}{\mathrm{d}u}\right|_{u=1}, \quad \gamma := \left.\frac{\partial f(u,v)}{\partial u}\right|_{u=1,v=1},$$

$$\psi := \left.\frac{\partial f(u,v)}{\partial v}\right|_{u=1,v=1}, \quad p := \left.\frac{\mathrm{d}m(v)}{\mathrm{d}v}\right|_{v=1}. \tag{8}$$

Like the time-scales, the parameters defined above can be interpreted in ecological terms. For this purpose we make use of a specific property of the normalization: If any given function in the model (say, $s(u)$) is linear then the corresponding parameter (in this case ϕ) is one. If the function rises faster than linearly then the parameter is larger than one. For example a quadratic function results in a parameter value of two. If, by contrast, the function rises less than linearly, the corresponding parameter is smaller than one. An extreme case would be a constant function, which corresponds to a parameter value of zero (for further discussion of these relationships see Gross, 2004; Gross and Feudel, 2006). In summary, we can say that the parameters introduced above describe the linearity or nonlinearity of the functions in the system. In the following we use this insight to discuss the parameters in more detail.

The parameter ϕ quantifies to which extend the growth of the prey population depends on its size. Following Bazykin (1998, Chapter 2) we distinguish between models of free populations and those of populations with limited external resources. In the first ones the growth of a population is unlimited. Hence we obtain positive values for ϕ. Free populations can be modeled by an exponential law $\partial U/\partial\tau \approx U$ or an hyperbolic approach $\partial U/\partial\tau \approx U^2$, for instance. In the first case we obtain $\phi = 1$, the second one yields $\phi = 2$. Considering populations that depend on external resources, we generally find, that ϕ is negative at least for high population densities. A good example for this kind of a model is given by the Verhulst–Pearle logistic equation, which is often used to model the growth of a prey population (e.g. Pascual, 1993; Sherratt, 2001; Alonso et al., 2002). Following the Verhulst–Pearle approach we find $\phi = \partial s/\partial u = 1 - U_s/(U_{\max} - U_s)$, where $U_{\max}$ is the carrying capacity. So, if U_s is small compared with $U_{\max}$, we obtain a parameter ϕ that is slightly smaller than 1. For larger values of U_s the values of ϕ decrease. In particular we obtain $\phi = 0$ at $U_s = U_{\max}/2$ and $\phi \to -\infty$ if $U_s \to U_{\max}$. We call the parameter ϕ which measures the intraspecific competition of prey individuals for nutrients sensitivity with respect to nutrients.

In analogy to ϕ the parameter γ describes the sensitivity of the predation to prey supply. If prey is scarce the predation rate generally increases linearly or quadratically with the prey density. This corresponds to $\gamma = 1$ or $\gamma = 2$, respectively. However, if prey is abundant the predation rate approaches a constant limiting value since predator saturation occurs. This corresponds to $\gamma = 0$. If prey is neither very abundant nor very scarce intermediate values of γ are found.

The parameter ψ describes the dependence of the predation rate on the density of the predator. In most specific models the predation rate increases linearly with predator density. This corresponds to $\psi = 1$. However, social interactions, interference or intraspecific competition in the predator population can decrease ψ. For instance, if we consider the system studied by Bartumeus et al. (2001), which has the specific predator equation

$$\frac{\partial V}{\partial t} = \eta \frac{bUV}{kV + U} - \mu V$$

we obtain $0 < \psi < 1$. Furthermore, we would like to draw the reader's attention to the fact, that for systems employing the forms of functional response and mortality used by Bartumeus et al. (2001) we obtain the relationship $\psi = 1 - \gamma$, regardless of what function for primary production is applied.

