Delay-induced patterns in a predator–prey model on complex networks with diffusion

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Keywords: reaction–diffusion system, time delay, predator–prey, wave patterns, complex networks

Abstract

Reaction–diffusion (RD) systems with time delays have been commonly used in modeling biological systems and can significantly change the dynamics of these systems. For predator–prey model with modified Leslie–Gower and Holling-type III schemes governed by RD equations, instability induced by time delay can generate spiral waves. Considering that populations are usually organized as networks instead of being continuously distributed in space, it is essential to study the predator–prey model on complex networks. In this paper, we investigate instability induced by time delay for the corresponding network organized system and explore pattern formations on several different networks including deterministic networks and random networks. We firstly obtain instability condition via linear stability analysis and then the condition is applied to study pattern formations for the model in question. The simulation results show that wave patterns can be generated on different networks. However, wave patterns on random networks differ significantly from patterns on deterministic networks. Finally, we discuss the influences of network topology on wave patterns from the aspects of amplitude and period, and reveal the ecology significance implied by these results.

1. Introduction

Reaction–diffusion (RD) system with time delay is used to describe evolution system which is affected by the present as well as the past state. Its characteristic is that the influences of history on the present situation of system are fully taken into account. As a powerful tool for describing spatiotemporal dynamics, the delay RD systems have been widely applied in many fields such as ecology [1], epidemiology [2], chemistry [3], physics [4], evolutionary games [5], and so on. Taken ecology as an example, factors that introduce time delay may include age structure of the population (influencing the birth and death rates), maturation periods, incubation periods, food storage or ingestion delays, resource regeneration times [6].

In continuous space, a RD system often leads to nonlinear RD equations—a class of typical partial differential equations (PDEs)—with reaction terms corresponding to the local dynamics and diffusion terms describing the motion of matter in space. Models governed by RD equations can show rich dynamic behaviors involving traveling waves, self-organized spatial patterns or chaos. Specifically, the introduction of time delay causes these models to exhibit much more complex dynamical behaviors than the counterpart without time delay. Therefore, RD models with time delays have received wide attention over the past years [7–11]. For example, the authors in [12, 13] derived the critical threshold for the spatiotemporal Hopf bifurcation by considering the delay as bifurcation parameter and further analyzed the influence of delay on pattern formations. Ghosh et al [3, 14] investigated RD systems with small time delays in which the authors reported that
small delays may result in a Turing instability of such systems so that spatial patterns were formed. The inadequacy of the method in [3, 14] is that only systems with small delays are valid. Subsequently, Zhang et al [15] proposed a method to deal with cases with larger time delays. With the help of this method, they studied Turing instability induced by large time delays and analyzed the effect of delay on this instability. Moreover, wave patterns of RD equations with time delays were studied in [8, 16, 17].

Many phenomena in nature can be modeled by continuous RD processes. However, there are some other RD processes occur in discrete spaces (random networks) rather than in continuous spaces. One typical example is that the species are usually organized in patches in a predator–prey system [18], instead of being continuously distributed in space. The patches can be represented as nodes of a network such that predator and prey interact locally in each patch and diffuse through connected nodes. Another example for discrete version of RD process is found in metapopulation epidemic models [19, 20]. In this case, network nodes represent different locations such as cities or urban areas, individuals—who are divided into classes denoting their state with respect to the modeled disease such as infected, susceptible, immune and so on—moving between connected locations, and the reaction processes account for the possibility that individuals in the same location may get in contact and change their state according to the infection dynamics.

With the development of network science, the dynamical behaviors of RD systems defined on networks have received considerable attention. Early in 1971 Othmer et al [21] pointed out that Turing instability can occur in network organized systems and proposed a general mathematical framework for the analysis of such instability. Earlier research, however, were limited to regular lattices or small networks [22–24], which cannot account for the effect of network scale and topology on pattern formation. Recently, the key research results given by Nakao et al [25] changed this situation. They studied Turing patterns in large random networks, which reveal striking differences from the classical behavior and pave the way for novel discoveries in an area of widespread interest. Subsequently, further exploration has been done based on this seminal work. Fernandes et al [26] investigated Turing patterns in a predator–prey food webs with more than two species on networks. Asllani et al [27] studied the theory of pattern formation on directed networks. Perc et al [28] reviewed the cyclic dominance in evolutionary games focusing on pattern formation in which species contact in different networks. Kouvaris et al [29] extended a predator–prey model on multiplex networks.

