

Analysis of Turing instability of the Fitzhugh-Nagumo model in complex networks

ABSTRACT

This study mainly investigates the dynamical analysis of the FitzHugh-Nagumo (FHN) neuron model. Firstly, it analyzes the equilibrium stability of the system in the absence of network diffusion. Then, it considers two types of network topologies: random networks and higher-order networks. The paper analyzes the Turing instability phenomenon in the presence of network diffusion, identifies the critical diffusion coefficient in the FHN model that leads to Turing instability, and plots the eigenvalue distribution diagram, known as the Turing pattern. The research findings indicate that networks with higher-order connections, as opposed to random networks, display a more intricate interplay among neurons. This heightened interconnection intensifies the Turing instability phenomenon, amplifying its significance within the system. The stability of the dynamical system can be associated with the onset of neurological disorders such as epilepsy, caused by abnormal neuronal firing. This analogy facilitates the transfer of content related to the instability of control systems to the regulation of neurological disorders.

Keywords: Fitzhugh-Nagumo model, Dynamic analysis, Complex networks, Turing instability.

1. Introduction

The FitzHugh-Nagumo neuron model was proposed by FitzHugh[1] and Nagumo et al. [2] which is a simplified model capturing the neural excitability of the original Hodgkin-Huxley equation. This model is commonly referred to as the FHN model. In recent years, many researchers have explored the dynamics using the FHN model[3]. Turing theory, originally proposed by Alan Turing for chemical systems, has been applied in various fields such as ecology[7], physics[9] and others. Nakao et al. [10] studied the Turing instability of activators and inhibitors in network diffusion, providing a theoretical foundation for subsequent research. Hens et al. [11] investigated the propagation of signals in complex networks. Zheng et al. [12] explored the Turing instability phenomenon in the FHN model and its relevance to short-term memory. Parker et al. [16] studied synaptic learning in the FHN model.

The study of the dynamic behavior of a single neuron model can no longer fully explain some of the phenomena encountered in biomathematics. Therefore, scholars have shifted their attention to studying the model within a network framework. The interconnection between nodes in the network signifies the transfer of information between neurons, which is more conducive to studying the dynamic analysis of the model. In recent years, higher-order networks have emerged, considering the interaction of three or even more nodes based on the original interactions between two nodes. This approach is more beneficial for exploring neurological diseases caused by abnormal electrical firing of neurons. Bianconi et al. explored the relevance of higher-order networks in [17], while review [19] provides a detailed introduction to higher-order network. Gao et al. [20] adopted a combination of three models to study Turing instability in a simplicial complex. Indeed, there are higher-order relationships beyond two nodes, and group interactions commonly occur in the neurobiology [21], ecology [22].

In [23], the authors investigated the correlation between epileptic seizures and kinetic behaviors. Hence, the dynamical phenomena of the FHN model are examined in random networks and higher-order networks. The section 2 entails an analysis of the single neuron model, followed by an examination of the FHN model under network diffusion in section 3,

where we derive relevant expressions concerning the diffusion coefficient. Finally, section 4 concludes and offers prospects for further research.

2. Dynamic analysis with FHN model without diffusive network

In this paper, we investigate the Fitzhugh-Nagumo (FHN) model, characterized by the following equations[1]:

$$\begin{cases} f(u, v) = u(1-u)(u+a) - v, \\ g(u, v) = bu - cv. \end{cases} \quad (1)$$

We assume that the parameters a, b, c are positive. Here, u represents the voltage of the membrane, while v denotes the recovery variable. Equation (1) implies that the equilibrium point is represented by (u^*, v^*) , where $v^* = bu^*/c$. The specific equilibrium point is

$$\begin{aligned} E_1 &= \left(u_1, \frac{bu_1}{c} \right), u_1 = \frac{-ac + c + \sqrt{a^2c^2 + 2ac^2 - 4bc + c^2}}{2c}. \\ E_2 &= \left(u_2, \frac{bu_2}{c} \right), u_2 = \frac{-ac + c - \sqrt{a^2c^2 + 2ac^2 - 4bc + c^2}}{2c}. \\ E_3 &= (0, 0). \end{aligned}$$

We have the Jacobian matrix in the equilibrium point:

$$J = \begin{pmatrix} f_u & f_v \\ g_u & g_v \end{pmatrix} = \begin{pmatrix} -3u^{*2} + 2(1-a)u^* + a & -1 \\ b & -c \end{pmatrix}.$$

Hence, the characteristic equation for Equation (1) is obtained as follows:

$$\lambda^2 - \lambda \text{tr} J + \det J = 0. \quad (2)$$

And

$$\begin{aligned} \text{tr} J &= a - c - 3u^{*2} + 2(1-a)u^*, \\ \det J &= c(3u^{*2} - 2(1-a)u^* - a) + b. \end{aligned}$$

By applying the Routh-Hurwitz criterion and the stability theorem, we can determine the range within which the equilibrium point of the system exhibits asymptotic stability. Initially, we study the FHN model without a diffusive network.