Finally, the parameter p is a generalized version of the well-known exponent of closure (Edwards and Bees, 2001). In many models, like for instance the classical Rosenzweig–MacArthur model, density independent per-capita mortality is assumed. In this case the total mortality rate is proportional to the population density. Consequently, the exponent of closure is one. However, if the abundance of predators is high, density dependent effects start to play an important role. For instance the effects of stress, diseases, overcrowding and intraspecific competition cause quadratic mortality rates which correspond to $p = 2$. In several natural systems this density-dependent mortality rates are more important than density independent causes of mortality. An exponent of closure of two is therefore used in a number of specific models (Edwards and Yool, 2000). If density dependent as well as density independent effects are taken into account p has a fractional value between one and two. A mortality term of this form has been used by Segel and Jackson (1972). Since the authors use a linear Lotka–Volterra term for predation (yielding $\gamma = \psi = 1$), they conclude that pattern formation is only possible if the quadratic mortality term is not zero (cf. the Turing condition given by Eq. (10)).

To compute the influence of the generalized parameters on the dynamics of the model, let us consider the Jacobian matrix given by Eq. (7). In this formula we substitute the sum $\kappa_x^2 + \kappa_y^2$ by κ^2. Thus we obtain

$$\mathbf{J}_d(\kappa) = \begin{bmatrix} \rho_r(\phi - \gamma) - \rho_d\kappa^2 & -\rho_r\psi \\ \gamma & \psi - p - \kappa^2 \end{bmatrix}. \quad (9)$$

Note that $\mathbf{J} := \mathbf{J}_d(0)$ is the Jacobian matrix for the local model which we obtain by neglecting the diffusion-terms in Eq. (6). This means that the model's sensitivity with respect to homogeneous perturbations is identical to the stability properties of the local model. Consequently, a homogeneous equilibrium cannot be destabilized by homogeneous perturbations, if the steady state corresponds to an attractor in the local model. Nevertheless, (Turing, 1952) showed that diffusion can induce the loss of stability with respect to perturbations of certain wave numbers, yielding the condition $\det(\mathbf{J}_d(\kappa^2)) < 0$. By straight forward analysis we find that $\det(\mathbf{J}_d(\kappa^2))$ is a quadratic polynomial with respect to κ^2. Its extremum is a minimum at some κ_e^2. Presuming the stability of the equilibrium in the local model (in particular $\det(\mathbf{J}) > 0$), we find two conditions for Turing-instabilities to occur (cf. Baurmann et al., 2004). The first one is $\kappa_e^2 > 0$. If this condition is violated, it follows from $\det(\mathbf{J}_d(0)) = \det(\mathbf{J}) > 0$ that $\det(\mathbf{J}_d(\kappa^2)) > 0$ for all $\kappa^2 > 0$. Thus $\kappa_e^2 > 0$ is a necessary condition for Turing instabilities to occur. The sufficient condition is $\det(\mathbf{J}_d(\kappa_e^2)) \leqslant 0$.

Let us now formulate the Turing conditions in terms of the generalized parameters $p, \gamma, \phi, \psi, \rho_r$ and ρ_d. In the predation–diffusion model we obtain as necessary condition

$$\frac{\rho_r}{\rho_d}(\phi - \gamma) > p - \psi. \quad (10)$$

In general, we have $p > \psi$, so that Eq. (10) implies $\phi > \gamma$. It follows, that the first diagonal entry of $\mathbf{J}$ is positive, the second is negative. Thus, we consider the prey species as activator while the predator plays the role of an inhibitor. Furthermore, since $\mathrm{tr}(\mathbf{J}) < 0$ we see that $\rho_r > \rho_d$.

The second and sufficient Turing condition for destabilization to occur is

$$\frac{\rho_r}{\rho_d} > \frac{1}{(\phi - \gamma)^2}\left(\sqrt{\phi\psi - \phi p + \gamma p} + \sqrt{\gamma\psi}\right)^2. \quad (11)$$

Since $\gamma\psi \geqslant 0$ and $\phi\psi - \phi p + \gamma p = \det(\mathbf{J})/\rho_r > 0$ it is guaranteed that all square roots in the formula are real.

