



# Pattern formation in a two-component reaction–diffusion system with delayed processes on a network

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## HIGHLIGHTS

- Time-delay in the reaction and diffusion fosters an oscillatory behavior.
- Emergence of a steady Turing pattern is still possible with two interacting species.
- The maximum delay, beyond which oscillations dominate, is computed.
- Stationary patterns cannot arise on connected networks with enough nodes.

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## ABSTRACT

Reaction–diffusion systems with time-delay defined on complex networks have been studied in the framework of the emergence of Turing instabilities. The use of the Lambert  $W$ -function allowed us to get explicit analytic conditions for the onset of patterns as a function of the main involved parameters, the time-delay, the network topology and the diffusion coefficients. Depending on these parameters, the analysis predicts whether the system will evolve towards a stationary Turing pattern or rather to a wave pattern associated to a Hopf bifurcation. The possible outcomes of the linear analysis overcome the respective limitations of the single-species case with delay, and that of the classical activator–inhibitor variant without delay. Numerical results gained from the Mimura–Murray model support the theoretical approach.

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## 1. Introduction

Self-organizing phenomena are widespread in Nature and have been studied for a long time in various domains, be it in physics, chemistry, biology, ecology, neurophysiology, to name a few [1]. Despite the rich literature on the subject, there is still need for understanding, analyzing and predicting their behaviors.

They are commonly based on local interaction rules which determine the creation and destruction of the entities at every place, upon which a diffusion process determines the migration of the components. For this reason reaction–diffusion systems are a common framework of modeling such systems [2].

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In a 1952 article in biomathematics, Turing considered a two-species model of morphogenesis [3]. For the first time, he established the conditions for a stable spatially homogeneous state, to migrate towards a new heterogeneous, spatially patched, equilibrium under the driving effect of diffusion, at odd with the idea that diffusion is a source of homogeneity. Even though the explanation for morphogenesis has evolved and now relies more on genetic programming, many actual results are based or inspired from this pioneering work. The so-called Turing instabilities help explain by a simple means the emergence of self-organized collective patterns.

The geometry of the underlying support where the reaction–diffusion acts, plays a relevant role in the patterned outcome, it can be because of the non flat geometry [4] (possibly growing) [5] or because of its anisotropy [6]. Pushing to the extreme the discreteness of the space, scholars have considered reaction–diffusion systems on complex networks; reactions occur at each node and then products are displaced across the network using the available links, thus possibly exhibiting Turing patterns [7]. Since then, the latter have been studied on other complex networks supports, for instance multiplex [8,9] and Cartesian product networks [10].

On the other hand, time-delays (also called time lags) kept making their way into more and more mathematical models. Time lags come into play in classical mechanical engineering applications, for load balancing in parallel computing, in traffic flow models and in still more fields of network theory. Indeed, they cannot be left alone in the understanding of the interactions between neurons in biology, for distributed, cooperative or remote control, in using networks of sensors. Roughly speaking, delays are inherent to virtually all systems where the time needed for transport, propagation, communication, reaction or decision making cannot be neglected [11,12].

The effects of time-delay on stability come under many flavors. In feedback systems for instance, time-delay can induce or help suppress oscillations (see Ref. [12, p. ix] and references therein). On the other hand, introducing the delay can be a very reasonable way to improve the models and avoid unnecessary or complex variants of delay-free approaches to refine the match between predictions and observations. In case of reaction–diffusion systems, this challenge was addressed by some previous work related to delay-driven irregular patterns, where Turing or Hopf bifurcations determine the evolution and the ultimate stable state of the system [13–15] on continuous domains. These studies focused on delay in the reaction kinetics, not in the diffusion part, and is some cases restricting to the small delays assumption.

In this work, the goal is to tackle a time-delay dependent problem with two profound distinctions with the above studies. First, our system evolves on a discrete domain –a network– and second, we do not limit ourselves to the case that only the reactions are delayed. That is, all the processes taking place in the nodes including the triggering of diffusion need some finite amount of time to occur, resulting in a pure-delay setting [16,17]. The retarded behavior could be due to inertia, some limiting physical, technological or human factor, correspond to the processing time, or be by design due to a wait-then-act strategy. A first step in this direction has been recently done in Ref. [18] where authors studied a reaction–diffusion system with time-delay on a top of a complex network; it has been shown that even with only one species, Turing-like traveling waves can emerge, but never stationary patterns. Observe that this result improves the classical one by Turing – i.e. reaction–diffusion without delay – for which at least two species are necessary to have patterns, so the outcome of Ref. [18] is due to the presence of the delay term in the diffusion part.

Building on the premises of the one-species case, we consider a two-component pure-delay reaction–diffusion system. Adapting the linear stability analysis to the time-delay setting and elaborating on the condition for instability by expanding the perturbation on a generalized basis formed by the eigenvectors of the network Laplacian matrix, we are able to characterize the possible onset of Turing instabilities, stationary patterns and traveling waves. To this end we use the scalar Lambert  $W$ -function which allows to cast analytical results in closed explicit form depending on the main model parameters: time-delay, diffusion coefficients and network topology. Let us observe that time-delayed systems exhibit a larger set of parameters for which Turing instabilities emerge; more precisely a time-delayed system can have Turing waves for sufficiently large time delays, while the same system without delay cannot develop stationary nor oscillatory patterns.

Hence after having determined the conditions for the emergence of patterns in terms of the time-delay, we analyze the role of the network topology, through its spectral properties, and that of the diffusion coefficients, and determine once again explicit conditions for the emergence of patterns. The analytical results are complemented and confirmed by direct numerical simulations using the prototype Mimura–Murray model.

The paper is organized as follows. In Section 2 we present the framework of the 2-species reaction–diffusion model with time delay on a complex network. Section 3 is devoted to a brief introduction of the Mimura–Murray model that will be subsequently used to check our analytical results. In Section 4 we will provide the conditions for the emergence of stationary patterns, involving the time-delay. Next, we devote Section 5 to the computation of the delay stability margin of the model, that we compare with the small time-delay approximation estimate of Section 6. Sections 7 and 8 will respectively be dedicated to the study of patterns emergence as a function of the network topology and species mobility. In the final Section 9 we will sum up and conclude.

## 2. A two-species model on a network with delayed node processes

We consider a two species activator–inhibitor reaction–diffusion system defined on an undirected network with  $n$  nodes and no self-loops. The network is described by its adjacency matrix,  $G$  and let  $k_i = \sum_{j=1}^n G_{ij}$  denote the degree of the  $i$ th node. The Laplacian matrix  $L$  of the network is defined by  $L_{ij} = G_{ij} - k_i \delta_{ij}$ . The time-dependent concentrations of the activator, respectively the inhibitor, in node  $i$  will be denoted by  $u_i(t)$ , respectively  $v_i(t)$ . The reaction of such quantities in each node is

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