Eur. Phys. J. B (2019) 92: 36
https://doi.org/10.1140/epjb/e2018-90638-x

THE EUROPEAN PHYSICAL JOURNAL B

Regular Article

Effects of partial time delays on synchronization patterns in Izhikevich neuronal networks

Mohadeseh Shafiei[1], Fatemeh Parastesh[1], Mahdi Jalili[2], Sajad Jafari[1], Matjaž Perc[3,4,a], and Mitja Slavinec[3]

[1] Biomedical Engineering Department, Amirkabir University of Technology, Tehran 15875-4413, Iran
[2] School of Engineering, RMIT University, Melbourne, Australia
[3] Faculty of Natural Sciences and Mathematics, University of Maribor, Koroška cesta 160, 2000 Maribor, Slovenia
[4] Complexity Science Hub Vienna, Josefstädterstraße 39, 1080 Vienna, Austria

Received 31 October 2018 / Received in final form 10 December 2018
Published online 13 February 2019

Abstract. Synchronization patterns have been observed in neuronal networks and are related to many cognitive functions and information processing and even some pathological brain states. In this paper, we study a ring network of non-locally coupled Izhikevich neurons with electrical synaptic coupling. Since it has been proved that time delays through gap junctions can simplify the synchronization, here we particularly investigate the effects of partial time delays on networks synchronization. By using two control parameters, the time delay and the probability of partial time delay, we show that partial time delays have a significant effect on the synchronization of this network. In particular, partial time delays can either increase or decrease the synchronization and also can induce synchronization transitions between coherent and incoherent states. Thus, partial time delays can cause chimera state, which is a special pattern when both synchronous and asynchronous states coexist and are strongly related to many real phenomena. Furthermore, partial time delays can change the period of synchronized neurons from period-1 to period-2 firing states that have different effects on information transmission in the brain.

1 Introduction

Study of synchronization in coupled networks is important in different fields such as physics and biology [1,2]. It has been frequently reported that synchronization of neuronal networks have a major role in functionality (or malfunction) of the brain [3,4]. Synchronization is a fundamental neural mechanism and has a subtle role in efficient processing and neuronal signal transmission and coding [5–7]. Many synchronization patterns have been identified in coupled systems, such as complete synchronization, phase and antiphase synchronization, phase-lock synchronization, cluster synchronization, and lag synchronization [8,9]. The rhythm of electrical activity of neurons carries more important information than the amplitude. Thus, phase synchronization, which is associated with neural integration and working memory, is usually more visible than complete synchronization in biological systems [5,10]. If perturbations occur in the synapses, the dynamics of the oscillations in the neurons and their periods change. Measuring the phase shifts of the neurons, which is defined as phase resetting curves, provides the information for predicting synchronization in neuronal networks [11,12].

Recently, cluster (or partial) synchronization has been studied [13–15]. Partial synchronization occurs when some parts of the network oscillate coherently as a cluster or group, while some others oscillate incoherently [16–19]. In a network of identical oscillators, chimera state represents the spatial coexistence of coherent and incoherent regions [20–24]. Studying chimera state in networks has attracted much attention within the research community [19,25–28]. Observations in unihemispheric sleep in birds and dolphins indicate that chimera exists in natural phenomena. It is also observed in some brain disorders such as epileptic seizures [3,29].

Many studies have investigated synchronization and chimera states in neuronal networks in the past years. Ma et al. [30] considered a neuronal network with imposed electromagnetic radiation and found that the synchronization transitions depend on the coupling strength together with the intensity of electromagnetic radiation. Rakshit et al. [31] presented a complete analysis of two coupled neuronal Rulkov maps and studied synchronization in the presence of both inner linking and chemical synapses. Volos et al. [32] investigated two coupled Hindmarsh–Rose models in the case of bidirectional and unidirectional couplings and showed that it can give rise to either complete synchronization or anti-synchronization. Wei et al. [33] represented that a neuronal network of Hindmarsh–Rose

[a] e-mail: matjaz.perc@gmail.com

models with alternating current, which exhibits coexistence of limit cycle and chaotic attractors, can evoke non-stationary chimera states. Schmidt et al. [34] studied two-dimensional (2D) networks of FitzHugh–Nagumo and leaky integrate-and-fire oscillators. They observed 2D chimera patterns such as spots, rings and stripes, and reported the effects of varying the initial conditions and coupling parameters on these states. Omelchenko et al. [35] investigated nonlocally coupled Fitzhugh–Nagumo models in both regular ring topologies with inhomogeneous parameters and irregular topologies with identical oscillators, and indicated that chimera states are robust under small inhomogeneities.