Considering these Turing conditions, we see that in the model defined in Eq. (6) destabilization due to diffusion is more likely if the values of the parameters γ (the predator sensitivity) and ρ_d (the ratio of diffusion coefficients) are low and that of the nutrient sensitivity ϕ and the relative time-scale ρ_r are high. A small exponent of closure p may guarantee the validity of Eq. (10) but can lead to a violation of Eq. (11). On the other hand the predator interference ψ should be small to fulfill Eq. (11) and has to take large values to promote the validity of Eq. (10). However, as we have shown above, the crucial condition for the loss of stability is Eq. (11), so that we can identify the following factors as promoters of diffusion-induced instabilities: (i) high supply of nutrient, no or weak competition of prey for nutrient; (ii) high abundance of prey; (iii) strong intraspecific competition of predator

individuals; (iv) high influence of the predators population density on the mortality of predators (quadratic mortality term); (v) fast diffusive motion of the predator, slow one of the prey; (vi) long time-scales for predators, short ones for prey. The points (v) and (vi) describe typical features that hold for many predator–prey systems found in nature. By contrast, (i)–(iv) address special features of a group of systems that can be found in nature under certain conditions, for instance in systems where nutrients are plenty. Hence, anthropogenic eutrophication that is observed in a large number of ecological systems (Verhoeven et al., 2006) can destabilize spatially homogeneous equilibria in predator–prey systems.

3. Turing instabilities and their relation to other bifurcations

So far we have analysed the Turing bifurcation. That means we have studied cases, in which diffusion leads to a destabilization of a homogeneous equilibrium that is attracting in a local predator–prey system. The Turing instability results in the emergence of stationary spatially inhomogeneous patterns of predators and prey species. However, the region of *local stability* is bounded by two bifurcations: the Hopf bifurcation in which an attracting equilibrium loses its stability and the transcritical bifurcation where two branches of equilibria meet and exchange their stability properties.

In the Hopf bifurcation the eigenvalues ($\lambda_{1,2}$) of the local system are a pair of conjugated imaginary values. So we can detect it by the following two conditions

$$\lambda_1+\lambda_2=\mathrm{tr}(\mathbf{J})=0,\quad \lambda_1\lambda_2=\det(\mathbf{J})>0. \tag{12}$$

For the generalized predator–prey system we find

$$\rho_r\phi-\rho_r\gamma+\psi-p=0,\quad \phi\psi-\phi p+\gamma p>0. \tag{13}$$

In a transcritical bifurcation one real eigenvalue of the local model vanishes. Hence we find this kind of bifurcation by solving

$$\lambda_1\lambda_2=\det(\mathbf{J})=0. \tag{14}$$

In the specific context this condition reads

$$\phi\psi-\phi p+\gamma p=0. \tag{15}$$

We rewrite all bifurcation conditions in terms of the predator sensitivity to prey γ:

$$\begin{aligned}
\text{transcritical}: &\quad \gamma_{SN}=\phi-\frac{\phi\psi}{p},\\
\text{Hopf}: &\quad \gamma_H=\phi+\frac{\psi-p}{\rho_r},\\
&\quad \gamma_H>\gamma_{SN},\\
\text{Turing}: &\quad \gamma_T=\frac{\rho_d}{\rho_r}\left(\sqrt{r\psi}-\sqrt{p+\frac{\rho_r}{\rho_d}\phi}\right)^2,\\
&\quad \gamma_H<\gamma_T<\phi+\rho_d/\rho_r(\psi-p).
\end{aligned} \tag{16}$$

