Pattern dynamics of network-organized system with cross-diffusion*

Qianqian Zheng(郑前前)[1], Zhijie Wang(王直杰)[1,†], and Jianwei Shen(申建伟)[2,‡]

[1] College of Information Science and Technology, Donghua University, Shanghai 201620, China

[2] Institute of Applied Mathematics, Xuchang University, Xuchang 461000, China

(Received 23 July 2016; revised manuscript received 20 November 2016; published online 10 January 2017)

Cross-diffusion is a ubiquitous phenomenon in complex networks, but it is often neglected in the study of reaction–diffusion networks. In fact, network connections are often random. In this paper, we investigate pattern dynamics of random networks with cross-diffusion by using the method of network analysis and obtain a condition under which the network loses stability and Turing bifurcation occurs. In addition, we also derive the amplitude equation for the network and prove the stability of the amplitude equation which is also an effective tool to investigate pattern dynamics of the random network with cross diffusion. In the meantime, the pattern formation consistently matches the stability of the system and the amplitude equation is verified by simulations. A novel approach to the investigation of specific real systems was presented in this paper. Finally, the example and simulation used in this paper validate our theoretical results.

Keywords: cross diffusion, random network, Turing instability, amplitude equation

PACS: 05.10.–a, 05.40.Ca, 05.65.+b, 05.70.Ln **DOI:** 10.1088/1674-1056/26/2/020501

1. Introduction

The pattern formation was first investigated and interpreted by Alan Turing in a reaction–diffusion system which describes the evolutionary change of two or more chemical systems in 1960s.[1] Since the seminal paper of Turing, most scholars began to pay much attention to theoretical models and try to explain self-organized pattern formations in many different areas, such as physics, chemistry, biology, geology, and so on. Some possible models have been proposed in Refs. [2], [3], and [4]. Green identified two chemicals that behave as an activator and inhibitor, giving rise to the regularly spaced ridges found in the roof of the mouth of mouse embryos. The researchers showed that the pattern of ridges is affected just as Turing's equation when the activity of those chemicals is increased and decreased.[5] Further, during the last few decades, spatial patterns in reaction–diffusion systems have attracted much interest for experimentalists and theorists. However, until now, no general analysis of the possible role of cross diffusion in dissipative pattern network has been proposed.[6]

Regarding networks, Lu, Henry and Chen introduced some basic mathematical models of complex dynamical networks, as well as their synchronization and control problems.[7] Further, McGraw and Menzinger examined numerically the three-way relationships among structure, Laplacian spectra, and frequency synchronization dynamics of complex networks.[8] Alex Arenas *et al.* reviewed several applications of synchronization in complex networks to different disciplines, such as biological systems and neuroscience, engineering and computer science, and economics and social sciences,[9] in addition, shen *et al.* presented promising results on network regulated by small RNAs.[10,11]

The application of these ideas to random networks can be traced back to early 70s, Othmer and Scriven pointed out that Turing instability is also possible in networks which is important for understanding of multi-cellular morphogenesis.[12] Later, Nakao, and Mikhailov investigated Turing patterns formed by activator–inhibitor systems in large random networks, and showed the striking difference between them and known classical systems. As a result, the approximate mean-field theory of nonlinear Turing patterns in the networks was constructed.[13,14] Although, recently much attention has been paid to complex networks and nonlinear dynamics, most of the researches focused on either synchronization phenomena of oscillator networks[15,16] or simple examples of regular lattices.[17–19] In addition, the amplitude equation was usually defined as the variance or not considered at all, and also the pattern formation was not presented.

Besides, in recent years, many scientists deemed that mathematical modeling could be used to investigate the differences at the dynamic level between healthy and pathologic configurations of biological pathways.[20–22] Many studies have been performed in these directions, including research on noise,[23–26] delay and bifurcation,[27–30] diffusion,[31–33] and so on. As practice shows researchers often investigate a system involving a variety of elements and interacting with each other, but most of them avoid interactions themselves and just study the random network or cross diffusion.

*Project supported by the National Natural Science Foundation of China (Grant Nos. 11272277, 11572278, and 11572084) and the Innovation Scientists and Technicians Troop Construction Projects of Henan Province, China (Grant No. 2017JR0013).

†Corresponding author. E-mail: wangzj@dhu.edu.cn

‡Corresponding author. E-mail: phdshen@126.com

© 2017 Chinese Physical Society and IOP Publishing Ltd

In order to better understand the reaction–diffusion model of the network, first, we propose to study the pattern formations on the network with cross diffusion. In this paper, we choose the PORD system[34] to try to explain somite patterning in embryos and obtain some interesting results in this direction. Then we investigate the relationship between the reaction–diffusion system and network, and reveal how the dynamics of the model regulation is affected by coefficients and random probability which provides a way to investigate the mechanism of pattern formation. In addition, we also derive the amplitude equation and prove its logical with simulations and try to explain some mechanisms with our results.