Among these references mentioned above only RD systems on networks without time delay were considered. Here we investigate Turing patterns of a Leslie–Gower Holling-type III predator–prey model with time delay on networks. We have investigated the continuous version with delay in [30] and network version without delay. The results showed that these models have complex dynamical properties and instability induced by diffusion can generate stationary patterns. Moreover, for the model in continuous media, spiral waves could be resulted in if the instability is induced by time delay. Now that there exists wave patterns for continuous RD model when delay satisfies certain conditions, could the model on networks have similar phenomenon? If so, what forms of wave patterns will display in? What are the effects of network topology on wave patterns? Through the research we try to address these problems.

The paper is structured in the following way. In section 2, we introduce the Leslie–Gower Holling-type III predator–prey model with time delay on networks. In section 3, we analyze instability of the model induced by time delay with the help of linear stability method. Wave patterns and the effects of network topology on them are given in section 4 through simulations. Finally, we make some discussions in section 5.

2. Model with delay on networks

Holling-type III response exists universally in population dynamics. A predator–prey model incorporates the Leslie–Gower functional response as well as Holling-type III functional response can be written as

\[
\begin{align*}
\frac{du}{dt} &= f(u, v) = u(1 - u) - \frac{uv}{\alpha u + \beta}, \\
\frac{dv}{dt} &= g(u, v) = \beta vq(u, v) = \beta v \left(1 - \frac{u}{\bar{u}}\right),
\end{align*}
\]

(2.1)

with initial condition \(u(0) = u_0 > 0, v(0) = v_0 > 0\). This model describes a prey population \(u\) which serves as food for a predator with population \(v\). The parameters \(\alpha, \beta, \gamma\) are assumed to be only positive values. It should be noted that the first equation of system (2.1) is standard while the second equation is not because that the right-hand side in the second equation contains a Leslie–Gower term. The Leslie–Gower formulation is based on the assumption that reduction in a predator population has a reciprocal relationship with per capita availability of its preferred food. Indeed, Leslie introduced a predator–prey model where the carrying capacity of the predator environment is proportional to the number of prey. He stresses the fact that there are upper limits to the rates of increase of both prey \(u\) and predator \(v\), which are not recognized in the Lotka–Volterra model (see, e.g. [31]).
Including time delay in model (2.1) is a more realistic approach to the understanding of predator–prey dynamics. We introduce a single discrete delay \( \tau > 0 \) in the negative feedback of the predator’s density, then obtain the following system

\[
\begin{aligned}
\frac{du}{dt} &= f(u, v), \\
\frac{dv}{dt} &= \beta v q(u(t - \tau), v(t - \tau)).
\end{aligned}
\]  

Taking into account the inhomogeneous distribution of the predator and its prey in different spatial locations within a fixed bounded domain \( \Omega \subset \mathbb{R}^2 \) at any given time, and the natural tendency of each species to diffuse to areas of smaller population concentration, we arrive at a continuous RD system defined on \( Q = \Omega \times (0, \infty) \) with \( \Omega = (a, b)^2, \ b - a \gg 1, \)

\[
\begin{aligned}
\frac{\partial u}{\partial t} &= f(u, v) + d \Delta u \quad \text{in } Q, \\
\frac{\partial v}{\partial t} &= \beta v q(u(t - \tau), v(t - \tau)) + \sigma d \Delta v \quad \text{in } Q, \\
\frac{\partial u}{\partial n} &= 0 \quad \text{on } \partial \Omega \times (0, T), \\
u &= u_0(x, y, t), \quad v = v_0(x, y, t) \quad \text{in } \Omega \times [-\tau, 0].
\end{aligned}
\]  

Here the positive constants \( d \) and \( \sigma d \) denote diffusion coefficients, \( \Delta = \partial^2/\partial x^2 + \partial^2/\partial y^2 \) is the Laplacian operator, \( n \) is the outward unit normal vector of the boundary \( \partial \Omega \), \( u_0, v_0 \) are continuous positive functions. Time delay plays an important role in dynamical system (2.3), where it has been recognized to contribute critically to the stable or unstable outcome of prey density due to predation (see [30]).