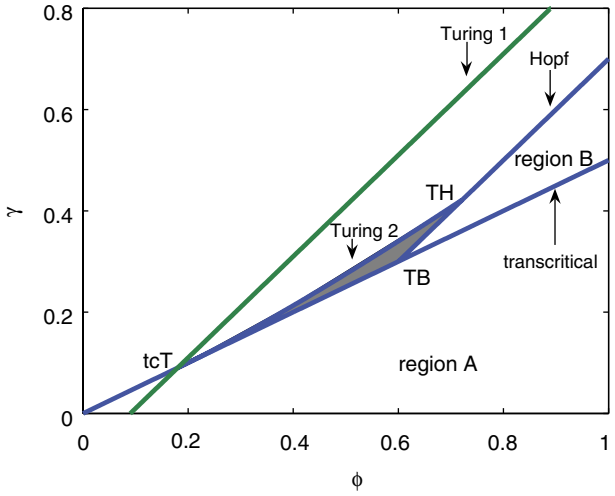

Fig. 1. Bifurcation diagram. We use $\rho_r=\frac{10}{3},\psi=1,p=2,\rho_d=0.3$ and vary ϕ. In the figure bifurcations are represented by solid lines. The Turing 1 curve is an auxiliary line, indicating the first Turing condition Eq. (10) formulated as $\kappa_c^2=0$. The figure shows the Turing space as the grey triangular area bounded by the Turing bifurcation, the Hopf bifurcation and the transcritical bifurcation. The whole parameter space above transcritical and Hopf bifurcation with exception of the Turing space corresponds to unconditionally stable equilibria.

According to our assumption $\psi<p$ the interval we refer to in the second Turing condition is non-empty if and only if $\rho_d<1$. This means that the diffusion of the predator diffuses faster than the prey.

Let us discuss the bifurcations represented by these formulas in the parameter space spanned by the predator sensitivity to prey γ and the nutrient sensitivity ϕ (Fig. 1). Note that each point in this parameter space represents a whole class of systems. The upper part of the displayed parameter space corresponds to systems with homogeneous equilibria, which are unconditionally stable. The qualitative behavior of such equilibria changes, if this region is left via a bifurcation (transcritical, Turing 2 or Hopf). If an equilibrium is represented by a point in the lower half of the parameter space shown in Fig. 1, it can be destabilized by a homogeneous perturbation (region A) and the system converges to another state depending on the specific choice of the predator–prey functional response. The same holds for equilibria, that can be found between the Hopf bifurcation and the transcritical bifurcation (region B). In this region we would expect homogeneous oscillations. As we will show later the dynamics is more complicated. The equilibria that can be found in the grey triangular area are stable with respect to homogeneous perturbations but loose their stability with respect to perturbations of specific wave numbers κ. In this region stationary inhomogeneous patterns can be observed.

The figure also shows three points, in which the bifurcation curves meet. These points correspond to bifurcations of codimension-2 and have the following

coordinates:

$$
\begin{aligned}
&\text{Takens}-\text{Bogdanov}(TB): && \lambda_1(0)=\lambda_2(0)=0,\\
&&& \phi_{TB}=\frac{p(p-\psi)}{\psi\rho_r} \quad \text{and} \quad \gamma_{TB}=(p-\psi)^2/(\rho_r\psi),\\
&\text{transcritical Turing}(tcT): && \kappa_e^2=0 \quad \text{and} \quad \lambda_1(0)=0,\\
&&& \phi_{tcT}=\frac{\rho_d p}{\rho_r\psi}(p-\psi) \quad \text{and} \quad \gamma_{tcT}=\frac{\rho_d}{\rho_r}(p-\psi)^2/\psi,\\
&\text{Turing}-\text{Hopf}(TH): && \lambda_{1,2}(0)=\pm ia\\
&&& \text{and } \lambda_1(\kappa_e^2)=0 \quad \text{for exactly one } \kappa_e^2\geqslant 0,\\
&&& \phi_{TH}=\frac{p-\psi}{\rho_r\psi}\left(\frac{(p-\psi)(\rho_d-1)^2}{4\rho_d}+p\right),\\
&&& \gamma_{TH}=\frac{(p-\psi)^2}{\rho_r\psi}\left(\frac{(\rho_d-1)^2}{4\rho_d}+1\right).
\end{aligned}
$$

For $\rho_d=1$ (i.e. $D_u=D_v$) we obtain a codimension-3 bifurcation at (ϕ_{TB},γ_{TB}). For an equilibrium at this point we can compute the eigenvalues and find $\lambda_1(\kappa^2)=\lambda_2(\kappa^2)=-\kappa^2$.

If we consider bifurcation diagrams like that in Fig. 1 and decrease the difference between the diffusion coefficients so that their ratio ρ_d approaches 1 then the Turing space contracts to the codimension-3 bifurcation point and vanishes there at $\rho_d=1$.

