

Synchronization and chimera states in a multilayer neuronal network with unidirectional interlayer links

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Abstract. The brain consists of many interconnected systems and sub-systems that have unidirectional or bidirectional connectivities. In this paper, we consider a two-layer neuronal network with unidirectional interlayer connections to investigate the effects of one layer on the other one. Two one-dimensional ring networks with non-local couplings construct the two-layer network. The couplings are considered to be linear to describe the electrical synapses. At first, the independent layer, which is not affected by the other layer, is analysed. Different patterns such as synchronizations, non-stationary chimera state and imperfect synchronization are observed in such solo network. Then the two-layer network is investigated by varying three coupling strengths of intra-layer and inter-layer links. The results show that the first layer can change the behaviour of neurons in the second layer according to the values of coupling strengths. The interaction of the interlayer and intralayer couplings can also induce the emergence of imperfect chimera state, which was not observed in the monolayer network. These self-organized phenomena can have strong relations with brain functions and malfunctions.

1 Introduction

The nervous system is a complex network, composed of billions of neurons. The neurons interact with each other and transfer the information from the brain to organs and vice versa. The communications between the neurons occur mainly through two different synapses: chemical and electrical synapse. At the synapses, the information from one neuron is transferred to another one. If the information transmission happens through release of neurotransmitters, it is called a chemical synapse and if the cytoplasm of adjacent cells are directly connected, it is called an electrical synapse or gap junction [1]. The electrical synapses have been observed in the retina, inferior

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olive, olfactory bulb, and several regions in the mammalian central nervous system [1]. Depending on the characteristics of the channel connexins, the gap junctions can be either unidirectional or bidirectional [2].

Different self-organizing phenomena, such as synchronization, have been observed in neuronal networks [3–6]. Neuronal synchronization, which is considered as a collective behaviour of many neurons, has been a challenging issue in neuroscience for decades [7]. Synchronization can be related to efficient processing, information transmission and signal coding, in the brain [8]. Moreover, many brain disorders are associated an enhancement or a decrement in synchronization. For example, epilepsy is known as a functional brain disorder, which has strong relations with synchronization. In fact, before or at the early stages of the epilepsy, desynchronization is observed, while high levels of synchronization appear at the end of the seizures [9]. Another brain disorder concerning synchronization is Alzheimer’s disease (AD). Comparing the electroencephalogram (EEG) signals of a group of AD patient with those from the control group revealed a considerable reduction in the synchronization of the AD group [10]. Attention deficit hyperactivity disorder (ADHD) is a common childhood behavioural disorder. The experimental EEG recordings of ADHD patients have demonstrated high synchronization in the alpha and beta bands [11].

Chimera patterns are another self-organized phenomenon having strong correspondences with neuronal functions [12]. These patterns are characterized by the coexistence of synchronized and de-synchronized domains in a network [13–17]. The unihemispheric sleep in some animals has the most association with the chimera state [18]. During this sleep, one hemisphere of the brain is slept and its neurons oscillate almost synchronously, while the other hemisphere is awake and its neurons oscillate asynchronously. It has also been reported that some brain disorders, such as Parkinson’s disease, Alzheimer’s disease, Epileptic seizures, etc., can be related to chimera states [18]. Therefore, chimera states are being widely investigated in neuronal networks [19–22].

Recently, the concept of multilayer networks has attracted much attention [23–26]. Multilayer networks are able to describe the interconnected systems through multiple connections [27]. The nervous system consists of multiple subsystems and layers of connectivity. Thus employing a multilayer network can have the benefits of considering the effects of subsystems on each other. In this paper we consider a two-layer neuronal network, consisting of two rings of non-locally coupled Hindmarsh–Rose neurons. All the communications between the neurons are through gap junctions. We aim to investigate the effects of one layer on the emerging patterns of the second layer. Thus, the two layers are connected with unidirectional links.

The paper is organized as follows. In the next section the mathematical equations of the network are described. Section 3 analyses the main results of the numerical simulations. The conclusions are given in Section 4.