Studies have shown that synchronization can be strongly affected by the time delay [36–38]. In neuronal networks, time delay has a crucial effect on synchronization, firing dynamics and spiking rate of neurons. Substantial features of the neuronal system such as increasing or decreasing in phase synchronization, switching between different synchronized states or spiking rate variation of neurons can be controlled by the time delay [5,39–41]. Masoliver et al. [42] showed that in a ring time delayed coupled network of FitzHugh–Nagumo oscillators, in the presence of noise, the number of nearest neighbors and time delay can control the coherence resonance. Jalili showed that in a time delayed chemical coupled network of Hindmarsh–Rose neurons, the time delay in both excitatory and inhibitory chemical connections may increase the phase synchronization [43]. He also showed that in Hindmarsh–Rose delayed-coupled neuronal network, synchronization of the network is different in the case of distributed or uniform time-delays [44]. It has also been investigated that when chimera state occurs, the values of significant parameters of the system can be controlled by time-delayed feedback [45]. Gjurchinovski et al. [46] demonstrated that type of the delay such as constant, time-varying or distributed can be a control factor for amplitude chimera lifetime in a nonlocal ring network of Stuart–Landau elements.

In real neuronal networks, some communication links might have time delay, while some other might be instantaneous, i.e. transmitting the signals without any delay [5]. In this paper, we study the effects of partial time delays on synchronization patterns in coupled Izhikevich neuron model oscillators. There are two control parameters, the time delay τ, and the probability P_d of existing the time delay in the link. It is illustrated that partial time delay has substantial influence on synchronization of the considered network.

2 System dynamics

In this paper, Izhikevich neuron models are considered to be connected on a ring network. This model is described as follows,

$$\begin{aligned} &\dot{v} = 0.04v^2 + 5v + 140 - u + I \\ &\dot{u} = a(bv - u) \\ &\text{if } v \geq 30[mv], \text{then } \begin{cases} v \leftarrow c \\ u \leftarrow u + d. \end{cases} \end{aligned} \tag{1}$$

Here, v and u represent the membrane potential of a neuron and the membrane recovery variable, respectively [47]. We extend equation (1) using a weak periodic signal I_e as follows:

$$\dot{v} = 0.04v^2 + 5v + 140 - u + I + I_e \tag{2}$$

in which we adopt $I_e = I_m \sin(2ft)$ with $I_m = 0.01$ and $f = 0.5$. Note that the sinusoidal signal is merely utilized as a typical example of a signal in a neuronal system. The mathematical equations of network are presented by:

$$\begin{aligned} &\dot{v_i} = 0.04v_i^2 + 5v_i + 140 - u_i + I \\ &\qquad +g\sum_{j=1}^{N} A_{(i,j)}(v_j(t-\tau) - v_i(t)) \\ &\dot{u_i} = a(bv_i - u_i) \\ &\text{if } v_i \geq 30[mv_i], \text{then } \begin{cases} v_i \leftarrow c \\ u_i \leftarrow u_i + d \end{cases} \end{aligned} \tag{3}$$

where the subscript i represents the ith neuron in the network with $i = 1, 2, \ldots, N$ with N is the total number of neurons in the network. $g\sum_{j=1}^{N} A_{(i,j)}(v_j(t-\tau) - v_i(t))$ is the coupling term with g is the coupling strength and $A = (A_{(i,j)})$ is the overall coupling matrix with $A_{(i,j)} = 1$ if the ith neuron is connected to the jth neuron and $A_{(i,j)} = 0$ otherwise. In this paper, we consider the connection network as a ring graph in which each node is connected to its $k = 4$ nearest neighbors. $\tau_{(i,j)}$ is the time delay that takes nonzero values with probability P_d.

3 Phase synchronization measure

The timing of the single spikes, i.e. spike trains, have some information about neuronal communications. In order to quantify the degree of phase synchronization, we use the parameter R that describes the collective behavior among multivariate spike trains [43]. The parameter R is determined as,

$$R = \tfrac{1}{N} \left| \sum_{j=1}^{N} \exp(i\varphi_j(t)) \right| \tag{4}$$

$\varphi_j(t)$ is the phase for the jth neuron at the time t and is defined as:

$$\begin{aligned} \varphi_j(t) &= 2\pi \tfrac{t - t_{j,k}}{t_{j,k+1} - t_{j,k}} \\ j &= 1, \ldots, N \end{aligned} \tag{5}$$

where $t_{(j,k)}$ is the time at which the kth spike of the jth neuron starts. Zero R shows no synchronization and $R = 1$ indicates full synchronization [5].