The obtained bifurcations of higher codimension can be relevant for ecological systems. Even though we analyse only bifurcations of equilibria we can use the knowledge about the occurrence of higher codimension bifurcations to draw conclusions about the possibility of complex dynamics in the systems under consideration. So the Takens–Bogdanov bifurcation is the starting point for a line of homoclinic bifurcations. A homoclinic bifurcation indicates the collision of a limit cycle with a saddle that causes the limit cycle to disappear.

The region around a Turing–Hopf bifurcation is of most interest for our study of the formation of complex spatio-temporal structures. In the neighborhood of this bifurcation we have to expect the appearance of spatio-temporal chaos in predator–prey systems which we analyse in the next section. In particular chaotic behavior can be observed in the neighborhood of this bifurcation.

4. Spatial patterns resulting from the bifurcations

In the previous section we have described how Turing instabilities can cause a small perturbation to diverge from a homogeneous equilibrium, that corresponds to an attracting steady state in the local model. However, we are mostly interested in the mechanisms which lead to complex spatio-temporal behavior. This situation occurs, if we focus on equilibria that are not attracting in the local model like the ones beyond the Hopf bifurcation—a case that is not addressed by the classical Turing theory. We find interesting dynamics for instance in the neighborhood of repelling foci. An analysis of this case is not possible on the basis of a linearization around the homogeneous solution. As well, normal form theory cannot be applied easily to our model. Thus, we study the model's behavior by performing simulations for the parameter sets of interest. For this purpose it is necessary to specify a certain model for the population dynamics. We employ the well-known Rosenzweig–McArthur model with a quadratic mortality term for the population dynamics

$$
\begin{aligned}
\frac{\partial U}{\partial t}&=rU\left(1-\frac{U}{k}\right)-q\frac{UV}{W+U}+D_u\nabla^2U,\\
\frac{\partial V}{\partial t}&=\eta q\frac{UV}{W+U}-MV^2+D_v\nabla^2V.
\end{aligned}
\tag{17}
$$

Generally, system equation (17) has one or three equilibria with positive (U_s,V_s). The normalization procedure described in Section 2 yields

$$
\begin{aligned}
\frac{\partial u}{\partial t}&=\rho_r\left(\frac{k-U_su}{k-U_s}u-\frac{W+U_s}{W+U_su}uv\right)+\rho_d\nabla^2u,\\
\frac{\partial v}{\partial t}&=\frac{W+U_s}{W+U_su}uv-v^2+\nabla^2v.
\end{aligned}
\tag{18}
$$

The corresponding parameters are

$$
\phi=\frac{k-2U_s}{k-U_s},\quad \gamma=\frac{W}{W+U_s},\quad \psi=1,\quad p=2 \tag{19}
$$

and

$$
\rho_r=\frac{V_s}{\eta U_s}=\frac{q}{W+U_s}. \tag{20}
$$

This way one can interpret the modified Rosenzweig–McArthur model as a specific example of the class of models considered in the generalized system. In the

following we can therefore plot the results of our numerical investigations as a function of the generalized parameters ϕ, γ, ρ_d and ρ_r. The other parameters ψ and p are constant in the studied model. If an additional linear mortality term is considered (cf. Segel and Jackson, 1972), p can take variable values between 1 and 2. We want to emphasize at this point, that for the dynamical behavior of the model the exact value of p is not essential, as long as it is larger than 1.

We start by computing the corresponding equations for the bifurcations. Substituting $\psi = 1$ and $p = 2$ we obtain

$$\begin{aligned} \text{transcritical}: \quad & \gamma_{SN} = \frac{\phi}{2}, \\ \text{Hopf}: \quad & \gamma_H = \phi - \frac{1}{\rho_r}, \\ & \gamma_H > \gamma_{SN}, \\ \text{Turing}: \quad & \gamma_T = \frac{\rho_d}{\rho_r}\left(1 - \sqrt{2 + \frac{\rho_r}{\rho_d}\phi}\right)^2, \\ & \gamma_T \in \left(\gamma_H, \phi - \frac{\rho_d}{\rho_r}\right). \end{aligned} \tag{21}$$

Thus the Takens–Bogdanov point has the coordinates $(\rho_r = 2/\phi, \gamma = \phi/2)$ and if we additionally have $\rho_d = 1$, the Takens–Bogdanov bifurcation collides with the Turing bifurcation and we obtain a codimension-3 bifurcation.