Now we extend model (2.3) to the network analogue version where prey and predator occupy discrete nodes of a network and are diffusively transported over links connecting them. The links represent diffusive connections between species from one habitat to another of a network and are diffusively transported over links connecting them. The links represent diffusive transport of populations in space, while this operator is replaced by Laplacian matrices of some special networks, we illustrate it in appendix. For a given undirected network with \( N \) nodes, the Laplacian matrix \( L = (l_{ij})_{N \times N} \) is symmetric with \( L = A - \text{diag}(k_1, \ldots, k_N) \), where \( A = (a_{ij})_{N \times N} \) is the adjacency matrix and \( k_i \) is the degree of node \( i \). The adjacency matrix defines the topology of network and is given by \( a_{ij} = 1 \) if nodes \( i \) and \( j \) are connected and \( a_{ij} = 0 \) if they are not. The degree \( k_i = \sum_j a_{ij} \) is the number of connections of node \( i \). For convenience, we always sort random network nodes \( \{i\} \) in decreasing order of their degrees \( \{k_i\} \) so that the condition \( k_1 \geq k_2 \geq \cdots \geq k_N \) holds. Equations describing network organized prey–predator system are thus given by

\[
\begin{aligned}
\frac{du_i}{dt} &= f(u_i, v_i) + d \sum_{j=1}^N l_{ij} u_j \quad \text{in } (0, \infty), \\
\frac{dv_i}{dt} &= \beta v_i q(u_i(t - \tau), v_i(t - \tau)) + \sigma d \sum_{j=1}^N l_{ij} v_j \quad \text{in } (0, \infty), \\
u_i &= u_{i0}(t), \quad v_i = v_{i0}(t) \quad \text{in } [-\tau, 0].
\end{aligned}
\]

Here \( u_i(t), v_i(t), i = 1, 2, \ldots, N \) represent populations of prey and predator at time \( t \) in the habitat \( i \), respectively.

In the present paper we study the instability induced by time delay for model (2.4) and investigate corresponding patterns on some networks with different topologies.

### 3. Delay induced instability

In this section we give the stability analysis of network organized systems with time delay. The analysis method is performed in close analogy to the method proposed in [3, 14] applied to continuous model except some details. Assuming \( \tau \) to be small we replace \( u_i(t - \tau) \approx u_i - \tau \frac{du_i}{dt} \) and \( v_i(t - \tau) \approx v_i - \tau \frac{dv_i}{dt} \) in the second equation of (2.4) to write as

\[
\frac{dv_i}{dt} = \beta v_i q(u_i - \tau \frac{du_i}{dt}, v_i - \tau \frac{dv_i}{dt}) + \sigma d \sum_{j=1}^N l_{ij} v_j.
\]  

\[3.1\]
Expanding in Taylor series and neglecting the high-order nonlinearities, equation (3.1) becomes
\[
\frac{dv_i}{dt} = g(u_i, v_i) - \beta \tau a_i(u_i, v_i) v_i \frac{du_i}{dt} - \beta \tau a_i(u_i, v_i) v_i \frac{dv_i}{dt} + \sigma d \sum_{j=1}^{N} l_{ij} v_j.
\]
(3.2)

Let \((u_a, v_a)\) be the positive equilibrium of system (2.1) defined by the positive solution of \(f(u_a, v_a) = 0, g(u_a, v_a) = 0\). We now consider small spatiotemporal perturbations to the fixed point \((u_a, v_a)\) as
\[
u_i = u_a + \hat{\nu}_i, \quad v_i = v_a + \hat{\nu}_i.
\]
By expanding the reaction terms around this equilibrium in a Taylor series up to first order, it follows from the first equation of (2.4) and equation (3.2) that
\[
\begin{aligned}
\frac{d\hat{\nu}_i}{dt} &= f_u \hat{u}_i + f_v \hat{v}_i + d \sum_{j=1}^{N} l_{ij} \hat{u}_j, \\
\frac{d\hat{\nu}_i}{dt} + \beta \tau a_i v^*_a \frac{d\hat{\nu}_i}{dt} + \beta \tau a_i v^*_a \frac{d\hat{v}_i}{dt} &= g_u \hat{u}_i + g_v \hat{v}_i + \sigma d \sum_{j=1}^{N} l_{ij} \hat{v}_j.
\end{aligned}
\]
(3.3)

