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Dynamical Robustness in Synaptically Coupled Neuronal Networks

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Abstract—Tolerance of biological networks against local perturbations is still not completely understood, because both structure and dynamics are often complex in such networks. Here we study the role of synaptic connections in robustness of dynamic activities in neuronal network models. We show that the dynamical robustness varies depending on the strength and the number of the synaptic connections. We also demonstrate that homogeneous networks are more tolerant than heterogeneous networks from the dynamical robustness viewpoint. This case study would contribute to understanding robustness of biological networks.

1. Introduction

Robustness is a fundamental feature of biological systems. For instance, homeostasis is the property of a system that regulates its internal environment and tends to maintain stable, constant conditions. Immunity is also a mechanism through which living things are able to be resistant to viruses and pathogens from the environment outside the body. However, some perturbations to biological systems often cause a fatal damage to them. For instance, brain infarct can result from a series of biochemical reactions initiated by ischemia (inadequate blood supply) in a local part. Therefore, biological systems are robust against some perturbations but vulnerable to other ones. A mathematical framework to understand this "robust but fragile" property in biological systems is still not fully established [1, 2].

Biological systems function in networks of diverse biological components, which are interacting with each other in various scales. Recent developments of imaging modalities and electrical devices have enabled to reveal connectivity properties in biological networks, including metabolic [3], protein [4], cellular [5], and brain networks [6]. The structures of these biological networks are quite complex and heterogeneous. Accordingly, complex topological structures of biological systems are necessary to be incorporated in mathematical models for understanding the robustness of biological systems [7].

Although the robustness of network structure has attracted much attention in complex network theory [8], less attention has been paid to the robustness of dynamics on networks. For many real networks consisting of components having intrinsic dynamics, it is reasonable to take into account both complex structure and dynamics. In particular, dynamical activities are responsible for information transmission and other normal functions in biological networks. Recently we have studied the robustness of dynamical activities in oscillator networks with complex topologies [9] by extending the framework which was first proposed for globally coupled oscillator networks [10] and subsequently applied to other networks [11, 12]. This study has shown that scale-free networks can be extremely fragile to inactivation of low-degree oscillators if there are dynamical processes where normal components compensate for failure components. Because it is widely recognized that scale-free networks are highly fragile to removal of high-degree nodes (hubs), our result on the property on dynamical robustness in oscillator networks is in strong contrast to the property on structural robustness. It is suggested that the property of dynamical robustness can depend on the type of dynamics of individual components and the interaction scheme between components.

In neuronal networks, neurons are coupled via synapses. Our aim is to understand the effect of synaptic connections on the robustness of firing activities in neuronal networks by using a mathematical model. The Morris-Lecar neuron model [13] is used as the components of neuronal networks. We assume that some neurons in the network are inactivated and become unable to generate a spike by themselves. As the ratio p of the inactivated neurons increases, the neuronal firing activities are gradually weakened. When p surpasses a critical value p_c, the firing activity in the whole network is lost due to a phase transition phenomenon. Therefore, a larger value of p_c implies a more dynamically robust network. By using this robustness measure, we investigate the dynamical robustness in synaptically coupled neuronal networks.

2. Methods

2.1. Neuronal network model

Spiking neuron models can be classified into two types according to the bifurcation mechanism of the onset of spike firing with an increase of the external input current: class I excitability (saddle-node bifurcation on invariant

circle bifurcation) and class II excitability (subcritical Hopf bifurcation) [14]. The Morris-Lecar neuron model [13] is one of the favorable conductance-based spiking neuron model widely used in computational neuroscience, which can reproduce both types of bifurcations depending on the parameter values [15]. The model that we study is a network of Morris-Lecar neuron models coupled via synapses. The model equations are described as follows [16]:

$$C_{\mathrm{M}}\frac{dV_i}{dt} = I_i^{ext} - I_i^{ion} - I_i^{syn}, \quad (1)$$

$$\frac{dW_i}{dt} = (W_\infty(V_i) - W_i)/\tau_W, \quad (2)$$

where V_i and W_i represent the membrane potential and the recovery variable (the fraction of open potassium channels) of the ith neuron ($i = 1, \ldots, N$), respectively. C_{M} represents the membrane capacitance per unit area, τ_W is a parameter controlling the time scale of the firing dynamics (or the inverse of the decay rate of W_i), I_i^{ext} is the external input current, I_i^{sym} is the synaptic current, and I_i^{ion} is the ionic current. The ionic current is given by

$$I_i^{ion} = g_{\mathrm{L}}(V_i - V_{\mathrm{L}}) + g_{\mathrm{Ca}}M_\infty(V_i)(V_i - V_{\mathrm{Ca}}) + g_{\mathrm{K}}W_i(V_i - V_{\mathrm{K}}), \quad (3)$$

where g_{L}, g_{Ca}, and g_{K} represent the conductances of the calcium, potassium, and leak currents, respectively. V_{L}, V_{Ca}, and V_{K} denote the resting potentials for the calcium, potassium, and leak currents, respectively. The calcium current is assumed to be in equilibrium with the following activation curve:

$$M_\infty(V) = (1 + \tanh((V - V_1)/V_2))/2, \quad (4)$$

where V_1 is the midpoint potential at which the calcium current is half-activated and V_2 corresponds to the steepness of the activation voltage dependence. The potassium activation is given by a voltage-dependent function as follows:

$$W_\infty(V) = (1 + \tanh((V - V_3)/V_4))/2, \quad (5)$$

where V_3 is the midpoint potential at which the potassium current is half-activated and V_4 corresponds to the slope of the potassium activation. The time constant τ_W is also assumed to be voltage-dependent as follows:

$$\tau_W = 1/(\phi \cosh((V - V_3)/2V_4)). \quad (6)$$

The single Morris-Lecar neuron which is not synaptically connected to other neurons (i.e. $I_i^{syn} = 0$) exhibits an onset of firing activity as the external input current I_i^{ext} increases. The normal neuron which is responsible for signal transmission shows spiking behavior as demonstrated in the upper panel of Fig. 1. The stationary behavior corresponds to a limit cycle oscillation. Suppose that the neuron has inactivated due to failure or deterioration. Then, the firing activity of the neuron is not observed as shown in the lower panel of Fig. 1. The inactivated neuron is excitable: it can generate spikes when an input current is injected.

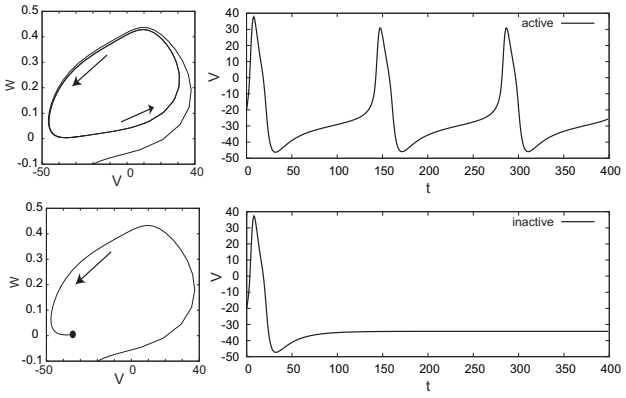

Figure 1: Behavior of the single Morris-Lecar neuron model. The left panels show the state space and the right panels show the time series of the membrane potential V. The normal active neuron (upper) exhibits repetitive spike firings corresponding to a limit cycle, whereas the inactivated neuron (lower) is excitable but not self-oscillatory. These two regimes are separated by a saddle-node on invariant circle bifurcation.

2.2. Synaptic connections

In a network of these normal and inactivated neurons, the inactivated neurons can generate spikes due to the synaptic inputs injected from neighboring normal neurons. Neurons typically have two types of synapses: electrical and chemical synapses. In electrical gap-junctions, the sypaptic current is proportional to the membrane potential difference between a neuron and its neighbors as follows:

$$I_i^{syn} = \sum_{j \in N_i} g_{ij}^{gap}(V_i - V_j), \quad (7)$$

where N_i stands for the set of the neighboring neurons coupled with neuron i and g_{ij}^{gap} represents the conductance of the synaptic channel.

2.3. Measure of network dynamics

To evaluate firing activities in the neuronal networks, we define the order parameter as follows:

$$R = \sqrt{\langle(\mathbf{x}_c - \langle\mathbf{x}_c\rangle)^2\rangle}, \quad (8)$$

where $\mathbf{x}_c = N^{-1}\sum_{j=1}^{N}(V_j(t), W_j(t))$ is the centroid and the brackets mean a long time average. The firing frequency is also related to the order parameter. A transition from a dynamic state to a quiescent state can be characterized by a change of R from positive to 0.

2.4. Simulation setting

We consider a network of N Morris-Lecar neurons coupled via gap-junctions. The proportion of the inactivated neurons is given by p and that of the normal neurons by

Table 1: Parameter values of the Morris-Lecar neurons [15]

Parameter	Value
C_M	20 μF/cm^2
g_K	8 mS/cm^2
g_{Ca}	2 mS/cm^2
g_L	2 mS/cm^2
V_K	-80 mV
V_{Ca}	120 mV
V_L	-60 mV
V_1	-1.2 mV
V_2	18 mV
ϕ	1/15 s^{-1}
V_3	12 mV
V_4	17.4 mV

$1-p$. As p increases, the order parameter decreases with lowering of the level of network dynamics. In numerical simulations, if the order parameter becomes smaller than 10^{-6}, the network state was regarded to be quiescent. The parameter values are set as shown in Table. 1 unless otherwise noted. The single Morris-Lecar neuron exhibits class-I excitability for these parameter conditions. The strength of the gap-junctions is assumed to be homogeneous: $g_{ij}^{gap} = g_{gap}$. To examine the effect of the network topology on the dynamical robustness, we compare dynamical robustness in homogeneous random [17] and heterogeneous scale-free networks [18] with the mean degree fixed. The mean number of gap-junctions for each neuron is represented by K_{gap}.