Through analyzing the Jacobian matrix corresponding to the equilibrium point in the system, we can obtain the stability analysis of the neuron model:

Theorem 1. When we have $b < ac$, the $E_3 = (0, 0)$ is saddle node.

Proof: By substituting the equilibrium point value into the Jacobian matrix, we derive the following expression.

$$J_{E_3} = \begin{pmatrix} a & -1 \\ b & -c \end{pmatrix}.$$

And we have the E_3 is saddle node when we have $\det J_{E_3} = b - ac < 0$, consequently, theorem 1 has been satisfied. \square

The stable states of the other two equilibrium points can then be calculated.

3 Dynamic analysis with FHN model in diffusive network

Neural networks are thought to be necessary for information transmission and integration. In this section, we give the instability analysis of network systems. In Eq (3), the expression of $f(u, v)$ and $g(u, v)$ equals to Eq(1), d_1 and d_2 represent the diffusion coefficient of u and v , respectively.

$$\begin{cases} \frac{du}{dt} = f(u, v) + d_1 \sum_{j=1}^N L_{ij} u, \\ \frac{dv}{dt} = g(u, v) + d_2 \sum_{j=1}^N L_{ij} v. \end{cases} \quad (3)$$

L is the Laplacian matrix in the network, the matrix element is computed as:

$$L_{ij} = A_{ij} - k_i \delta_{ij}, \delta_{ij} = \begin{cases} 1, & i = j, \\ 0, & i \neq j. \end{cases}$$

A is the adjacent matrix in the network, k_i is the degree of node i . δ_{ij} represent the Kronecker product, once node i and node j are connected, we have $\delta_{ij} = 1$. The characteristic equation for the Laplace matrix is:

$$\sum_{\alpha=1}^N L_{ij} \Phi_j^{(\alpha)} = \Lambda_{\alpha} \Phi_i^{(\alpha)}, \text{ with } \alpha = 1 \cdots N. \quad (4)$$

Where $\Phi^{(\alpha)}$ is the Laplacian eigenvector and Λ_{α} is the eigenvalue. Therefore, we have the characteristic equation of Eq(3) is:

$$\lambda_{\alpha}^2 - \lambda_{\alpha} \cdot P(\Lambda_{\alpha}) + Q(\Lambda_{\alpha}) = 0. \quad (5)$$

And

$$\begin{aligned} P(\Lambda_{\alpha}) &= f_u + g_v + \Lambda_{\alpha} (d_1 + d_2), \\ Q(\Lambda_{\alpha}) &= d_1 d_2 \Lambda_{\alpha}^2 + \Lambda_{\alpha} (d_1 g_v + d_2 f_u) + (f_u g_v - f_v g_u). \end{aligned}$$

The solution to this Eq (5) is

$$\begin{aligned} \lambda_{\alpha 1} &= \frac{1}{2} \left[P(\Lambda_{\alpha}) - \sqrt{P(\Lambda_{\alpha})^2 - 4 \cdot Q(\Lambda_{\alpha})} \right], \\ \lambda_{\alpha 2} &= \frac{1}{2} \left[P(\Lambda_{\alpha}) + \sqrt{P(\Lambda_{\alpha})^2 - 4 \cdot Q(\Lambda_{\alpha})} \right]. \end{aligned}$$

There is a critical value when Turing bifurcation occurs in the neuronal model: $Q(\Lambda_{\alpha}) = 0$, Turing instability occurs when the following conditions are satisfied: $\Lambda_{\alpha} \in (\Lambda_{\alpha 1}, \Lambda_{\alpha 2})$. And we have

$$\begin{aligned} \Lambda_{\alpha 1} &= \frac{-(d_1 g_v + d_2 f_u) - \sqrt{(d_1 g_v + d_2 f_u)^2 - 4 d_1 d_2 (f_u g_v - f_v g_u)}}{2 d_1 d_2}, \\ \Lambda_{\alpha 2} &= \frac{-(d_1 g_v + d_2 f_u) + \sqrt{(d_1 g_v + d_2 f_u)^2 - 4 d_1 d_2 (f_u g_v - f_v g_u)}}{2 d_1 d_2}. \end{aligned}$$

Therefore, we have the following theorem.