Note that the Laplacian of a network \(L\) is a real, symmetric and negative semi-definite matrix, its eigenvalues are real, non-positive. The eigenvalue \(\mu_i\) and its corresponding eigenvector \(\Phi_i\) are determined by \(L \Phi_i = \mu_i \Phi_i\) with \(\Phi_i = (\phi_i^{(1)}, \cdots, \phi_i^{(N)})^T, s = 1, \cdots, N\). Expressing spatiotemporal perturbation \(\hat{u}_i\) and \(\hat{v}_i\) in the form
\[
\hat{u}_i = \sum_{s=1}^{N} c_{is} e^{\lambda t} \phi_i^{(s)}, \quad \hat{v}_i = \sum_{s=1}^{N} c_{vs} e^{\lambda t} \phi_i^{(s)},
\]
upon inserting them in equation (3.3) and using
\[
\sum_{j=1}^{N} l_{ij} \phi_j^{(s)} = \mu_i \phi_j^{(s)}, \quad u_a = \gamma v_a, \quad q_u(u_a, v_a)v_a = 1, \quad q_v(u_a, v_a)v_a = -1,
\]
we obtain, for each mode \(s\), the following matrix equation for linear growth rates \(\lambda_i\):
\[
\begin{pmatrix}
\lambda_i - f_u - d \mu_i & -\frac{f_v}{\gamma} \\
\frac{\beta \tau}{\gamma} \lambda_i - g_u & \lambda_i(1 - \beta \tau) - g_v - \sigma d \mu_i
\end{pmatrix}
\begin{pmatrix}
c_{is} \\
c_{vs}
\end{pmatrix} = 0.
\]
Thus \(\lambda_i\) are given by the roots of characteristic polynomial
\[
\lambda^2 - A(\tau, \mu_i) \lambda_i + B(\tau, \mu_i) = 0,
\]
where
\[
A(\tau, \mu_i) = \frac{f_u + g_v - \tau \beta(f_u + \frac{1}{\gamma} f_v) + \mu_i[(1 + \sigma)d + \tau \beta d]}{1 - \tau \beta},
\]
\[
B(\tau, \mu_i) = \frac{\sigma d^2 \mu_i^2 + \mu_i d(\sigma f_u + g_v) + (f_u g_v - f_v g_u)}{1 - \tau \beta}.
\]
It is well known that the stability condition for system (2.4) is
\[
A(\tau, \mu_i) < 0, B(\tau, \mu_i) > 0, \quad \forall s \in \{1, 2, \cdots, N\}.
\]
(3.4)

In the absence of time delay, following Turing’s idea [25, 32], the equilibrium was originally stable, but become unstable under the influence of diffusion so that biological patterns formed. The former is equivalent to
\[
A(0, \mu_i) < 0, \quad B(0, \mu_i) > 0, \quad \forall s \in \{1, 2, \cdots, N\}.
\]
(3.5)

The latter requires that \(B(0, \mu_i) < 0\) for some mode \(s\). We now consider the first condition in (3.4) is not satisfied to explore instability induced by time delay for system (2.4). Let the homogeneous equilibrium for the system without delay be stable, i.e. condition (3.5) is satisfied. Note that \(\mu_i \leq 0\), the inequality \(A(\tau, \mu_i) > 0\) holds if
\[
(f_u + g_v) - \tau \beta(f_u + \frac{1}{\gamma} f_v) < 0 \quad \text{and} \quad 1 - \tau \beta < 0,
\]
which means

\[
\frac{1}{\beta} < \tau < \frac{f_u + g_v}{\beta(f_u + \frac{1}{2}f_v)}. \tag{3.6}
\]

In summary, (3.5)–(3.6) are sufficient conditions for instability induced by time delay. Under these two conditions Turing patterns can be observed.

### 4. Wave patterns on networks

In this section, we perform extensive numerical simulations on three different types of network with \(N = 10000\) nodes to investigate pattern formations of system (2.4). Each type of network include several cases with different average degrees as follows. (1) Two-dimensional (2D) lattices: \(N\) nodes are equally arranged in a lattice with \(M\) columns, \(M\) rows and the distances between columns (rows) are \(h = 1\). In the first case, two nodes are connected if the distance between them equals to 1. We name this case 'LA4' because that its average degree is \(\langle k \rangle = 4\). In the second and third cases, two nodes are connected if the distance between them is less than or equal to 2 and 3 respectively, which leads to \(\langle k \rangle = 12\) and \(\langle k \rangle = 23\), we name them 'LA12' and 'LA23' respectively. See figures 1(a), (b) for LA4 and LA12. (2) Erdős–Rényi (ER) random networks: Nine cases with average degrees \(\langle k \rangle = 2i, i = 2, 3, \ldots, 10\) will be used, we name them 'ER4, ER6, ..., ER20'. See figure 1(c) for ER4 and ER10. (3) Barabási-Albert (BA) scale-free networks: Nine cases with average degrees \(\langle k \rangle = 2i, i = 2, 3, \ldots, 10\) will be used, we name them 'BA4, BA6, ..., BA20'. See figure 1(d) for BA4 and BA10.