3. Results

We numerically study dynamical robustness in neuronal networks coupled via electrical gap-junctions. We assume that the neurons in the network are randomly inactivated with ratio p. Namely, the number of normal active neurons is $(1-p)N$ and that of inactivated neurons is pN. When there is no inactive neurons (i.e. $p = 0$), all the neurons exhibit synchronized spiking behavior. On the other hand, when all the neurons are inactivated (i.e. $p = 1$), the dynamic behavior in the whole network vanishes. Hence, at a critical value of $p = p_c$, a phase transition between the two different regimes must take place. This critical value can be a measure of dynamical robustness of the network, because a larger value of p_c means that the network is more tolerant to the components failure.

Figure 2 shows the critical value against the strength of the gap-junction in random networks with three different values of the mean degree. As the connection strength increases, the network becomes less robust because of a decrease in the value of p_c. For a sufficiently large connection strength, the value of p_c converges to a certain value independently of the mean degree. The result also shows

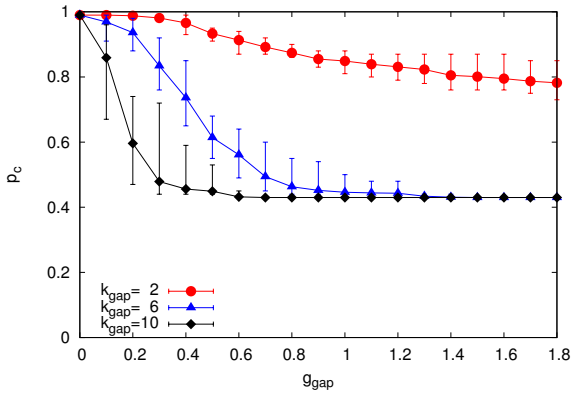

Figure 2: Critical ratio p_c vs the strength g_{gap} of the electrical synapses in random networks with $N = 200$. For each parameter we conducted 10 trials with different network configurations and initial conditions.

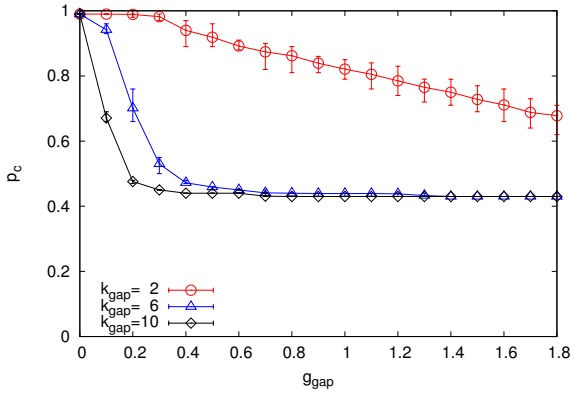

Figure 3: Critical ratio p_c vs the strength g_{gap} of the electrical synapses in scale-free networks with $N = 200$. For each parameter we conducted 10 trials with different network configurations and initial conditions.

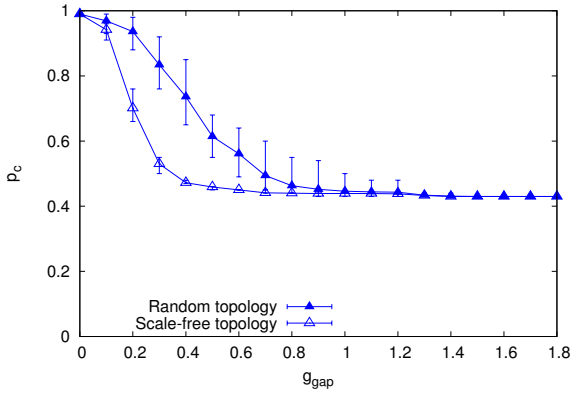

Figure 4: Effet of network topology on the critical ratio p_c. The p_c value is larger for random networks than for scale-free networks. Both are the networks with $N = 200$ and $K_{gap} = 6$.

that the network with denser connections is less robust in terms of dynamics. Similar tendency can be confirmed in the scale-free networks as shown in Fig. 3. However, the decay rate of the value of p_c seems to depend on the network topology.

Figure 4 compares the curves of p_c in random and scale-free networks with the same mean degree $K_{gap} = 6$. It is clearly observed that the value of p_c is larger in random networks than in scale-free networks. In other words, the random networks are more tolerant to random inactivation of neurons than the scale-free networks in terms of dynamical robustness. This is opposite to the well-known property that scale-free networks are highly robust to random failure of components compared with random networks in terms of structural robustness [19].

4. Summary

Robustness of dynamic activity in synaptically coupled neuronal networks has been studied in terms of phase transition phenomena. We have shown that an increase in the connection strength and the number of the electrical synapses makes the network dynamics less robust. The comparison between random and scale-free networks with the same mean degree has revealed that, surprisingly, the firing dynamics in homogeneous networks is more tolerant to the random inactivation of the neurons than that in heterogeneous networks. Theoretical verification of this remarkable property is an issue to be done. A future work is to examine the role of chemical synapses in the dynamical robustness of neuronal networks.

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