The paper is organized as follows. In Section 2, we provide a model of the network-organized system. In Section 3, we derive the amplitude equation. In Section 4, we utilize an example to illustrate the application of these ideas and using simulations validate theoretical results and present some interesting pattern dynamical phenomena. Finally, we summarize our results and conclusion in the last section.

2. Network-organized systems with cross diffusion

Diffusion and random are familiar phenomena in the nature. It is well known that diffusion in the network interaction is often cross-diffusive and its connections are usually random. Complex networks with cross diffusion and random connection can emerge different patterns and make the network lose instabilities. These phenomena can be accompanied by spatial changes and can induce miscellaneous mechanisms in the biological and physical systems. In order to study the reaction–diffusion network and its instability, we develop the matrix and amplitude equation of the network and induce the condition under which the network loses stability and Turing bifurcation occurs. By using these methods and tools, we can obtain the spatial mechanism of reaction–diffusion network which induces surprising patterns. Now we analyze the dynamics and behavior of networks (as shown in Fig. 1) with cross diffusion.[34] For models of somite patterning, the interaction is abstracted as a simple diffusion process and a far greater number of nodes in the network and the system should be considered in the network, which also satisfies the mechanism of somite.

The kinetic equation with cross diffusion can be rewritten as follows:

$$\begin{aligned}\frac{\partial u_i}{\partial t} &= f(u_i,v_i)+d_1\nabla^2 u_j+d_2\nabla^2 v_j,\\ \frac{\partial v_i}{\partial t} &= g(u_i,v_i)+d_3\nabla^2 u_j+d_4\nabla^2 v_j,\end{aligned} \tag{1}$$

where

$$f(u,v)=\frac{k_1u-k_2v+\beta}{1+k_1u-k_2v+\beta}-k_4u,$$

$$g(u,v)=\frac{k_3u}{1+k_3u}-k_4v,$$

and u_i and v_i are densities of a cell-autonomous activator and a diffusible repressor, k_1, k_2, and k_3 define strengths of regulatory interactions between u_i and v_i, k_4 is a fixed decay constant, β is regulatory input. Here $(k_1u-k_2v+\beta)/(1+k_1u-k_2v+\beta)$ may be negative and is allowable in the Turing system.[1] d_i, $i=1,2,3,4$ are the diffusion constants of species, and we assume $(k_1u-k_2v+\beta)/(1+k_1u-k_2v+\beta)>0$.

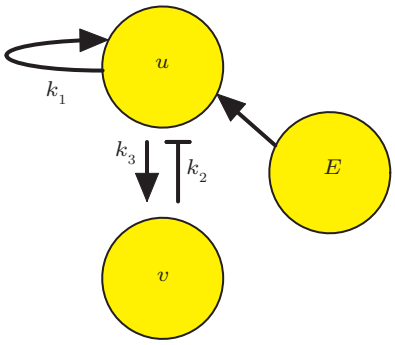


Fig. 1. (color online) A network of two nodes: a cell-autonomous activator (u), a diffusible repressor (v), and external input (E).

In general, the diffusive mobility of species u_i and v_i on the network are different. Equation (1) on the network was presented, where the species occupy discrete nodes of a network and are diffusively transported over the links connecting them and the connected network consisting of N nodes, $i=1,\ldots,N$ is considered in this paper. Then the equations describing cross-diffusion systems with Laplacian matrix on the network[12] have the following form,

$$\begin{aligned}\frac{\partial u_i}{\partial t} &= f(u_i,v_i)+d_1\sum_{j=1}^{N}L_{ij}u_j+d_2\sum_{j=1}^{N}L_{ij}v_j,\\ \frac{\partial v_i}{\partial t} &= g(u_i,v_i)+d_3\sum_{j=1}^{N}L_{ij}u_j+d_4\sum_{j=1}^{N}L_{ij}v_j,\end{aligned} \tag{2}$$

where $i=1,\ldots,N$, and L represents a random network matrix. Here, the local species dynamics of individual nodes are defined as $f(u_i,v_i)$ and $g(u_i,v_i)$.