4 Results

In the following, the results of changing partial time delay on synchronization of the network are presented. To do this, influences of τ on synchronization patterns for different P_d are studied. We set constant parameters as $a = 0.1$, $b = 0.2$, $c = -65$, $d = 10$ and $I = 10$ at which a single neuron exhibits periodic behavior. The numerical results are

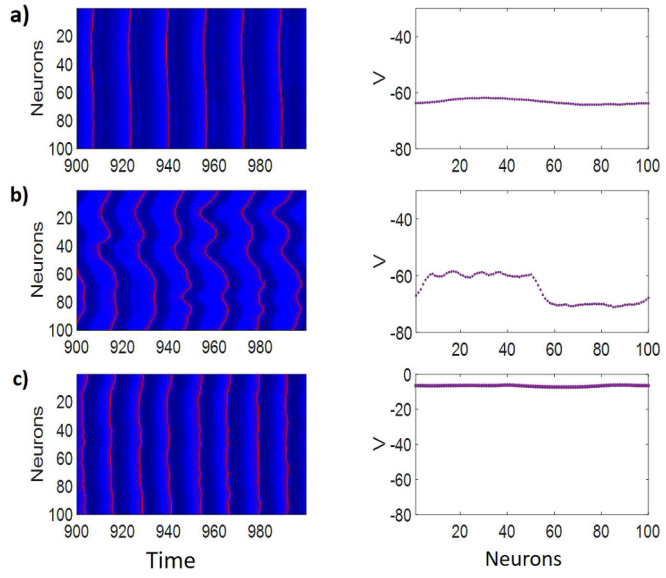

Fig. 1. Spatiotemporal patterns and snapshots at $t = 1000$ of the network with $P_d = 0.3$. (a) Zero-lag synchronization for $\tau = 2.5$, (b) distorted sine-like wave for $\tau = 7$ and (c) phase synchronization for $\tau = 10$. According to the figure, time delays even with small probability influence on the network synchronization pattern. It is observed that increasing time delay changes synchronization pattern from an ordered state to the distorted sine-like wave and then to phase synchronization.

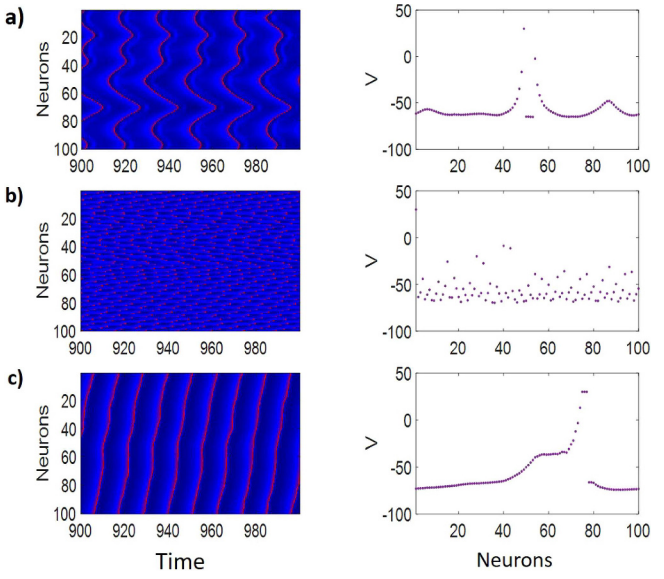

Fig. 2. Spatiotemporal patterns and snapshots at $t = 1000$ of the network with $P_d = 0.7$. (a) Imperfect distorted sine-like wave for $\tau = 2.5$, (b) asynchronization for $\tau = 7$ and (c) imperfect phase synchronization for $\tau = 10$. Figure indicates that at this probability value, delay can change the network pattern from synchronous to asynchronous patterns or intermediate states. It is also observed that higher probability value increases the disorders in the network.

obtained by using 4th order Runge–Kutta method with time step = 0.1.