Fig. 2 shows the bifurcation lines in the lower diagram. In order to obtain more detailed informations about the model's dynamics, we analyse a series of simulations. For that purpose we employ the model (17) on a two-dimensional domain with periodic boundary conditions. As initial condition we use a homogeneous distribution $u(x, y) = 1, v(x, y) = 1$ and add a small perturbation. We run the simulations until they reach a stationary state or until they show a behavior that does not seem to change its characteristics anymore. To avoid numerical instabilities and artifacts we use the `dopri5` algorithm, an explicit Runge–Kutta method of order 5, which we have taken from Hairer and Wanner (2002). In the simulations different types of dynamics are observed and we have found that the distributions of predator and prey are always of the same type. Consequently, we can restrict our analysis of pattern formation to one distribution (in the panels 1–4 of Fig. 2 we show the distribution of prey, for instance). For the purpose of classification we distinguish the types of dynamics as follows:

Homogeneous distributions: The simulation may converge to a homogeneous profile on a level representing one of the model's local stable equilibria.

Stationary patterns: The system approaches a stationary, inhomogeneous pattern. Depending on the particular structure we speak of *hot spots* (isolated zones with high population densities of prey), *stripes* (interlaced stripes of high and low population densities) or *cold spots* (isolated zones with low population densities of prey).

Spatio-temporal patterns: The model does not always converge to a stationary distribution. We have found regular structures with small defects in their patterns that do not change their locations (see panel 3 in Fig.2). The zones in which defects occur appear to oscillate. These oscillation seem to continue in the stationary structures but they are damped out with increasing distance to the defect. In other cases all regular structures become extinct and large temporal changes are found always everywhere on the domain at almost every time step. In these cases we consider the dynamics of the model as chaotic in space and time. However, the existence of chaos has not been proved rigorously.

The bifurcation diagram in Fig. 2 shows that homogeneous distributions with $u = 1$ and $v = 1$ only occur for high values of the predator sensitivity to prey γ and low values of the nutrient sensitivity ϕ. This numerical result corresponds perfectly to our theoretical findings: above the Hopf and Turing bifurcation (and only there) all simulations converge to a stable homogeneous (1, 1)-distribution. Crossing the Turing bifurcation the homogeneous profile becomes unstable and we find that cold spot structures evolve, instead. An increase of ϕ and γ causes a change in the structure via stripes towards hot spots. Along the Hopf bifurcation (on the repelling focus side) we find that no homogeneous oscillations occur—as one could have expected—but, chaotic dynamics is more frequently encountered. Some simulations with parameter values close to those leading to cold spots show a different spatiotemporal behavior: The squares in light green below the transcritical bifurcation indicate that the corresponding simulations show the coexistence of zones with different patterns. In particular, we obtain a cold spot pattern that is disrupted by homogeneous zones (see Fig. 2, panel 3). While the cold spot regions seem to be stationary we obtain a wave like dynamics on the homogeneous zones (from their centers to their margins). For high values of ϕ and low values of γ we observe convergence to homogeneous distributions that have prey-densities with a value different from 1. Consequently, we distinguish these distributions from the homogeneous ones, located on the other side of the bifurcation lines. To get a deeper insight into the model's behavior we select different values of parameter ρ_d, producing for each value a diagram similar to the one shown in Fig. 2. An increase of ρ_d does not change the transcritical and Hopf bifurcations, but causes the Turing bifurcation line to shift towards higher ϕ and to loose steepness. This leads to successive contraction of the Turing space and its extinction for $\rho_d > 1$. This phenomenon agrees with the Turing theory. The choice $\rho_d = 1$ means that activator and inhibitor diffuse equally fast, so that no Turing instability can occur. In the case that $\rho_d > 1$, the prey (activator) moves faster than the predator which violates the instability condition. According to these changes the area in parameter space corresponding to simulations converging to the homogeneous (1,1)-profiles becomes larger. Similarly, chaotic behavior becomes more likely when ρ_d takes higher values. Our simulations show that chaotic dynamics replace the convergence to