2 The network model

Each neuron of the network is described by the Hindmarsh–Rose neuron model, given by:

$$\begin{aligned}\dot{x} &= y + bx^2 - ax^3 - z + I \\ \dot{y} &= c - dx^2 - y \\ \dot{z} &= r[s(x + 1.6) - z]\end{aligned}\tag{1}$$

where the variables x, y, z , represent the membrane potential of the neuron, fast recovery variable and slow adaption variable respectively. I is the external forcing

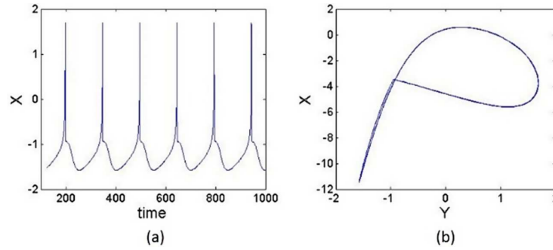


Fig. 1. (a) The membrane voltage of the neuron of model (1), when $I = 1.5$. (b) the attractor of the neuron of model (1) in $(x-y)$ plane. The neuron shows spiking behaviour at defined parameters.

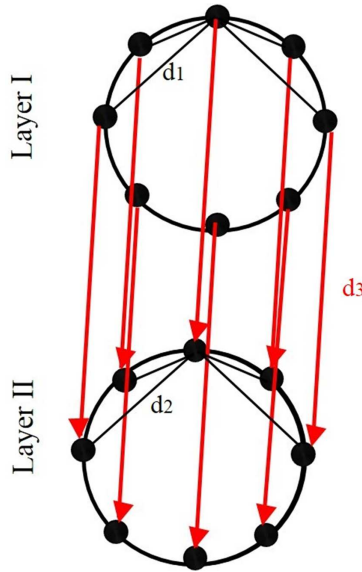


Fig. 2. The schematic of the two-layer network. The black lines show intra-layer non-local links and the red lines show unidirectional inter-layer links.

current. We fix the parameters at $a = 1, b = 3, c = 1, d = 5, r = 0.006, s = 4$ and $I = 1.5$. Therefore, a single neuron shows periodic spiking as shown in Figure 1.

We construct a two-layer network of the above-mentioned neuron models. Each layer of the network is a one-dimensional ring, consisting of non-locally coupled neurons via electrical synapses. Two layers are coupled with unidirectional coupling connections in a way that only the second layer is influenced by the first layer. Figure 2 shows a schematic of this network. Thus the equations of the first layer are described by:

$$\begin{aligned} \dot{x}_{1i} &= y_{1i} + bx_{1i}^2 - ax_{1i}^3 - z_{1i} + I + d_1 \sum_{j=i-P}^{i+P} [x_{1j} - x_{1i}] \\ \dot{y}_{1i} &= c - dx_{1i}^2 - y_{1i} \\ \dot{z}_{1i} &= r [s(x_{1i} + 1.6) - z_{1i}] \end{aligned} \tag{2}$$

and the equations of the second layer are described by:

$$\begin{aligned}\dot{x}_{2i} &= y_{2i} + bx_{2i}^2 - ax_{2i}^3 - z_{2i} + I + d_2 \sum_{j=i-P}^{i+P} [x_{2j} - x_{2i}] + d_3(x_{1i} - x_{2i}) \\ \dot{y}_{2i} &= c - dx_{2i}^2 - y_{2i} \\ \dot{z}_{2i} &= r[s(x_{2i} + 1.6) - z_{2i}]\end{aligned}\quad (3)$$

where i is the index of neuron, $i = 1, \dots, N$, N is the number of neurons in each layer, P is the number of neurons at each side of the non-local coupling. The parameters are fixed at $N = 100$, $P = 20$. The strengths of the first intra-layer, second intra-layer and the inter-layer couplings are represented by d_1 , d_2 and d_3 respectively.

3 Numerical results

In this section, the main results of the numerical solutions of the network are presented. As discussed before, both of the layers are considered to be the same and the first layer is not influenced by the second one. Therefore, for the first step we investigate the patterns of the first layer ring network by varying its coupling strength. Then the two-layer network is considered and the effect of the first layer on the second layer behaviour is investigated via unidirectional electrical couplings.