The spatiotemporal patterns of spiking activity of network for different values of τ and at $P_d = 0.3$, $P_d = 0.7$ and $P_d = 1$ are presented in Figures 1–3, respectively. Due to using probability, the network has been solved repeatedly for each parameter values and the same patterns were observed. In Figure 1, we have studied a situation in which a small part of the network has been delayed with $P_d = 0.3$. This figure indicates that τ induces an exchange of different spatiotemporal patterns alternately. In detail, the spatiotemporal pattern is ordered when $\tau = 2.5$, in this case, all neurons in the network fire and come to rest, simultaneously. As one can see in the snapshot at a particular moment $t = 1000$, all neurons have the same membrane voltage level (see Fig. 1a). Actually, the network is zero-lag phase synchronized when connections are delayed with relatively small τ. When τ increases to $\tau = 7$, the ordered spatiotemporal pattern becomes rather disordered (see Fig. 1b). In this case, the neurons of the network fire by specific phase difference with their neighboring neurons. This type of firing of the neurons in the network, which is affected by the quantity of the latency, creates a special pattern which is called the distorted sine-like wave [33]. Snapshot at $t = 1000$ also suggests that the membrane voltage of the neurons is not the same at a particular moment and this specific pattern is due to the nonzero phase difference in the firing of the neurons. As τ increases to larger values of $\tau = 10$, synchronization pattern of network becomes ordered again (see Fig. 1c). Therefore, it can be seen that for a network with a small percentage of latency $P_d = 0.3$, for large network latencies $\tau = 10$, it is again moving toward phase synchronization. Figure 1 indicates that only a small part of delayed connections with $P_d = 0.3$ can have a significant effect on synchronization pattern of neuronal network. The time delay is a control factor that changes the synchronization pattern to different patterns by increasing or decreasing the latency of the network pattern.

Let consider the network with more delayed connections with $P_d = 0.7$. The results are shown in Figure 2. When $\tau = 2.5$, the observed pattern is imperfect distorted sine-like wave (see Fig. 2a). In this case, the neurons of the network fire with a certain phase difference with their neighboring neurons. Snapshots at $t = 1000$ confirm that the neurons have been shaped like a sinewave pattern. As one can see in the snapshot, although most of neurons have negative membrane voltage, a few neurons have different and positive membrane voltage. Therefore, we call it imperfect distorted sine-like wave. When τ increases to $\tau = 7$, the ordered spatiotemporal pattern becomes disordered (see Fig. 2b). The network is almost in its most disordered pattern. In this case, each neuron fires asynchronously with irregular phase differences with its neighboring neurons. Hence, the observed spatiotemporal pattern and snapshot are disordered. Further increase of time delay to larger values makes the spatiotemporal patterns of network become rather ordered. Figure 2c shows the network pattern for $\tau = 10$. In this regard, snapshot at $t = 1000$ shows that few neurons, in contrast to other

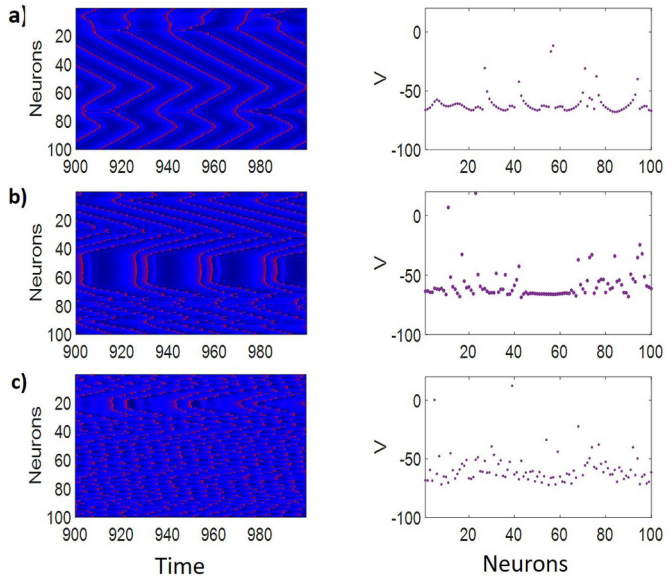

Fig. 3. Spatiotemporal patterns and snapshots at $t = 1000$ of the network with $P_d = 1$. We see in (a) imperfect phase synchronization for $\tau = 1$, (b) chimera states for $\tau = 2.5$ and (c) asynchronization for $\tau = 5$. According to the figure when all network connections are delayed, the delay factor can cause different phenomena like chimeras, in addition to changing the network synchronization pattern from synchronous to asynchronous ones.