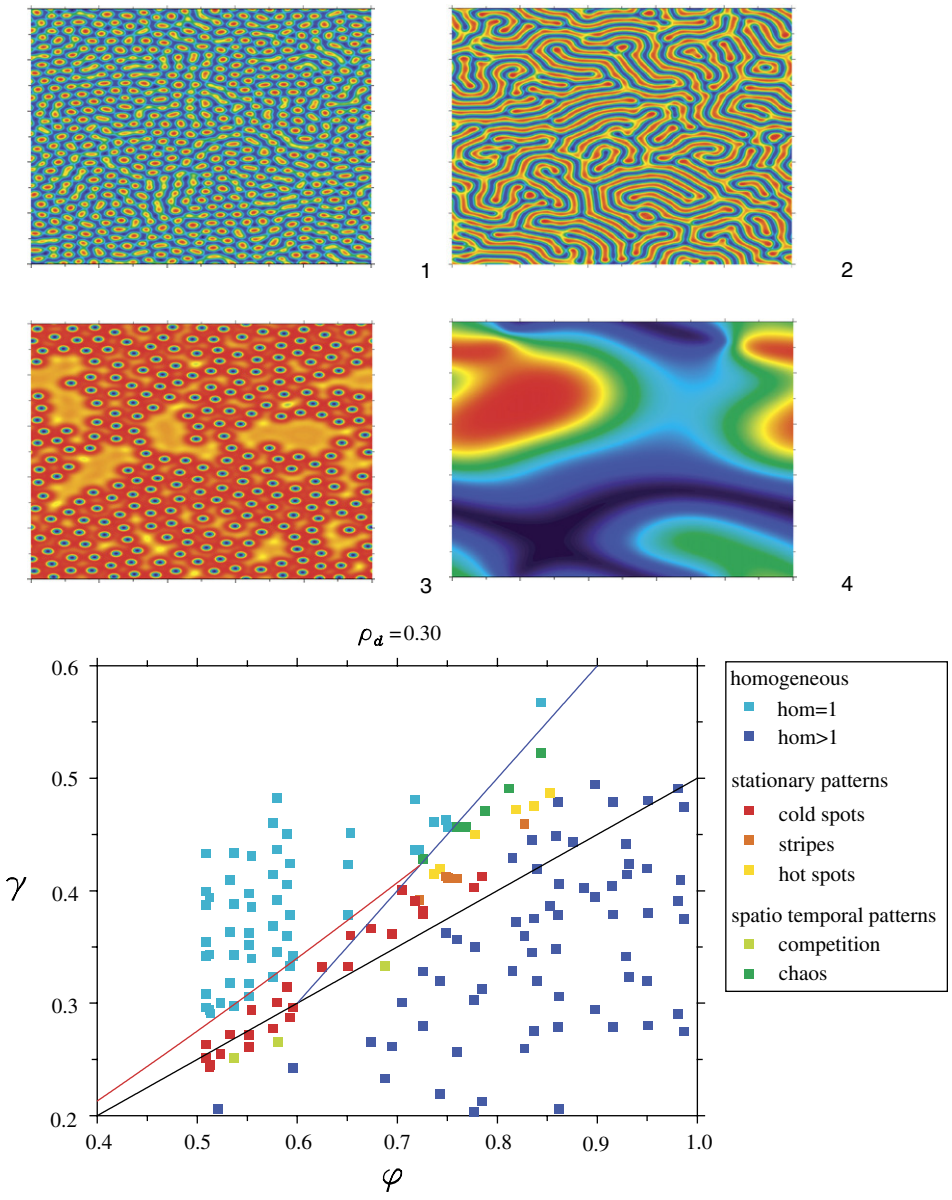

Fig. 2. Bifurcation diagram for the modified Rosenzweig–McArthur model for $\rho_d = 0.30$ and $\rho_r = 3.333$. The lower figure shows the bifurcations of the model (transcritical: black; Hopf: blue; Turing: red). The symbols represent the results of simulations: blue squares correspond to homogeneous equilibria either with the values (1, 1) (light blue) or with other positive values (dark blue). The red and orange squares symbolize stationary inhomogeneous structures like hot spots (orange, panel 1), stripes (light red, panel 2) or cold spots (dark red). Spatio-temporal patterns have been observed for simulations represented by green squares. We distinguish between persistent competition of stationary structures (generally cold spots) with spatio-temporal dynamics (light green, panel 3) and a chaotic behavior on the entire domain (dark green, panel 4).

stationary patterns. At a value of $\rho_d = 0.55$ we find that this process has been completed: all red and orange symbols are replaced by dark green squares. Competing dynamics (cf. Fig. 2, panel 3) vanish together with the stationary patterns, so that for high values of ρ_d we find only chaotic behavior and homogeneous equilibria (of both types). In our series of simulations the area of homogeneous distributions on a prey level larger than 1 appears to be a more or less invariant region in the lower right corner of the diagram that covers (for each ρ_d) approximately 40% of the studied parameter space.

5. Discussion

We study the dynamics of generalized predator–prey models with spatial interactions. The formulation and subsequent normalization of the generalized model allows us to perform a qualitative analysis of a whole class of predator–prey models without specifying the predator–prey functional response. Within this new framework we formulate the conditions for transcritical, Hopf and Turing bifurcations. In this paper mainly the Turing bifurcation is discussed. It indicates the destabilization of

the homogeneous distribution of predators and prey and the emergence of diffusion-induced pattern formation phenomena. We show that a high supply of nutrients, a high abundance of prey, a strong competition between predator individuals for prey and a high influence of intraspecific stress and/or diseases on the mortality of predators is advantageous for the occurrence of Turing instabilities. Fast population dynamics and slow diffusive motion of the prey (with respect to the corresponding rates affecting the predator) have the same effect.