First, the linear stability analysis is performed at (u_0,v_0) for system (2), where the uniform stationary state (u_0,v_0) satisfies $f(u_0,v_0)=0$, $g(u_0,v_0)=0$. We obtain the following equations with small perturbations u_i and v_i by linearization of the system (2),

$$\begin{aligned}\frac{\partial u_i}{\partial t} &= f_uu_i+f_vv_i+d_1\sum_{j=1}^{N}L_{ij}u_j+d_2\sum_{j=1}^{N}L_{ij}v_j,\\ \frac{\partial v_i}{\partial t} &= g_uu_i+g_vv_i+d_3\sum_{j=1}^{N}L_{ij}u_j+d_4\sum_{j=1}^{N}L_{ij}v_j.\end{aligned} \tag{3}$$

As we all know that the solution of the system (3) has the

following form in Fourier space,

$$u_i = \sum_{k=1}^{N} C_k \mathrm{e}^{\lambda_k t} \Phi_i^k,$$
$$v_i = \sum_{k=1}^{N} B_k C_k \mathrm{e}^{\lambda_k t} \Phi_i^k, \tag{4}$$

where $\sum_{j=1}^{N} L_{ij}\Phi_i^k = \alpha_k \Phi_i^k$ and α_k is the eigenvalue of L. By substituting Eq. (4) into Eq. (3), the characteristic equation can be obtained by expanding the perturbations over the Laplacian normal modes,

$$\lambda_k \begin{pmatrix} 1 \\ B_k \end{pmatrix} = \begin{pmatrix} f_{u_i} + d_1\alpha_k & f_{v_i} + d_2\alpha_k \\ g_{u_i} + d_3\alpha_k & g_{v_i} + d_4\alpha_k \end{pmatrix} \begin{pmatrix} 1 \\ B_k \end{pmatrix},$$

and,

$$\begin{vmatrix} \lambda_k - f_{u_i} - d_1\alpha_k & -f_{v_i} - d_2\alpha_k \\ -g_{u_i} - d_3\alpha_k & \lambda_k - g_{v_i} - d_4\alpha_k \end{vmatrix} = 0,$$
$$\lambda_k^2 - b\lambda_k + c = 0, \tag{5}$$

where,

$$b = g_{v_i} + d_4\alpha_k + f_{u_i} + d_1\alpha_k,$$
$$c = (d_1d_4 - d_3d_2)\alpha_k^2 + (d_1g_{v_i} + d_4f_{u_i} - d_3f_{v_i} - d_2g_{u_i})\alpha_k + f_{u_i}g_{v_i} - f_{v_i}g_{u_i},$$
$$\lambda_k = \frac{b \pm \sqrt{b^2 - 4ac}}{2}.$$

The linear growth rate λ_k of the k-th mode is determined from the characteristic equation. Based on the theory of stability, when $\mathrm{Re}\,\lambda_k$ is positive, the k-th mode is unstable. At the instability threshold, $\mathrm{Re}\,\lambda_k = 0$ for some $k = k_\mathrm{c}$ and $\mathrm{Re}\,\lambda_k < 0$ for all other modes. As we know that the system is unstable with diffusion when the system without diffusion is stable, which means the Turing instability occurs. In this system, the condition of the Turing instability should satisfy the following condition.

(i) $b < 0$ and $c > 0$ when $d_i = 0$, $i = 1,2,3,4$.

(ii) $c < 0$ when $d_i \neq 0$, $i = 1,2,3,4$.

In addition, we can obtain the critical value of $c < 0$ about α_k and make the minimum $c(\alpha_{kc}) = 0$, where

$$\alpha_{kc} = \frac{d_1g_{v_i} + d_4f_{u_i} - d_3f_{v_i} - d_2g_{u_i}}{-2(d_1d_4 - d_3d_2)}.$$

3. Amplitude equation of network-organized systems

As is shown in many research papers, the amplitude equation plays an important role in studying the pattern formation dynamics, which is always defined as variance in network-organized systems and the derivation process is also complicated. It was seldom investigated in some biological systems. Here we shall derive the amplitude equation in the network with cross diffusion, study the stability of the amplitude equation, and present the biological mechanism about pattern formation.

In general, the equation has the following form,

$$\frac{\partial c}{\partial t} = Lc + N.$$

For convenience, only linear part will be considered in the following and it is valid for nonlinear part in the same way. The linear part can be rewritten as,

$$\frac{\partial u_i}{\partial t} = f_u u_i + f_v v_i + d_1 \sum_{j=1}^{N} L_{ij}u_j + d_2 \sum_{j=1}^{N} L_{ij}v_j,$$
$$\frac{\partial v_i}{\partial t} = g_u u_i + g_v v_i + d_3 \sum_{j=1}^{N} L_{ij}u_j + d_4 \sum_{j=1}^{N} L_{ij}v_j,$$
$$\frac{\partial c}{\partial t} = Lc, \tag{6}$$

where,

$$c = \begin{pmatrix} u_i \\ v_i \end{pmatrix}$$

is the variables and

$$L = \begin{pmatrix} f_u + d_1\nabla^2 & f_v + d_2\nabla^2 \\ g_u + d_3\nabla^2 & g_v + d_4\nabla^2 \end{pmatrix}$$

is a linear operator.