The positive equilibrium \((u_\infty, v_\infty)\) of (2.4) involves the root of a cubic polynomial, one can refer to [30] for details and we omit it here. The model parameters are set to

![Image](image_url)
According to the above parameters, we obtain the density of prey and predator in all nodes is a random perturbation around densities display cyclic variation over time and the oscillations of average density on LA12 and LA23 are more

Here $\text{randN}$ means condition $\text{l} = 0.3, \text{v}_b = \text{u}_b/\gamma$. According to the above parameters, we obtain $f(u) + g(v) = -0.770 732 < 0, f(u)g(v) = 0.470 732 > 0$ which means condition (3.5) holds and condition (3.6) can be stated as $1 < \tau < 1.637 306$. In the following simulation we choose time delay $\tau = 1.6$. Moreover, two different initial values are used in our computation, i.e.

(I) $u_{i,0}(t) = u_b + 0.0005 \times \text{randN}(0, 1), \quad v_{i,0}(t) = v_b + 0.0005 \times \text{randN}(0, 1);$

(II) $u_{i,0}(t) = u_b + 10^{-6}(m - 50), \quad v_{i,0}(t) = v_b + 10^{-6}(n - 50),$

Here $\text{randN}(0, 1)$ denotes random number of standard normal distribution. The initial condition (I) means that the density of prey and predator in all nodes is a random perturbation around $(u_b, v_b)$. The condition (II) we assume that the density is a small perturbation around $(u_b, v_b)$ except the nodes on some lines. For 2D lattices, if one node in $m$ column $n$ row then $i = (m - 1)M + n$ with $m, n = 1, \ldots, M, N = M^2$. For ER and BA networks, we firstly arrange the nodes equally in a $M \times M$ lattice and order them according to decreasing degree from left to right, bottom to top, then $m$ and $n$ are column and row indices of node $i$ respectively.

4.1. Wave patterns on 2D lattices

We study simulation results on 2D lattices in this subsection. Figures 2(a)–(c) show that periodic spiral waves are generated starting from initial data (I) on 2D lattices. The spiral waves appear as polycentric microspirals owing to the influence of initial condition (I) which is a small random perturbation around the positive equilibrium for all nodes. Comparing figure 2(a)–(c), it can be easily noticed that the spatial frequency of microspirals on LA12 is smaller than that on LA4 and larger than that on LA23. Specifically, the microspirals on LA4 have more centers than those on LA12 and LA23. In figures 2(d)–(f), single-arm spiral waves are generated on 2D lattices with initial data (II). Here again we see that the spatial frequency of spiral wave on LA12 is smaller than spatial frequency on LA4 while larger than that on LA23. The most essential reason for this phenomenon is that the nodes on LA12 and LA23 communicate more with other nodes. We show average densities of all nodes for prey on 2D lattices with initial values (I) and (II) in figures 3(a)–(c) and (d)–(f) respectively. It is apparent that average densities display cyclic variation over time and the oscillations of average density on LA12 and LA23 are more regular.

Changes in the average density of prey have followed a cyclical wave pattern. The two main characteristics of wave pattern are amplitude (i.e. peak and trough) and period. We next explore the influences of network topology on wave pattern from these two characteristics. Numerical results in figure 4 (see black lines) illustrate that average densities on 2D lattices maintaining a steady state when $t \in [300, 1000]$. This stabilization displays in two aspects: Firstly, with the increases of average degree of 2D lattice, the differences of maximum values, minimum values and amplitudes of average density are very small. In fact, the maximums range from 0.29 to 0.33 and the minimums from 0.26 to 0.29 on LA4, LA12 and LA23 (see figure 4(a), (c)). Secondly, the periods of average density are almost constant (approximately 8, see figures 4(b), (d)) as the average degree changes.
4.2. Wave patterns on random networks

In this subsection we investigate simulation results on random networks. Under the present parameters setting, the prey densities, similar to results on 2D lattices, display periodic variation on ER and BA networks. Figures 5 and 6 show prey densities at different times with initial condition (I) on ER4, ER10 and BA4, BA10 networks respectively; figures 9 and 10 show the corresponding results with initial condition (II). The prey distributions

Figure 3. Average densities of all nodes for prey on (a) LA4, (b) LA12, (c) LA23 with initial condition (I) and (d) LA4, (e) LA12, (f) LA23 with initial condition (II).

Figure 4. The maximum values, minimum values and periods of average density for prey with $t \in [300, 1000]$ and (a), (b) initial condition (I) and (c), (d) initial condition (II) on each type of network.
on network nodes are roughly separated into one group, while only a limited fraction of nodes do not belong to this group (see panels (a)–(c) in figures 5, 6 and 9, 10). Along with the increases of average degree of random network, the number of nodes which dissociate from the group becomes fewer and fewer (see panels (d)–(f) in figures 5, 6 and 9, 10). In other words, the differences of prey density among most nodes are very small at a definite time, especially on BA10 network, all nodes have the same prey density (see panels (d)–(f) in figures 6 and 10).