3.1 A single layer network

Firstly, we consider the network given in equation (2), and vary the coupling strength d_1 . Figure 3 shows the different spatiotemporal patterns observed from the network, by increasing the coupling strength. When the coupling strength is between 0.001 and 0.004, all the neuron's of the network spike synchronously as shown in Figure 3a. For $d > 0.004$, the neurons behaviour is changed to bursts. In this case the pattern of the network seems to be the chimera state, but the synchronous and asynchronous neurons are not stationary at times. This state is called non-stationary chimera. Figure 3b shows the non-stationary chimera for $d = 0.005$. The non-stationary states are almost observed until $d = 0.009$. By increasing the coupling strength to $d = 0.01$, depending on the initial conditions, two cases of imperfect synchronization or burst synchronization can be observed. Figure 3c shows the imperfect synchronization for $d = 0.01$. In this case, although most of the neurons are spiking synchronously, a few of them exhibit chaotic bursting. Figure 3d shows the burst synchronization state in which the neurons start to burst synchronously, for $d = 0.01$ and with different initial conditions with Figure 3c. Finally, when the coupling strength gets higher values, all the neurons become synchronous, exhibiting synchronous spikes as shown in Figure 3e.

3.2 Two-layer network

If the inter-layer coupling strength is equal to zero, then the second layer will have the same behaviours as the first layer, as described in the previous subsection. By increasing the interlayer coupling strength, we consider the effects of the first layer on the second one. To this aim, the values of the intra-layers coupling strengths are set at 0.001, 0.005, 0.01, 0.02, their dynamics were described in the previous subsection, and then the inter-layer coupling strength is varied.

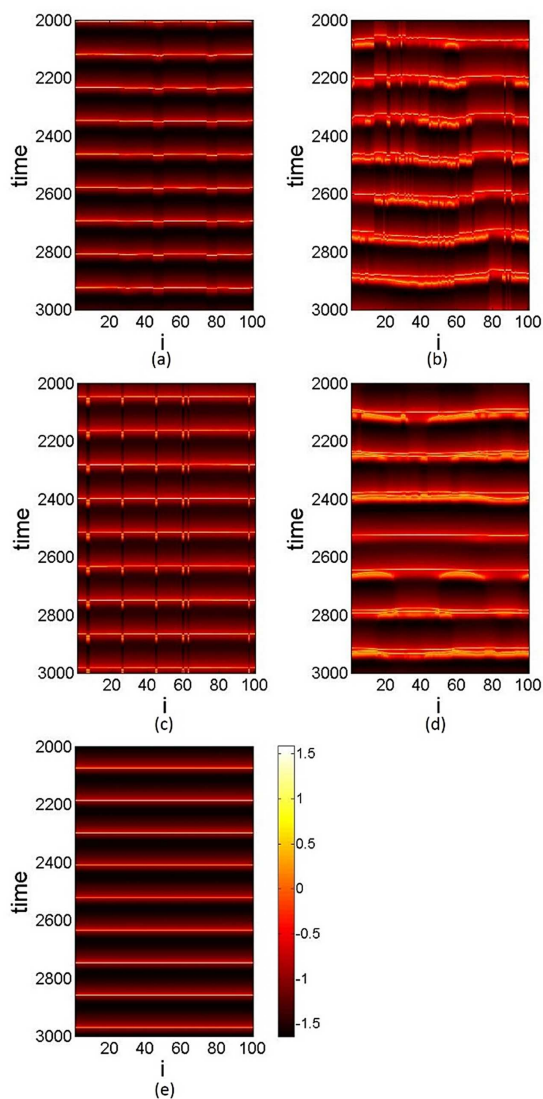


Fig. 3. Different spatiotemporal patterns of the network of equation (2) by increasing the coupling strength. (a) $d_1 = 0.003$: synchronization, (b) $d_1 = 0.005$: non-stationary chimera state, (c) $d_1 = 0.01$: imperfect synchronization, (d) $d_1 = 0.01$: burst synchronization with different initial conditions, (e) $d_1 = 0.02$: synchronization. Increasing the coupling strength causes transitions from synchronization to chimera state and again to synchronization. It also can change the spiking patterns of neurons to bursts.

The obtained results show that when $d_3 < 0.01$, there is no considerable change in the second-layer patterns, in comparison to the absence of inter-layer coupling. As the inter-layer coupling raises from $d_3 = 0.01$, the pattern of the second layer is affected by the first layer.