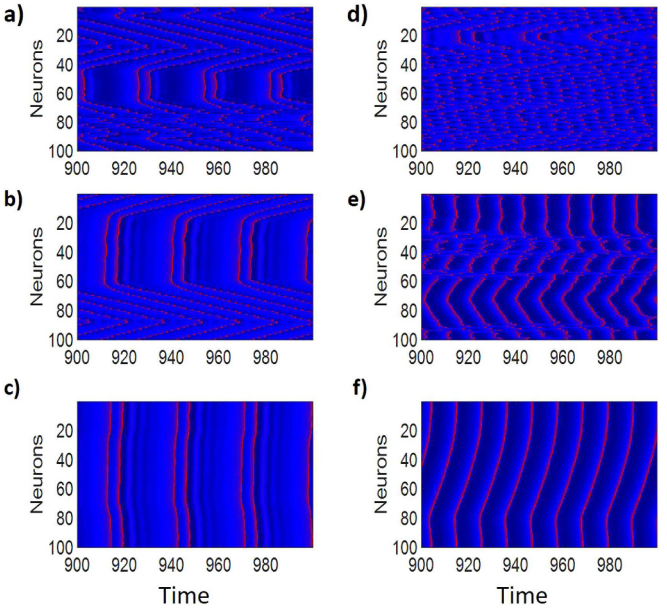

Fig. 4. Spatiotemporal patterns of the network with $P_d = 1$. We see in (a) chimera state for $\tau = 2.5$, (b) two clusters of phase synchronization for $\tau = 3.4$, (c) synchronized period-2 firings states for $\tau = 3.5$, (d) incoherent for $\tau = 5$, (e) chimera states for $\tau = 8.5$ and (f) phase synchronization for $\tau = 9.8$. Figure shows that in the case of full time delay, the network is capable of exhibiting various states in different time delays. However, there is no linear relevance between time delay and the level of regularity in the network.

neurons, have none negative membrane voltage at specific moment and abruptly break the continuous snapshot pattern and get positive voltage values, so we call it the imperfect phase synchronized pattern.

The results for the case of full time delay that all connections have delay ($P_d = 1$) are presented in Figure 3. When $\tau = 1$, due to the presence of a few neurons with positive voltage level among other neurons that are at the negative voltage level with phase difference, the observed pattern is imperfect phase synchronization. The spatiotemporal pattern shows that neurons tend to be ordered with a certain phase difference with their neighboring neurons. Also, the snapshot indicates the existence of phase difference in the membrane voltage of the neurons (see Fig. 3a). When τ increases to $\tau = 2.5$, the ordered spatiotemporal pattern becomes semi-disordered (see Fig. 3b). In this case, a coexistence of incoherent and coherent regions which is called chimera state is seen. The snapshot also indicates the presence of a group of zero-lag synchronous neurons with the same voltage level at the specific moment $t = 1000$, among the other asynchronous neurons. As τ increases to $\tau = 5$ (see Fig. 3c), synchronization regions of network become weaker and chimera state disappears and only incoherent regions are observed.

Figure 4 indicates full time delay network in wider delay range for better demonstrating the transitions. Chimera state in Figure 4a changes to a synchronization pattern of clusters of phase lag and zero lag regions in Figure 4b when τ changes from 2.5 to 3.4. An interesting phenomenon occurs here that the increasing of delay can induce the neuronal network transmitting from a period-1 firing state to a synchronized period-2 firing state (see Fig. 4c). Spike trains for $\tau = 0$, $\tau = 3.5$ and $\tau = 6$ are shown in Figure 5, respectively. This figure clearly shows the transitions between period-1 firing and period-2 firing or different period-1 firing patterns. There is a notable point that by increasing delay, the spatiotemporal pattern recovers to an ordered state (see Fig. 4c) and phase synchronization appears. With further increase of delay, the regularity of the spatiotemporal patterns become worse (see Fig. 4d) but then recover again and chimera states appear again (see Fig. 4e). Finally, the spatiotemporal pattern becomes ordered to cluster of zero lag and phase lag regions (see Fig. 4f).

In order to quantify the effect of partial time delay, we calculate the dependence of the order parameter R on delay at $P_d = 0.3$, $P_d = 0.7$ and $P_d = 1$ and the results are presented in Figure 6. An interesting point is that there is no direct relationship between increasing or decreasing latency with the order parameter R. Figure 6a shows that when $P_d = 0.3$, as delay increases, R increases and decreases alternately. The diagram shows that in general, for delay values up to $\tau = 4$, although the synchronous parameter increases or decreases, but it is near $R = 1$ and it can be said that the network is relatively synchronous. But with increasing this delay, for intermediate values of less than $\tau = 8$, we encounter a decrease in the level of the order parameter, which is typically shown in Figure 1b, for $\tau = 7$. Finally, the increase in delay up to $\tau = 10$, like low