Figures 7 and 11 show average densities of all nodes for prey on ER 2i, i = 2, ..., 7 networks with initial conditions (I) and (II) respectively, from which periodic wave patterns can be observed. It is intuitively plausible that, on ER4, ER6, ER8, ER10 and ER12, the waves with larger average degrees have higher peaks, lower troughs as well as longer periods. Indeed, red lines in figure 4 confirm these observation results. Take simulation results on ER networks with initial condition (II) for example, the maximum values of prey average density on ER4, ER6, ER8, ER10, ER12 are 0.6780, 0.8329, 0.9062, 0.9658, 1.0000, the minimum values are 0.0883, 0.0320, 0.0192, 0.0164, 0.0157, the periods are 9.72, 10.58, 11.61, 13.45, 152. It is worth emphasizing that the period has
a rapid increase when the maximum value increases to 1, thereafter the prey–predator system reaches a steady state: the maximum is stabilized at 1, the minimum at 0.016 and the period at 152. Moreover, all nodes have an identical density at any given time once the system enters into the steady state, which implies that figures 7, 11(e), (f) are, in fact, density evolution of each node. Figures 8 and 12 show prey average densities on BA2i, i = 2, ..., 7 networks with initial conditions (I) and (II) respectively. One can observe that changing initial condition or network type do not alter the steady state. The differences are that the amplitudes and periods of average density on BA networks approach steady state more quickly (see blue lines in figure 4) which can also be confirmed via numerical results on ER10 network and BA10 network (see panels (d) in figures 7, 8 and 11, 12). These results demonstrate that average degree of random network has a significant effect on wave patterns, and the effect becomes more prominent on BA networks.

Through extensive simulation experiments, we found that the prey–predator system (2.4) can achieve stability both on 2D lattices and on random networks with some certain average degrees. However, there are fundamental differences between these two steady states. For steady state on 2D lattices, the amplitudes and
periods of wave patterns are small and the prey can keep a certain number. While for steady state on random networks, the wave patterns have long periods, the prey density in a period is kept to peak value 1 most of the time, and near 0 in a very short time. In addition, average degree has only a small effect on the steady state for 2D lattices, but has significant effects for random networks. Trace its root, nodes of 2D lattices have the same degree and share the same important place in the RD process. However, node degrees of random networks especially for BA networks can vary widely which leads to these nodes have different status and play different roles in the RD process.

From the viewpoint of ecology, the steady state on random networks is a double-edged sword: on the one hand, population number maintains a high level for much of the time is benefit to protect species diversity. On the other hand, biological invasion may lead to species extinction at the time when prey density near 0. Thus, understanding network topology of population distribution is significant for us to maintain the ecological balance.

Figure 9. The prey densities at different times on ER networks (ER4 (a)–(c), ER10 (d)–(f)) with initial condition (II). (a), (d) \( t = 900 \), (b), (e) \( t = 906 \), (c), (f) \( t = 912 \).

Figure 10. The prey densities at different times on BA networks (BA4 (a)–(c), BA10 (d)–(f)) with initial condition (II). (a) \( t = 900 \), (b) \( t = 906 \), (c) \( t = 912 \), (d) \( t = 930 \), (e) \( t = 936 \), (f) \( t = 942 \).
5. Discussion

We have studied instability induced by time delay for a predator–prey model with modified Leslie–Gower and Holling-type III schemes on complex networks. Instability conditions were obtained via linear stability analysis of network organized systems which are performed similarly with continuous model. Extensive simulations show that wave patterns can be generated for the present model if time delay satisfies instability conditions. Because of considering network organized systems, wave patterns are influenced by network topology inevitably. We explored the influences mainly from two aspects, i.e. wave amplitude (the size of peak and trough) and wave period using deterministic networks—2D lattices and random networks—ER networks and BA networks.

Spiral waves for prey densities can be generated on 2D lattices and spatial frequencies of waves decrease with the increases of average degree. Even so, prey average densities on LA4, LA12 and LA23 are still in a steady state.

Figure 11. Average densities of all nodes for prey on ER networks with initial condition (II) and different average degrees. (a) ER4, (b) ER6, (c) ER8, (d) ER10, (e) ER12, (f) ER14.