In the following, we use multiple scale analysis to derive the amplitude equations when $\alpha_k = \alpha_{kc}$. The dynamical behavior should be investigated when the controlled parameter is larger than the critical value of the Turing point. Then we expand f_v and the variables when f_v is close to f_{vc}.

$$f_v - f_{vc} = \varepsilon f_1 + \varepsilon^2 f_2 + \cdots,$$
$$\begin{pmatrix} u_i \\ v_i \end{pmatrix} = \begin{pmatrix} u_{i1} \\ v_{i1} \end{pmatrix} \varepsilon + \begin{pmatrix} u_{i2} \\ v_{i2} \end{pmatrix} \varepsilon^2 + \cdots.$$

The linear operator L can be expanded as

$$L = L_c + (f_v - f_{vc})M,$$
$$L_c = \begin{pmatrix} f_u + d_1\nabla^2 & f_{vc} + d_2\nabla^2 \\ g_u + d_3\nabla^2 & g_v + d_4\nabla^2 \end{pmatrix}, \quad M = \begin{pmatrix} 0 & 1 \\ 0 & 0 \end{pmatrix}. \tag{7}$$

Let

$$T_0 = t, \quad T_1 = \varepsilon t, \quad T_2 = \varepsilon^2 t, \ldots$$

and T_i is a dependent variable. For the derivation of time, we have that

$$\frac{\partial}{\partial t} = \frac{\partial}{\partial T_0} + \varepsilon\frac{\partial}{\partial T_1} + \varepsilon^2\frac{\partial}{\partial T_2} + \cdots,$$

where the amplitude of the pattern formation is considered as slow variable and the $\partial/\partial T_0$ does not work. Then,

$$\frac{\partial}{\partial t} = \varepsilon\frac{\partial}{\partial T_1} + \varepsilon^2\frac{\partial}{\partial T_2} + \cdots.$$

Substituting the above equations into Eq. (6) and expanding Eq. (6) to different orders of ε, we can obtain three equations as follows:

$$\varepsilon : L_c \begin{pmatrix} u_{i1} \\ v_{i1} \end{pmatrix} = 0,$$

$$\varepsilon^2 : L_c \begin{pmatrix} u_{i2} \\ v_{i2} \end{pmatrix} = \frac{\partial}{\partial T_1} \begin{pmatrix} u_{i1} \\ v_{i1} \end{pmatrix} - f_1 M \begin{pmatrix} u_{i1} \\ v_{i1} \end{pmatrix}.$$

We first consider the case of the first order ε. Since L_c is the linear operator of the system close to the onset, $(u_{i1}, v_{i1})^{\mathrm{T}}$ is a linear combination of the eigenvectors that corresponds to the eigenvalue zero. Therefore,

$$\begin{pmatrix} u_i \\ v_i \end{pmatrix} = \sum_{j=1}^{3} \begin{pmatrix} x_{ij} \\ y_{ij} \end{pmatrix} \mathrm{e}^{\mathrm{i}k_j r} + \text{c.c.},$$

and we obtain $x_i = By_i$. Let $x_{ij} = B$ by assuming $y_{ij} = 1$ then,

$$\begin{pmatrix} u_{i1} \\ v_{i1} \end{pmatrix} = \begin{pmatrix} A \\ 1 \end{pmatrix} (W_j \mathrm{e}^{\mathrm{i}k_j r} + \text{c.c.}), \quad (j = 1,2,3),$$

where

$$A = \frac{d_2 k_c^2 - f_{vc}}{f_u - d_1 k_c^2},$$

$|\alpha_c| = k_c^2$, and W_j is the amplitude of the mode $\mathrm{e}^{\mathrm{i}k_j r}$.

Now, we consider the case of the second order ε^2. According to the Fredholm solubility condition, the vector function of the right-hand side of the above equation must be orthogonal to the zero eigenvectors of operator L_c^+. And the zero eigenvectors of adjoint operator L_c^+ are,

$$\begin{pmatrix} B \\ 1 \end{pmatrix} \mathrm{e}^{-\mathrm{i}k_i r},$$

where

$$B = \frac{d_3 k_c^2 - g_u}{f_u - d_1 k_c^2}.$$

It is found from the orthogonality condition that,

$$\begin{pmatrix} B & 1 \end{pmatrix} \mathrm{e}^{-\mathrm{i}k_i r} L_c \begin{pmatrix} u_2 \\ v_2 \end{pmatrix} = 0,$$

and

$$\frac{\partial}{\partial T_1}(Bu_{i1} + v_{i1}) - f_1 B v_{i1} = 0.$$

We will investigate $\mathrm{e}^{\mathrm{i}k_1 r}$ only in the following case, we can achieve results in other cases by changing subscripts and we will not describe in details here, thus we have

$$\frac{\partial W_i}{\partial T} = \frac{(f_v - f_{vc})B}{AB+1} W_i = \mu W_i, \tag{8}$$

where

$$\mu = \frac{(d_3 k_c^2 - g_u)(f_u - d_1 k_c^2)}{f_u - d_1 k_c^2 + (d_3 k_c^2 - g_u)(d_2 k_c^2 - f_v)} (f_v - f_{vc}).$$

It is unstable when $\mu > 0$, and stable when $\mu < 0$ based on the theory of stability.