At first, we consider $d_2 = 0.001$. By setting this, the second layer neurons spike synchronously in the case of no inter-layer coupling. However when $d_3 = 0.05$ and $d_1 = 0.005$, the second layer state changes to non-stationary chimera as shown in Figure 4a. If d_2 is increased to $d_2 = 0.005$, by setting $d_1 = 0.02$ and $d_3 = 0.05$, the

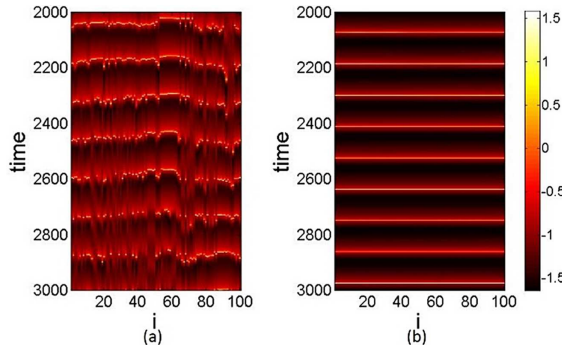


Fig. 4. The spatiotemporal patterns of layer II, (a) $d_1 = 0.005, d_2 = 0.001, d_3 = 0.05$: non-stationary chimera state. (b) $d_1 = 0.02, d_2 = 0.005, d_3 = 0.05$: synchronization. The pattern of the second layer changes according to the first-layer behaviour due to the unidirectional links.

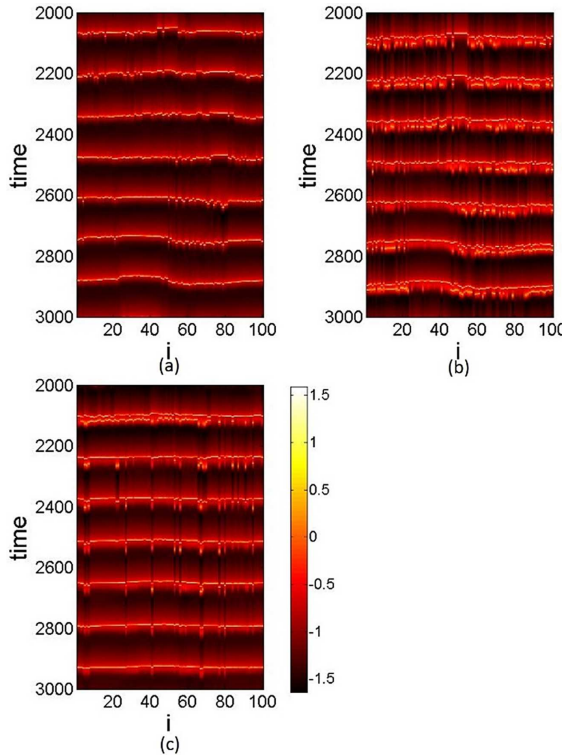


Fig. 5. The spatiotemporal patterns of layer II, (a) $d_1 = 0.005, d_2 = 0.001, d_3 = 0.1$: non-stationary chimera state. (b) $d_1 = 0.005, d_2 = 0.004, d_3 = 0.1$: non-stationary chimera state. (c) $d_1 = 0.01, d_2 = 0.004, d_3 = 0.1$: imperfect chimera state. The pattern of the second-layer changes according to the first-layer behaviour due to the unidirectional links.

second layer neurons spike synchronously instead of showing non-stationary chimera (Fig. 4b).

If the value of d_3 increases, the first layer can cause more different patterns in layer II. Figure 5 shows some of the results of $d_3 = 0.1$ on the second layer pattern. If $d_2 = 0.001$, in the case of no inter-layer coupling, the neurons should spike synchronously,