Figure 12. Average densities of all nodes for prey on BA networks with initial condition (II) and different average degrees. (a) BA4, (b) BA6, (c) BA8, (d) BA10, (e) BA12, (f) BA14.
This is shown in two aspects: the amplitudes of average density are very small (the peaks and troughs of average density equal approximately to 0.3) and the periods change a little (equal to about 8). In this sense, 2D lattices have little effect on wave patterns. In contrast, numerical results on ER and BA networks particularly the latter suggest that the effects of these two networks on wave patterns are huge. In the beginning, the amplitudes and periods increase with the increases of average degree of ER/BA networks. Then, there is a sudden-change of period (increase from 14 on ER10 and 12 on BA8 to 152 on ER12 and BA10) when the maximum value of average density up to 1. After that, the prey average density enters into another steady state. Compared with the steady state on 2D lattices, the former has a higher peak (equals to 1), lower trough (equals to about 0.016) as well as longer period (equals to about 152). The characteristic of results on ER and BA networks has two sides from the point of view of ecosystem equilibrium and environmental protection. In particular, protection efforts should be enhanced when population density at the wave trough to prevent population extinction.

In the process of instability conditions derivation we assume that the time delay is small so that a Taylor expansion could be employed to reduce system with time delay to those without delay. Then Turing’s idea can be followed. In practice, however, time delays in biological systems may be large. One of our future work is to study the influences of large time delay and network topology (such as community structure, clustering coefficient and average degree) on pattern formations. Besides, metapopulation epidemic models can be described by RD process on networks. Study of disease spreading using network organized system is also our further work.

Acknowledgments

This research is supported by the National Natural Science Foundation of China under grants 11701348, U1803263, 11671241 and 61873154, the Fundamental Research Funds for the Central Universities (Grant No. 3102018zy034), the National 1000 Young Talent Plan (Grant No. W099102), Shaanxi Natural Science Foundation-Key Program (Grant No. 2019ZDLGY17-07), Key Area R & D Program of Guangdong Province (No. 2019B010137004).

Appendix. From continuous model to discrete model

The continuous model (2.3) can natural transition to its network version (2.4) if one chooses an especial network. To this end, we need to rely on numeric discreteness of continuous model. Of varied numerical methods, finite difference method replaces the derivative in PDEs with difference formula, which is simple, intuitionistic and widely used. Now we introduce the spatially semidiscrete approximation to (2.3) using finite difference method.

Let $\Omega = (1, b) \times (1, b)$ with $b \gg 1$ and $h = (b - 1)/(M - 1)$, $M \in \mathbb{N}^+$, we get the mesh

$$\Omega_h = \{(x_m, y_n); x_m = (m - 1)h + 1, y_n = (n - 1)h + 1, m, n = 1, 2, \cdots, M\}.$$ 

Generally, the process of pattern forms in space for continuous model is an asymptotic behavior with large scale and long time, therefore setting the mesh size $h = 1$ is acceptable when $b \gg 1$ (see figure 1(a)).

According to Taylor expansion, one has

$$\frac{\partial^2 u(x_m, y_n)}{\partial x^2} = \frac{u(x_{m+1}, y_n) - 2u(x_m, y_n) + u(x_{m-1}, y_n)}{h^2} + O(h^2). \quad (A.1)$$

Denoting $u_{mn}$ is the finite difference approximation to $u(x_m, y_n)$ and using (A.1) with $h = 1$, the spatially semidiscrete scheme for prey equation in (2.3) takes the form

$$\frac{du_{mn}}{dt} = f(u_{mn}, v_{mn}) + d(u_{m-1,n} + u_{m,n-1} - 4u_{mn} + u_{m,n+1} + u_{m+1,n}). \quad (A.2)$$

There is no problem for (A.2) if $(x_m, y_n)$ is an internal node, i.e. $(x_m, y_n) \in \Omega_h$. If $(x_m, y_n)$ is a boundary node, takes $(x_1, y_1)$ as example, it follows from (A.2) that

$$\frac{du_{11}}{dt} = f(u_{11}, v_{11}) + d(u_{01} + u_{10} - 4u_{11} + u_{12} + u_{21}). \quad (A.3)$$

The above formula include nodes $(0, 1), (1, 0)$ which do not belong to $\Omega_h$. The zero-flux boundary conditions in (2.3) are needed to eliminate the terms relating to those nodes. Note that the boundary condition of $u$ is equivalent to
Using Taylor expansion again, we have

\[
\frac{\partial u}{\partial x} \bigg|_{x=1} = \frac{\partial u}{\partial x} \bigg|_{x=b} = 0, \quad \frac{\partial u}{\partial y} \bigg|_{y=1} = \frac{\partial u}{\partial y} \bigg|_{y=b} = 0.
\]

Thereby \( u_{01} = u_{10} = u_{11} \) and (A.3) can be rewritten as

\[
\frac{du_{11}}{dt} = f(u_{11}, v_{11}) + d(-2u_{11} + u_{12} + u_{21}). \tag{A.4}
\]

Other boundary nodes are handled similarly.