4. Simulation

In this section, we will carry out a detailed approximate numerical investigation of generic Turing system and examine the effects of the network on pattern formations. All the numerical simulations employ the method of lines finite difference approximation and the periodic domain boundary conditions. Here the negative value just stands for the size of concentration and is allowable in the Turing system.[1] Scale-free networks (as shown in Fig. 2) are generated randomly according to diffusion. Simple networks are generated by preparing $N = 128^2$ nodes and then randomly connecting two arbitrary nodes with probability $p = 10^{-6}$ which also stands for the level of complexity, yielding the mean degree of Np.[18]

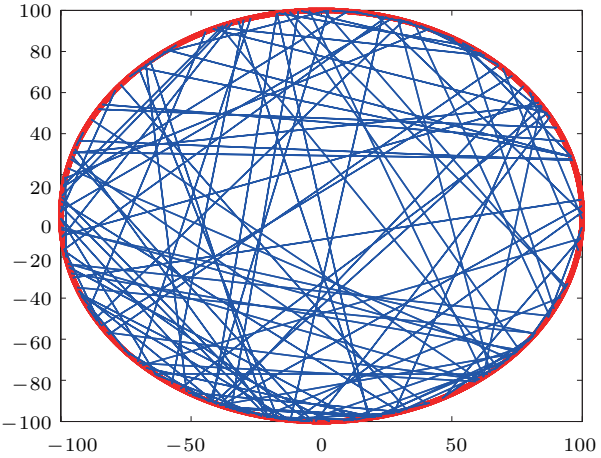


Fig. 2. (color online) Network graphs are generated randomly according to diffusion when $p = 10^{-6}$, $N = 128^2$.

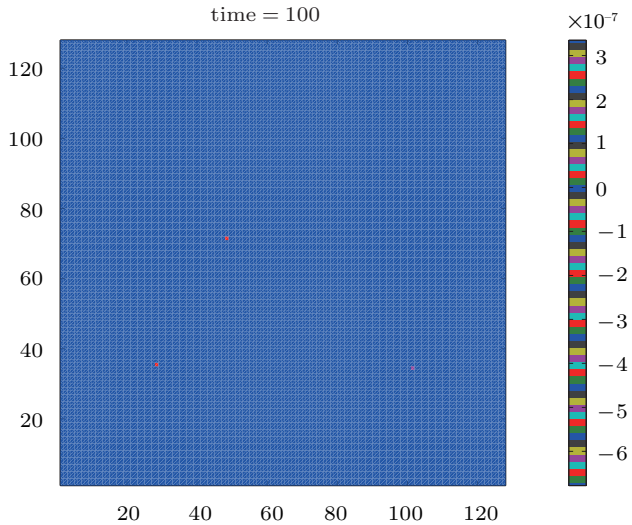


Fig. 3. (color online) The pattern formation when $k_2 = 3$, $p = 10^{-6}$, and $d_1 = 3$, $d_2 = 2$, $d_3 = 1$, $d_4 = 1$, $k_1 = 1$, $k_3 = 3$, $k_4 = 4$.

Here we denote $d_1 = 3$, $d_2 = 2$, $d_3 = 1$, $d_4 = 1$, $k_1 = 1$, $k_3 = 3$, $k_4 = 4$, and obtain the critical value $\alpha_c = (17 - k_2)/2$ from the Turing instability condition. The system (1) is stable (as shown in Fig. 5) when $k_2 = 3$ and $d_1 = 3$, $d_2 = 2$, $d_3 = 1$, $d_4 = 1$, $k_1 = 1$, $k_3 = 3$, and $k_4 = 4$ corresponding to the case that the pattern formation is homogeneous (as shown

in Fig. 3), which means cells stop oscillating and their fate has become committed to a given part of a presumptive somite.[34] The Turing instability occurs (as shown in Fig. 5) when $k_2 = 7$, $d_1 = 3$, $d_2 = 2$, $d_3 = 1$, $d_4 = 1$, $k_1 = 1$, $k_3 = 3$, and $k_4 = 4$ and the pattern formation is shown in Fig. 4 which means the activator and repressor are working with the morphogen signal diffusion, i.e., the pattern formation occurs. Comparing with Ref. [2], we can obtain the corresponding biological mechanism with the pattern formation.