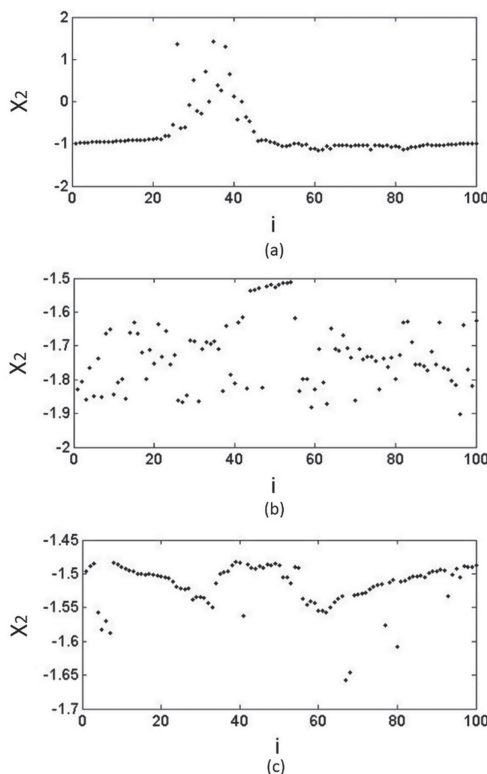


Fig. 6. The snapshots of the membrane potentials of neurons in layer II, corresponding to the states of Figure 5. (a) $d_1 = 0.005, d_2 = 0.001, d_3 = 0.1$: non-stationary chimera state. (b) $d_1 = 0.005, d_2 = 0.004, d_3 = 0.1$: non-stationary chimera state. (c) $d_1 = 0.01, d_2 = 0.004, d_3 = 0.1$: imperfect chimera state.

but with setting $d_1 = 0.005$ and $d_3 = 0.1$, although the neurons are still spiking, the layer pattern changes to non-stationary chimera, as shown in Figure 5a. Even by increasing d_2 , the first layer still affects the second one. Figure 5b shows the pattern of layer II for $d_2 = 0.004$, in which the neuron’s behaviour is changed to bursting and the layer exhibits non-stationary chimera. Increasing the d_1 value makes the second layer to be more similar to the first layer. For example, if d_1 is set at 0.01, the second-layer pattern shows imperfect chimera. In this case there are some bursting neurons within the synchronous spiking group (Fig. 5c). The imperfect chimera is the result of inter-layer coupling and was not observed in the first layer. Figure 6 shows the snapshot of the membrane potentials of the second-layer neurons, corresponding to the states shown in Figure 5.

In order to have a comprehensive comparison between the observed states in different coupling strengths, some obtained results are presented in Tables 1–3.

4 Conclusion

In this paper, a two-layer neuronal network was studied. Each layer of the network was composed of one-dimensional ring of non-locally coupled Hindmarsh–Rose neurons. The brain consists of many interconnected subsystems that can affect each other unidirectionally or bidirectionally. Here, the two layers were connected via unidirectional links to study the effect of one subsystem on the other one. All of the

Table 1. The observed states in the second layer for $d_3 = 0.01$. (S S: spike synchronization, B S: burst synchronization, NS Ch: non-stationary chimera, Imp S: imperfect synchronization, As: asynchronization).

d_2	d_1			
	0.01	0.05	0.001	0.002
0.001	S S	S S	S S	S S
0.005	NS Ch	NS Ch	NS Ch	NS Ch
0.01	S S	S S	Imp S	Imp S
0.02	S S	S S	S S	S S

Table 2. The observed states in the second layer for $d_3 = 0.05$. (Abbreviations as in Tab. 1).

d_2	d_1			
	0.001	0.005	0.01	0.02
0.001	S S	NS Ch	As	S S
0.005	NS Ch	NS Ch	NS Ch	S S
0.01	S S	B S	B S	S S
0.02	S S	B S	S S	S S

Table 3. The observed states in the second layer for $d_3 = 0.1$. (Abbreviations as in Tab. 1).

d_2	d_1			
	0.001	0.005	0.01	0.02
0.001	S S	NS Ch	S S	S S
0.005	NS Ch	NS Ch	NS Ch	NS Ch
0.01	NS Ch	B S	B S	S S
0.02	S S	B S	B S	S S

couplings were defined by a linear function, to describe the electrical synapses. At first, we analysed the ring network of the first layer, which was not influenced by the second layer. The strength of the coupling was varied and different emerging patterns, such as synchronization, non-stationary chimera, imperfect synchronization and burst synchronization, were observed. Since the two layer structures were the same, if there were no interlayer connections, the second layer would behave similar to the first one. It was observed that the interlayer coupling changes the pattern of the second layer, according to the first layer behaviour. Actually, the emerging pattern in layer II, was the result of interaction of all three coupling strengths. Even this interaction caused an emergence of imperfect chimera, which was not observed in the one-dimensional ring network. These emerging patterns can have strong relations with the brain disorders. For example, the increasing or decreasing in the strength of synapses can act as some disturbances and cause stronger or weaker effects between the subsystems. As a result, the synchronization in special parts may be enhanced or weakened and initiate a disorder.

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Author contribution statement

All the authors designed research, performed research, and wrote the paper. Xiaodong Li and Tao Xu contributed equally to the paper.