Theorem2. The diffusion coefficient $k = d_2/d_1$ critical condition of Turing bifurcation is:

$$k f_u - c = 2 \sqrt{k(b - c f_u)}. \quad (6)$$

And $f_u = -3u^{*2} + 2(1-a)u^{*} + a$.

Proof: When Turing bifurcation is happened, we have $\text{Re } \lambda_{\alpha} > 0$, saddle-node bifurcation occurs in the system, as $Q(\Lambda_{\alpha}) < 0$. When we take $\Lambda_{\alpha} = -\frac{d_1 g_v + d_2 f_u}{2 d_1 d_2}$, we have:

$$Q(\Lambda_{\alpha})_{\min} = (f_u g_v - f_v g_u) - \frac{(d_1 g_v + d_2 f_u)^2}{4 d_1 d_2}.$$

So, we have $Q(\Lambda_{\alpha})_{\min} < 0$ in the Eq (5), which simplifies to Eq (6). \square

4. Simulation

In this part, we simulate the FHN model in a certain parameter. We set $a = 0.8, b = 0.5, c = 0.7$ in the Eq(1), we have $E_1 = (0.4094, 0.2924)$, $E_2 = (-0.2094, -0.0558)$, $E_3 = (0, 0)$. When we take the initial value $(u, v) = (0.01, 0.01)$. In Fig 1, we show the diagram of the equilibrium point of the Eq (1), the curve containing an arrow in the image represents a vector field. Therefore, Figure 1 shows that this is a bistable system, E_3 is an unstable equilibrium point, E_1 and E_2 are stable equilibrium point.

Then, we show phase diagram and time series diagram of the Eq (1) in the Fig 2, as time goes by, u and v tend to stabilize and remain unchanged after a small oscillation amplitude. The equilibrium point remains stable in E_1 .

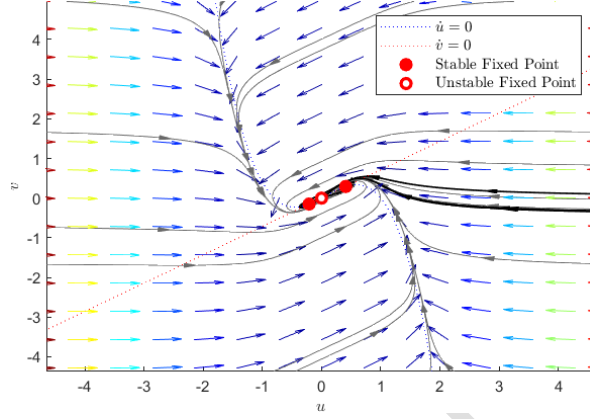


Fig1. Equilibrium point state of Eq(1).

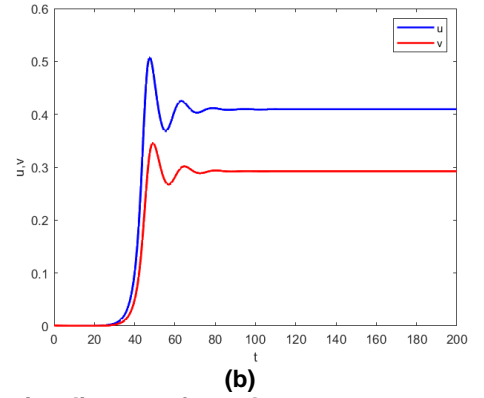
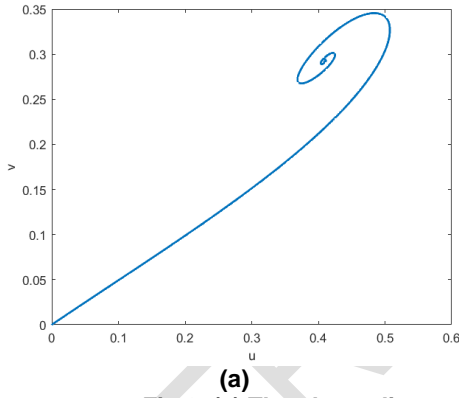


Fig 2. (a) The phase diagram in Eq(1). (b) The time series diagram of u and v .