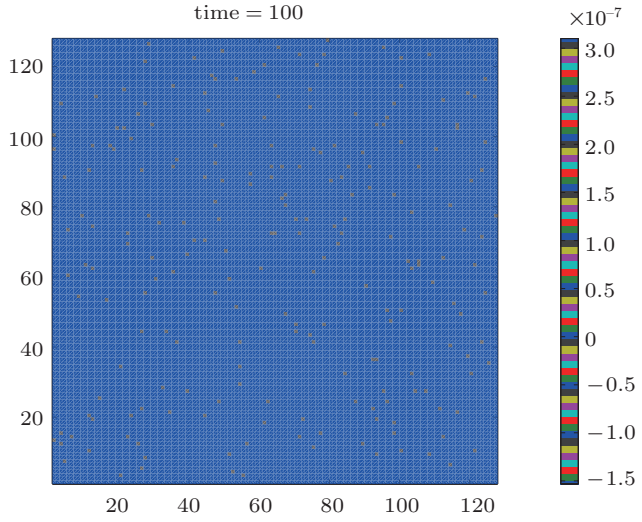


Fig. 4. (color online) Turing instability occurs when $k_2 = 7$, $p = 10^{-6}$, and $d_1 = 3$, $d_2 = 2$, $d_3 = 1$, $d_4 = 1$, $k_1 = 1$, $k_3 = 3$, $k_4 = 4$.

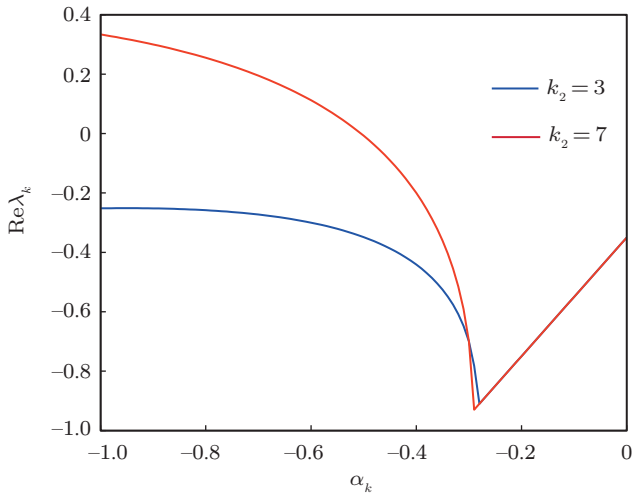


Fig. 5. (color online) The real part value with k_2, $p = 10^{-6}$, and $d_1 = 3$, $d_2 = 2$, $d_3 = 1$, $d_4 = 1$, $k_1 = 1$, $k_3 = 3$, $k_4 = 4$.

For the amplitude equation (8), which has the same biology mechanism, but it is always defined as variance.[13]) In this paper, we drive the amplitude equation and we know that it is stable (as shown in Fig. 6 when $k_2 = 2$ and $d_1 = 3$, $d_2 = 2$, $d_3 = 1$, $d_4 = 1$, $k_1 = 1$, $k_3 = 3$, and $k_4 = 4$, this corresponds to the case that the pattern formation is homogeneous (as shown in Fig. 7), and the spot pattern formation is shown in Fig. 8 when the amplitude equation is unstable (shown in Fig. 6). So the system consistently matches the stability of the system (1) and the amplitude equation (8) with the pattern formation, which also means they play an important role in investigating the reaction diffusion. In addition, the system has rich pattern formation for different probabilities p as shown in Figs. 8 and 9, indicating that the probability also plays a very important role in the mechanism of the pattern formation.

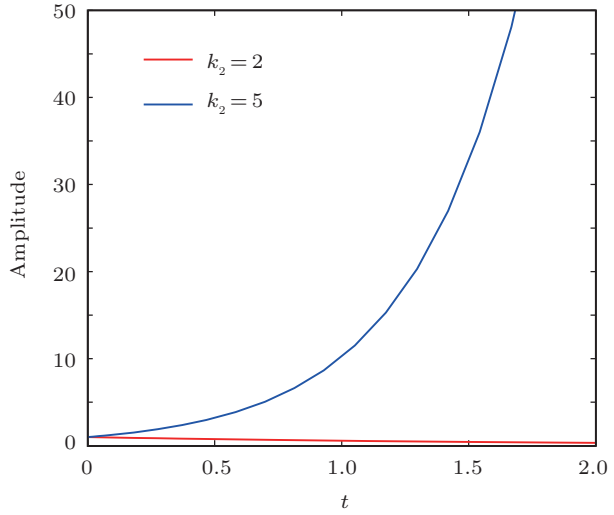


Fig. 6. (color online) The amplitude equation with k_2, $p = 10^{-6}$, and $d_1 = 3$, $d_2 = 2$, $d_3 = 1$, $d_4 = 1$, $k_1 = 1$, $k_3 = 3$, $k_4 = 4$.