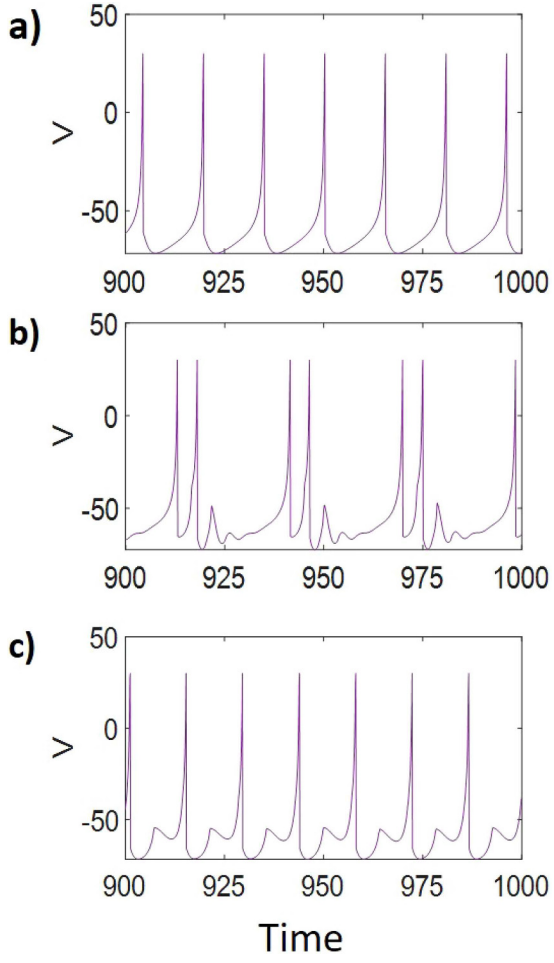

Fig. 5. Spike trains of a randomly chosen neuron when (a) $\tau = 0$, (b) $\tau = 3.5$ and (c) $\tau = 6$. According to the figure, the delay factor causes a change in dynamics of the firing neurons and transitions between period-1 and period-2 or different period-1 firings.

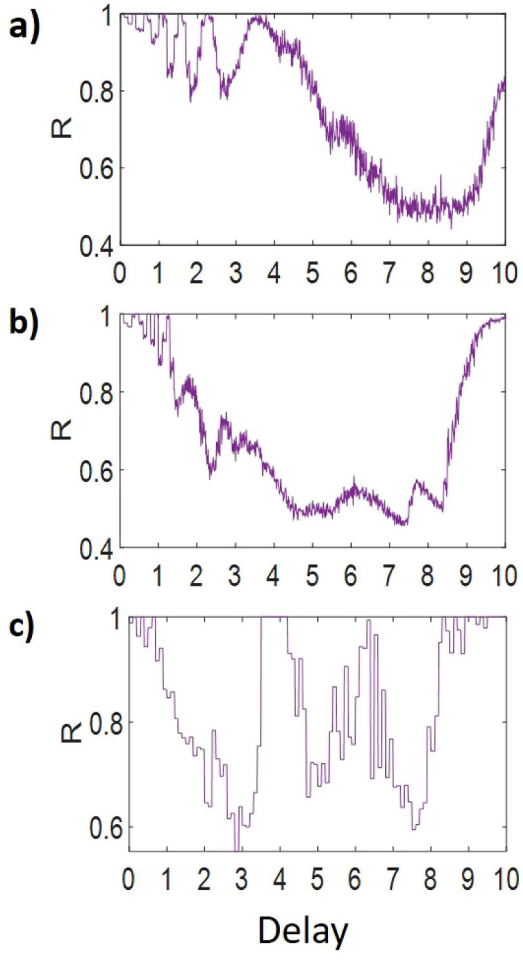

Fig. 6. Dependence of parameter R on the time delay τ for different values of P_d. (a) $P_d = 0.3$, (b) $P_d = 0.7$ and (c) $P_d = 1$. Figure indicates that both the delay and the probability are important factors in network pattern.

latency values of less than $\tau = 5$, is associated with an increase in the level of the order parameter.

Figure 6b shows the time delay effect for a situation where network connections are delayed by $P_d = 0.7$, as can be seen, for delay values less than $\tau = 5$. The order parameter R decreases with increasing time delay. Also Figures 1a and 1b confirm that the network pattern has become more disordered by varying the latency from $\tau = 2.5$ to $\tau = 7$. The worst-case scenario of the network patterns occurs for time delays of about $\tau = 4$ to $\tau = 8$ as shown in Figure 2b, which is completely asynchronous for $\tau = 7$. By increasing the time delay up to $\tau = 10$ network returns to be ordered again, when the network synchronization pattern becomes imperfect phase synchronized (see Fig. 2c).