As a result of the Turing bifurcation stationary, spatially inhomogeneous distributions of prey and predator arise. Since the generalized model exhibits also a loss of stability due to transcritical or Hopf bifurcations, bifurcations of higher codimension like Takens–Bogdanov, Turing–Hopf and transcritical Turing appear in the space of generalized parameters. The behavior of the predator–prey system in the neighborhood of these bifurcations is of particular interest. The existence of a Takens–Bogdanov bifurcation guarantees that predator–prey systems show homoclinic bifurcations, as well. In a homoclinic bifurcation a limit cycle collides with a saddle and disappears. This phenomenon can lead to a high excitability of the system. This means that in predator–prey systems with certain functional responses small perturbations can induce large population outbreaks and crashes.

On the other hand in the neighborhood of a Turing–Hopf bifurcation there are parameter regions in which the dynamics shows spatio-temporal behavior that is influenced by temporal oscillations as well as by Turing instabilities. To study this dynamics and to determine the parameter regions in which they occur, we cannot use analytical methods or normal forms. Thus we had to specify an appropriate model to perform simulations. The aim is to check whether a spatially homogeneous equilibrium is stable, and for the case that it is not, to which solution the model converges. For this purpose we have studied a Rosenzweig–McArthur model with quadratic mortality. We performed a series of simulations on a two-dimensional spatial domain varying three of the generalized parameters (ϕ, γ and ρ_r).

These simulations reveal that a large variety of different spatio-temporal dynamics can be found in this example of a rather simple predator–prey model with diffusion-like spatial interactions. Since our approach using generalized models shows that Turing–Hopf bifurcations leading to spatio-temporal patterns in their neighborhood are generic in a large class of predator–prey systems we can expect such dynamics to occur in many predation-diffusion models. Thus we can conclude that the "interaction" of Turing and Hopf instabilities can be considered as one important mechanism for the appearance of complex spatio-temporal dynamics in population dynamical models. Further studies are necessary to analyse the behavior of more complex predator–prey models regarding both, the number of their species as well as the nature of considered processes of motion.

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