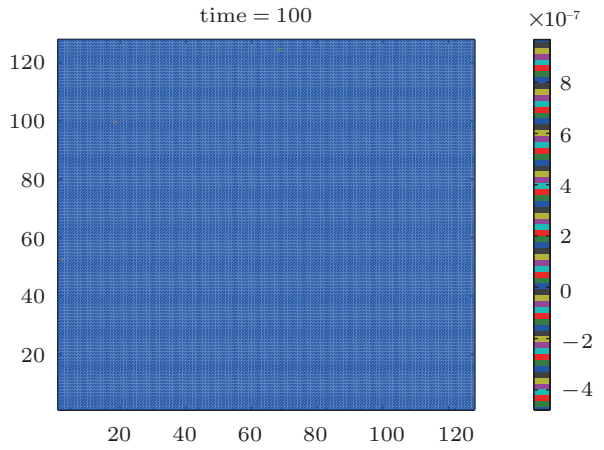


Fig. 7. (color online) The pattern formation when $k_2 = 2$, $p = 10^{-6}$, and $d_1 = 3$, $d_2 = 2$, $d_3 = 1$, $d_4 = 1$, $k_1 = 1$, $k_3 = 3$, $k_4 = 4$.

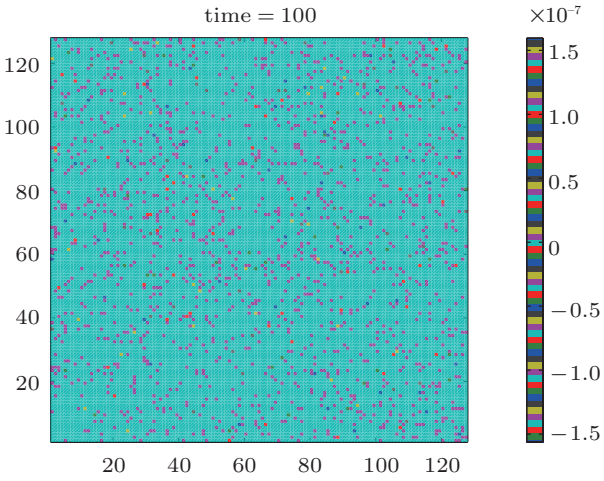


Fig. 8. (color online) The pattern formation when $k_2 = 5$, $p = 10^{-6}$, and $d_1 = 3$, $d_2 = 2$, $d_3 = 1$, $d_4 = 1$, $k_1 = 1$, $k_3 = 3$, $k_4 = 4$.

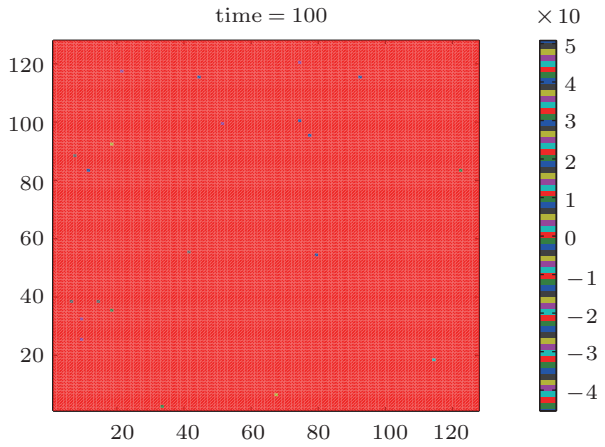


Fig. 9. (color online) The pattern formation when $k_2 = 5$, $p = 10^{-5}$, and $d_1 = 3, d_2 = 2, d_3 = 1, d_4 = 1, k_1 = 1, k_3 = 3, k_4 = 4$.