Figure 6c indicates the variation of the order parameter relative to the time delay variation when $P_d = 1$. As the time delay increases up to about $\tau = 2$, the order parameter R decreases. For latencies from $\tau = 2$ to $\tau = 8$, the order parameter becomes continuously low and high. In this interval, chimera phenomenon occurs for some delay values. Also, for some values of the delay, the dynamics of firing of neurons change (see Fig. 5). It can be said that changing the dynamics of the firing of neurons changes the network synchronization pattern (see Fig. 4). Also, according to Figure 4, for some of the intermediate values, close to $\tau = 4$, the network reaches to its maximum order. For larger values of time delay, the network goes towards full synchronization, in which case the parameter values are equal or close to 1.

5 Conclusion

Brain is a complex system and studying its collective behaviors such as synchronization transition, pattern selection, pattern stability, stochastic resonance, etc. can be very helpful in understanding different neuronal mechanisms [10]. It has been shown that however gap junctions and chemical synapses have different effects on synchronization transitions [48], gap junctions with time delays can provide synchronization states, destabilize synchronous states and generate various spatiotemporal

patterns [48,49]. Thus, in this paper, we generally studied the effects of partial time delays on a neuronal network of electrically coupled Izhikevich neuron model oscillators and investigated the network patterns via changing two control parameters of time delay and the probability of partial time delay. It was inferred that when the probability of partial time delays is small, increasing of delay can change the synchronization type from zero-lag to distorted sine-like and then to phase synchronization. If the probability of partial time delays increases, it can cause the emergence of asynchronous state for special time delay, therefore varying time delay induces transitions from synchronous to asynchronous states. Finally, in the case of full time delay, raising the value of partial time delays can increase or decrease synchronization and cause synchronization transitions between coherent and incoherent or chimera states. It can also influence on synchronized neurons to switch between different periodic patterns. The observed patterns play significant role in the understanding of information processing and totally the structure and function of the human brain.

The neurons connectivity in the brain are coupled mainly locally, with sparse long distance connections which supports small-worldness of the brain. So in the future, we will try to apply numerical analysis to a small world network of Izhikevich neuron model oscillators with partial time delays.

This research was supported by the Slovenian Research Agency (Grants J1-7009, J4-9302 and J1-9112).

Author contribution statement

All the authors designed research, performed research and wrote the paper.