References

1. A.E. Pereda, *Nat. Rev. Neurosci.* **15**, 250 (2014)
2. A. Pereda, D.S. Faber, *Front. Mol. Neurosci.* **11**, 427 (2018)
3. M. Dhamala, V.K. Jirsa, M. Ding, *Phys. Rev. Lett.* **92**, 074104 (2004)
4. Y. Hao, Y. Gong, L. Wang, X. Ma, C. Yang, *Chaos Solitons Fractals* **44**, 260 (2011)
5. X. Sun, J. Lei, M. Perc, J. Kurths, G. Chen, *Chaos* **21**, 016110 (2011)
6. J. Tang, J. Ma, M. Yi, H. Xia, X. Yang, *Phys. Rev. E* **83**, 046207 (2011)
7. T.I. Netoff, M.I. Banks, A.D. Dorval, C.D. Acker, J.S. Haas, N. Kopell, J.A. White, *J. Neurophysiol.* **93**, 1197 (2005)
8. I. Belykh, E. de Lange, M. Hasler, *Phys. Rev. Lett.* **94**, 188101 (2005)
9. P. Jiruska, M. De Curtis, J.G. Jefferys, C.A. Schevon, S.J. Schiff, K. Schindler, *J. Physiol.* **591**, 787 (2013)
10. M.G. Knyazeva, C. Carmeli, A. Khadivi, J. Ghika, R. Meuli, R.S. Frackowiak, *Neurobiol. Aging* **34**, 694 (2013)
11. T. Liu, P. Lin, Y. Chen, J. Wang, *Bio-med. Mater. Eng.* **24**, 1035 (2014)
12. S. Majhi, M. Perc, D. Ghosh, *Sci. Rep.* **6**, 39033 (2016)
13. D.M. Abrams, S.H. Strogatz, *Phys. Rev. Lett.* **93**, 174102 (2004)
14. D. Dudkowski, Y. Maistrenko, T. Kapitaniak, *Phys. Rev. E* **90**, 032920 (2014)
15. J. Hizanidis, N.E. Kouvaris, Z.-L. Gorka, A. Diaz-Guilera, C.G. Antonopoulos, *Sci. Rep.* **6**, 19845 (2016)
16. P. Jaros, Y. Maistrenko, T. Kapitaniak, *Phys. Rev. E* **91**, 022907 (2015)
17. F.P. Kemeth, S.W. Haugland, L. Schmidt, I.G. Kevrekidis, K. Krischer, *Chaos* **26**, 094815 (2016)
18. S. Majhi, M. Perc, D. Ghosh, *Chaos* **27**, 073109 (2017)
19. A. Mishra, S. Saha, D. Ghosh, G.V. Osipov, S.K. Dana, *Opera Med. Physiol.* **3**, 14 (2017)
20. A. Schmidt, T. Kasimatis, J. Hizanidis, A. Provata, P. Hövel, *Phys. Rev. E* **95**, 032224 (2017)
21. Z. Wei, F. Parastesh, H. Azarnoush, S. Jafari, D. Ghosh, M. Perc, M. Slavinec, *Europhys. Lett.* **123**, 48003 (2018)
22. Z.-M. Wu, H.-Y. Cheng, Y. Feng, H.-H. Li, Q.-L. Dai, J.-Z. Yang, *Front. Phys.* **13**, 130503 (2018)
23. F. Battiston, V. Nicosia, V. Latora, *Phys. Rev. E* **89**, 032804 (2014)
24. S. Boccaletti, G. Bianconi, R. Criado, C.I. Del Genio, J. Gómez-Gardenes, M. Romance, I. Sendina-Nadal, Z. Wang, M. Zanin, *Phys. Rep.* **544**, 1 (2014)
25. S. Gomez, A. Diaz-Guilera, J. Gomez-Gardenes, C.J. Perez-Vicente, Y. Moreno, A. Arenas, *Phys. Rev. Lett.* **110**, 028701 (2013)
26. X. Zhang, S. Boccaletti, S. Guan, Z. Liu, *Phys. Rev. Lett.* **114**, 038701 (2015)
27. M. Kivelä, A. Arenas, M. Barthelemy, J.P. Gleeson, Y. Moreno, M.A. Porter, *J. Complex Networks* **2**, 203 (2014)