For node \((m, n)\) we denote \(i = (m - 1)M + n\) with \(m, n = 1, \cdots, M\), then the spatially semidiscrete scheme for prey equation is given as follows,

\[
\frac{du_{i}}{dt} = f(u_{i}, v_{i}) + d \sum_{j=1}^{N} l_{ij} u_{j} \quad \text{in}(0, \infty). \tag{A.5}
\]

Here the matrix \(L = (l_{ij})_{N \times N}\) is discrete Laplacian operator, its elements satisfy

\[
l_{ij} = \begin{cases} 
0, & \text{nodes } i, j \text{ are not connected and } i \neq j, \\
1, & \text{nodes } i, j \text{ are connected and } i \neq j, \\
-2, & i = j \text{ and node } i \text{ in the corner of } \Omega, \\
-3, & i = j \text{ and node } i \text{ at the boundary of } \Omega \text{ except corner,} \\
-4, & i = j \text{ and node } i \text{ in the interior of } \Omega.
\end{cases} \tag{A.6}
\]

Considering the mesh \(\Omega_{h}\) as a network, it is deterministic with average degree \((k) = 4\) and \(L\) is its Laplacian matrix.

References

[1] Choudhury S R 1994 SIAM J. Appl. Math. 54 1425
[2] Wang W D and Zhao X Q 2011 SIAM J. Appl. Math. 71 147
[3] Ghosh P 2011 Phys. Rev. E 84 016222
[4] Yuan G Y, Yang S P, Wang G R and Chen S G 2008 Commun. Theor. Phys. 49 174
[5] Szolnoki A and Perc M 2013 Phys. Rev. E 87 054801
[6] Martin A and Ruan S G 2001 J. Math. Biol. 43 247
[7] Faria T 2001 J. Math. Anal. Appl. 254 433
[8] Gourley SA and Kuang Y 2005 SIAM J. Appl. Math. 65 550
[9] Hadeler K P and Ruan S G 2007 Discrete Contin. Dyn. B 8 95
[10] Gan Q T, Xu R and Yang P H 2011 Nonlinear Anal.: RWA 12 52
[11] Chang L L and Jin Z 2018 Appl. Math. Comput. 316 138
[12] Piotrowska M J 2003 Math. Comput. Model. 42 123
[13] Banerjee M and Zhang L 2014 Chaos Solitons Fractals 67 73
[14] Sen S, Ghosh P, Riaz S S and Ray D S 2009 Phys. Rev. E 80 046212
[15] Zhang T H and Zhang H 2014 Phys. Rev. E 90 052908
[16] Ruan S G 1998 IMA J. Appl. Math. 61 15
[17] Wang Q Y, Perc M, Duan Z S and Chen G R 2008 Phys. Lett. A 372 5681
[18] Jansen V A A and Lloyd A L 2000 J. Math. Biol. 41 232
[19] Colizza V, Pastor-Satorras R and Vespignani A 2007 Nat. Phys. 3 276
[20] Masuda N 2010 New J. Phys. 12 093009
[21] Othmer H G and Scriven L E 1971 J. Theor. Biol. 32 507
[22] Othmer H G and Scriven L E 1974 J. Theor. Biol. 43 83
[23] Platé E 2001 J. Math. Biol. 43 411
[24] Moore P K and Horshiemke W 2005 Physica D 206 121
[25] Nakao H and Mikhailov A S 2010 Nat. Phys. 6 644
[26] Fernandes L D and de Aguiar M A M 2012 Phys. Rev. E 86 056203
[27] Asllani M, Challenger J D, Pavone F S, Sacconli L and Fanelli D 2014 Nat. Commun. 5 4517
[28] Szolnoki A et al 2014 J. R. Soc. Interface 11 20140735
[29] Kouvaris N E, Hata S and Gullera A D 2015 Sci. Rep. 5 10840
[30] Wang C X, Chang L L and Liu H F 2016 PLoS One 11 e0150503
[31] Nindjina A F, Aziz-Alaoui M A and Cadivel M 2006 Nonlinear Anal.: RWA 7 1104
[32] Turing A M 1952 Phil. Trans. R. Soc. B 237 37