References

1. S. Krishnagopal, J. Lehnert, W. Poel, A. Zakharova, E. Schöll, Philos. Trans. R. Soc. A **375**, 20160216 (2017)
2. P. Jaros, L. Borkowski, B. Witkowski, K. Czolczynski, T. Kapitaniak, Eur. Phys. J. Special Topics **224**, 1605 (2015)
3. T. Chouzouris, I. Omelchenko, A. Zakharova, J. Hlinka, P. Jiruska, E. Schöll, Chaos **28**, 045112 (2018)
4. M. Jalili, in *International Conference on Information Management and Engineering, ICIME'09* (IEEE, Piscataway, NJ, 2009), p. 17
5. X. Sun, M. Perc, J. Kurths, Chaos **27**, 053113 (2017)
6. I. Belykh, E. de Lange, M. Hasler, Phys. Rev. Lett. **94**, 188101 (2005)
7. B. Tadić, M. Andjelković, B.M. Boshkoska, Z. Levnajić, PloS One **11**, e0166787 (2016)
8. M. Jalili, Physica A **466**, 325 (2017)
9. J. Tang, J. Ma, M. Yi, H. Xia, X. Yang, Phys. Rev. E **83**, 046207 (2011)
10. J. Ma, J. Tang, Nonlinear Dynam. **89**, 1569 (2017)
11. S. Achuthan, C.C. Canavier, J. Neurosci. **29**, 5218 (2009)
12. Z. Levnajić, A. Pikovsky, Phys. Rev. E **82**, 056202 (2010)
13. Y.S. Cho, T. Nishikawa, A.E. Motter, Phys. Rev. Lett. **119**, 084101 (2017)
14. S. Rakshit, B.K. Bera, M. Perc, D. Ghosh, Sci. Rep. **7**, 2412 (2017)
15. Z. Faghani, Z. Arab, F. Parastesh, S. Jafari, M. Perc, M. Slavinec, Chaos Soliton. Fract. **114**, 306 (2018)
16. Z.G. Nicolaou, H. Riecke, A.E. Motter, Phys. Rev. Lett. **119**, 244101 (2017)
17. C. Wang, M. Lv, A. Alsaedi, J. Ma, Chaos **27**, 113108 (2017)
18. J. Ma, F. Wu, C. Wang, Int. J. Mod. Phys. B **31**, 1650251 (2017)
19. F. Parastesh, S. Jafari, H. Azarnoush, B. Hatef, A. Bountis, Chaos Soliton. Fract. **110**, 203 (2018)
20. I. Omelchenko, E. Omel'chenko, A. Zakharova, E. Schöll, Phys. Rev. E **97**, 012216 (2018)
21. A. Mishra, S. Saha, P.K. Roy, T. Kapitaniak, S.K. Dana, Chaos **27**, 023110 (2017)
22. Y. Maistrenko, S. Brezetsky, P. Jaros, R. Levchenko, T. Kapitaniak, Phys. Rev. E **95**, 010203 (2017)
23. D. Dudkowski, Y. Maistrenko, T. Kapitaniak, Chaos **26**, 116306 (2016)
24. J. Wojewoda, K. Czolczynski, Y. Maistrenko, T. Kapitaniak, Sci. Rep. **6**, 34329 (2016)
25. R. Mukherjee, A. Sen, Chaos **28**, 053109 (2018)
26. F.P. Kemeth, S.W. Haugland, K. Krischer, Phys. Rev. Lett. **120**, 214101 (2018)
27. J.F. Totz, J. Rode, M.R. Tinsley, K. Showalter, H. Engel, Nat. Phys. **14**, 282 (2018)
28. M. Bolotov, L. Smirnov, G. Osipov, A. Pikovsky, Chaos **28**, 045101 (2018)
29. S. Majhi, M. Perc, D. Ghosh, Chaos **27**, 073109 (2017)
30. J. Ma, F. Wu, C. Wang, Int. J. Mod. Phys. B **31**, 1650251 (2017)
31. S. Rakshit, A. Ray, B.K. Bera, D. Ghosh, Nonlinear Dynam. **94**, 785 (2018)
32. C.K. Volos, D. Prousalis, I.M. Kyprianidis, I. Stouboulos, S. Vaidyanathan, V.T. Pham, Int. J. Control Theory Appl. **9**, 101 (2016)
33. Z. Wei, F. Parastesh, H. Azarnoush, S. Jafari, D. Ghosh, M. Perc, M. Slavinec, Europhys. Lett. **123**, 48003 (2018)
34. A. Schmidt, T. Kasimatis, J. Hizanidis, A. Provata, P. Hövel, Phys. Rev. E **95**, 032224 (2017)
35. I. Omelchenko, A. Provata, J. Hizanidis, E. Schöll, P. Hövel, Phys. Rev. E **91**, 022917 (2015)
36. J.M. Sausedo-Solorio, A.N. Pisarchik, Eur. Phys. J. Special Topics **226**, 1911 (2017)
37. J.L. Wang, H.N. Wu, T. Huang, Automatica **56**, 105 (2015)
38. Z. Levnajić, B. Tadić, J. Stat. Mech. Theory Exp. **3**, P03003 (2018)
39. H. Gu, Z. Zhao, PloS One **10**, e0138593 (2015)
40. Y. Çakir, Turk. J. Electr. Eng. Comp. Sci. **25**, 2595 (2017)
41. E. Rossoni, Y. Chen, M. Ding, J. Feng, Phys. Rev. E **71**, 061904 (2005)
42. M. Masoliver, N. Malik, E. Schöll, A. Zakharova, Chaos **27**, 101102 (2017)
43. M. Jalili, Neurocomputing **74**, 1551 (2011)
44. M. Jalili, Chaos **23**, 013146 (2013)
45. A. Zakharova, N. Semenova, V. Anishchenko, E. Schöll, Chaos **27**, 114320 (2017)

46. A. Gjurchinovski, E. Schöll, A. Zakharova, Phys. Rev. E **95**, 042218 (2017)
47. S. Nobukawa, H. Nishimura, T. Yamanishi, Sci. Rep. **7**, 1331 (2017)
48. Y. Hao, Y. Gong, L. Wang, X. Ma, C. Yang, Chaos Soliton. Fract. **44**, 260 (2011)
49. M.V. Bennett, R.S. Zukin, Neuron **41**, 495 (2004)