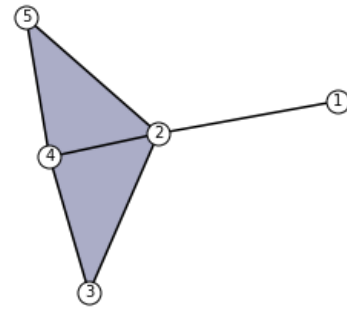
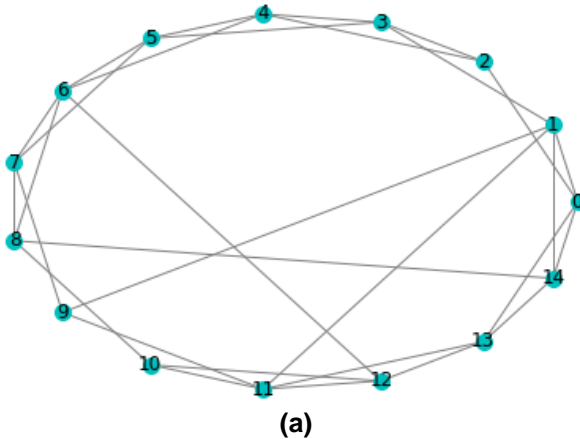


Fig 3. Two types of network structure diagram.
(a) Random network. (b) Higher-order network.

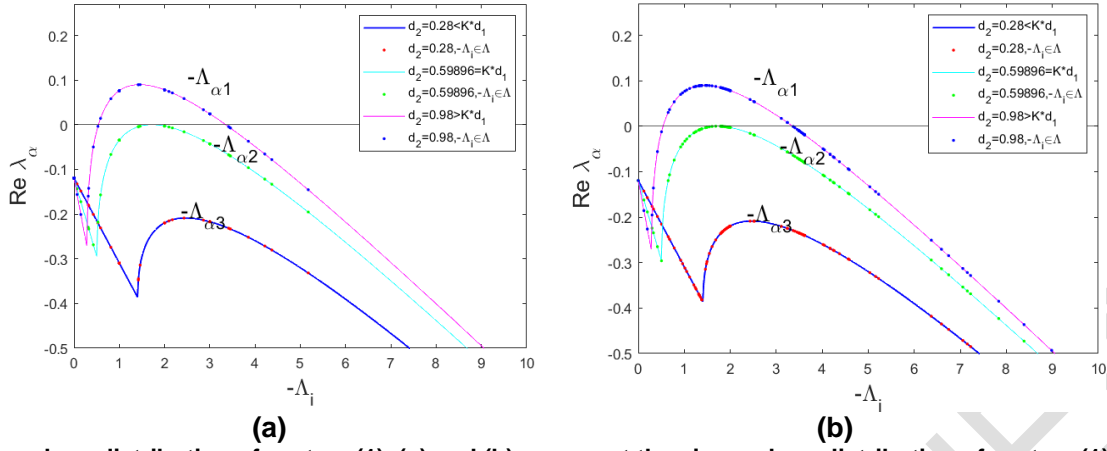


Fig 4. The eigenvalues distribution of system (1). (a) and (b) represent the eigenvalues distribution of system (1) in the random network and higher-order network, respectively.

In Figure 3, we show the topology of random networks and higher-order networks. The Fig3(a) represent the random network with $N=10$, which indicates that nodes are randomly connected with a certain probability. The Fig3(b) shows the higher-order network with $N=5$, which means the interconnection between multiple nodes. In the higher-order network, a node is a 0-simplex, a link is a 1-simplex, a triangle is a 2-simplex^[17], therefore, we have five 0-simplices(nodes), six links(1-simplices) and two triangles(2-simplices)^[17].

Figures 4(a) and 4(b) represent the eigenvalue distribution of system (1) in the random network and higher-order network, respectively. The curves in the figure represent the eigenvalue distribution of system (1), while the scatter points represent the eigenvalues in the constructed network. The zero axis is utilized to distinguish whether Turing instability occurs. Scatter points situated above the zero axis indicate the occurrence of Turing instability, whereas the curve below the zero axis indicates that the system is stable. By comparing Figure 4(a) and Figure 4(b), it can be observed that the phenomenon of Turing instability is more obvious in the higher-order network than in the random network.

5. Conclusion

In this paper, we discuss the dynamics analysis of the FHN model, considering the phase diagram and time series diagram of a single neuron model, and also considering the Turing instability of the system in the presence of network diffusion.

We explored changes in equilibrium point stability, analyzing the network topology structure. The research indicates that the Turing instability phenomenon becomes more pronounced as the network's topology becomes more complex and the connections between nodes become more compact.

The transmission of information in the neuronal model is not only related to the time delay, but also to the electromagnetic field stimulus and synapse. Therefore, in future research, we will further study the dynamic behavior of the external stimulus and its impact on the network model.

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