5. Conclusion

The cross diffusion is a ubiquitous phenomenon in complex networks, and it is often investigated, but for probability p was not in continuous media.[2–4] In this article, we considered a network-organized system with cross diffusion and presented a systematical analytical and numerical study of the Turing instability, developed nonlinear patterns in large random networks. Compared with Ref. [2], the biological mechanism with pattern formation was presented. It is found that the pattern formation is homogeneous (as shown in Fig. 3) when the system (1) is stable (see Fig. 5), which means cells stop oscillating and their fate has become committed to a given part of a presumptive somite.[34] And the spot pattern formation is shown in Fig. 4 when the Turing instability occurs (shown in Fig. 5, which means the activator and repressor are working with the morphogen signaling diffusion. In general, the Turing instability and the pattern formation occur in our model, but the spot patterns are very different from those found in the continuous model. We derived the amplitude equation of the cross-diffusion network-organized model, which is always defined as variance.[14] The equation provides a new tool to investigate these systems and prove the theoretical research bringing into the correspondence with our simulation. Then we explained some phenomena in embryos with our results. The amplitude equation (8) has the same biology mechanism: the pattern formation is homogeneous (shown in Fig. 7 when the amplitude equation is stable (shown in Fig. 6) and the spot pattern formation is shown in Fig. 8 when the amplitude equation is unstable. So the system consistently matches the stability of the system (1) and amplitude equation (8) with the pattern formation. These behaviors indicate that the amplitude equation plays an essential role in investigating the reaction diffusion. Finally, we found that the system has rich pattern formation behavior for different probabilities p as shown in Figs. 8 and 9, so the probability also plays a very important role in the mechanism of pattern formation.

References

[1] Turing A M 1952 *Trans. R. Soc. B* **237** 37
[2] Zheng Q Q and Shen J W 2014 *Nonlinear Dynamics* **78** 1301
[3] Wang L 2010 *Chin. Phys. B* **19** 090206
[4] Cohen S, Brennecke J and Stark A 2006 *Genes Dev.* **20** 2769
[5] Economou A D, Ohazama A, Porntaveetus T, Sharpe P T, Kondo S, Basson M A, Gritli-Linde A, Cobourne M T and Green J B 2012 *Nature Genetics* **44** 348
[6] Zemskov E P, Vanag V K and Epstein I R 2011 *Phys. Rev. E* **84** 036216
[7] Lu J, Leung H and Chen G R 2004 *Discrete and Impulsive Systems Series B: Applications and Algorithms* **11** 70
[8] McGraw P N and Menzinger M 2008 *Phys. Rev. E* **77** 031102
[9] Arenas A, Díaz-Guilerac A, Kurthsd J, Morenob Y and Zhou C S 2008 *Phys. Rep.* **469** 93
[10] Shen J W 2011 *Commun. Theor. Phys.* **55** 465
[11] Shen J W, Liu Z R, Zheng W X, Xu F D and Chen L N 2009 *Physca A* **388** 2995
[12] Othmer H G and Scriven L E 1971 *J. Theor. Biol.* **32** 507
[13] Nakao H and Mikhailov A S 2010 *Nat. Phys.* **6** 544
[14] Hata S, Nakao H and Mikhailov A S 2012 *Europhys. Lett.* **98** 64004
[15] Boccaletti S, Latorab V, Morenod Y, Chavez M and Hwangaet D U 2008 *Phys. Rep.* **424** 175
[16] Arenas A, LatoraV, MorenoY, Chavez M and Hwang D U 2008 *Phys. Rep.* **469** 3
[17] Horsthemke W, Lam K and Moore P K 2004 *Phys.Lett.A* **328** 444
[18] Moore P K 2005 *Physica D* **206** 121
[19] Brenton J, Matthew J and Mark D 2011 *Front. Comput. Neurosci.* **5** 6
[20] Gunaratne G H, Ouyang Q and Swinney H L 1994 *Phys. Rev. E* **50** 2802
[21] Daniel M B, Planchon G, Asllani M, Carletti T and Fanelli D 2015 *Stat. Mech.* **88** 222
[22] Kitano H 2004 *Nat. Rev. Genet.* **5** 826
[23] Thattai M and Oudenaarden V 2001 *Proc. Natl. Acad. Sci. USA* **98** 8614
[24] Wang B Y and Gong Y B 2015 *Chin. Phys.B* **24** 118702
[25] Strzyz P 2016 *Nat. Rev. Mol. Cell Biol.* **17** 2
[26] Waldron D 2016 *Nat. Rev. Gen.* **16** 264
[27] Chaplain M, Ptashnyk M and Sturrock M 2015 *Phys. Rev. E* **25** 1179
[28] Jansena M and Pfaffelhuberb P 2015 *Journal of Theoretical Biology* **364** 355
[29] Jahanpanah J, Rezazadeh M and Rahdar A A 2014 *Chin. Phys.B* **23** 124205
[30] Zhan M and Zou W2010 *Chin. Phys. B* **19** 100509
[31] Shi J P, Xie Z F and Little K 2011 *J. Appl. Anal. Comput.* **1** 95
[32] Kondo S and Miura T 2010 *Science* **329** 5999
[33] Li X Z, Bai Z G, Li Y, He Y F and Zhao K 2015 *Chin. Phys.B* **24** 048201
[34] James C, Alexandre R M and James S 2015 *Cell System* **1** 257