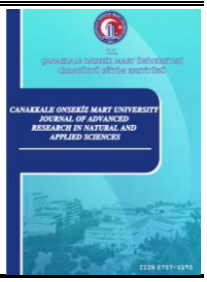
Çanakkale Onsekiz Mart University

Journal of Advanced Research in Natural and Applied Sciences

dergipark.org.tr/tr/pub/jarnas

Open Access



e-ISSN: 2757-5195 2024, Vol. 10, Issue 1, Pages: 80-90 doi.org/10.28979/jarnas.1140961

On the Synchronizability of Quadratic Integrate and Fire Neurons

Koray Çiftçi[1]

[1]Department of Biomedical Engineering, Çorlu Faculty of Engineering, Tekirdağ Namık Kemal University, Tekirdağ, Türkiye

Article History

Received: 5 Jul 2022

Accepted: 31 Oct 2023

Published: 15 Mar 2024

Research Article

Abstract − Synchronization is a property of complex systems that manifests itself as the emergence of collective behavior from local interactions. Neurons are the basic building blocks of the nervous system, and in neuronal networks, the firing times of the neurons get synchronized via the electrical and chemical synapses among them. This property has been observed in both computational models and experimental studies. However, this synchronization's mechanisms have not yet been totally revealed. Here, we investigate the synchronization properties of quadratic integrate and fire (QIF) neurons from a computational modeling perspective. QIF neurons are simple yet effective models in the sense that they have the ability to capture complex behavior observed in neurons. We present analytical results concerning the spiking frequency of the QIF neurons and the relationships between membrane voltage and phase of the neurons. We give simulation results for a simple network of all-to-all coupled QIF neurons, demonstrating the effects of different types of coupling among the network members. We show that electrical and inhibitory chemical synapses play complementary roles in the formation of synchronized behavior in a neuronal network. Our results contribute to our understanding of the brain to produce cognitive abilities and coordinated action.

Keywords − *Integrate and fire, neuron, phase, synchronization*

1. Introduction

Computational modeling efforts in neuroscience generally seek a balance between biological realism and computational tractability. Biological realism brings complexity to the models, whereas simplicity is crucial to be able to arrive at generalizable inferences. The need for simplicity becomes even more crucial with the increasing usage of network models in neuroscience. Conductance-based models [1] can reproduce the output of many electrophysiological recordings. However, because of its inherent complexity, extending conductance-based single-neuron models to large networks of neurons is not easy. On the other hand, integrate and fire neuron models offer a great reduction in complexity but with the capability of representing the rich dynamical repertoire of neurons, especially at regions close to the firing threshold [2].

Integrate and fire neuron model is one of the earliest models in neuroscience, dating back to 1907 [3]. The model tracks the membrane potential and acts as an integrator of subthreshold stimuli. It incorporates a reset mechanism for the neuron to return to resting potential after triggering a spike [4]. Quadratic integrate and fire (QIF) neuron model is a type of integrate and fire neuron model, and it is actually the simplest form of a spiking neuron. Although simplistic in the sense that it is based on a single variable, the QIF model is widely used to model a wide range of neuronal phenomena [5], including learning [6], memory [7], and movement-induced rhythms [8]. It was also demonstrated that QIF is appropriate for modeling neural oscillations [9,10].

Neurons exhibit an oscillatory behavior [11], and a prominent feature of a neuron is its ability to synchronize

[1]kciftci@nku.edu.tr (Corresponding Author)

these oscillations with other neurons [12]. This synchronization takes place under the action of synaptic couplings and is the basis of all attention [13], memory [14], coordinated action [15], and related phenomena. Although the underlying mechanisms of this synchronization are still not totally revealed, it is known that it is an emergent property with no central executive coordinating this synchronization process [16]. Thus, synchronization is a global phenomenon arising from local interactions among the neurons [17].

Oscillatory behavior and activity synchronization are not an exclusive property of neurons and are observed in many natural systems [18,19]. Observing collective behavior at different levels in living systems, from micro to macro, is possible. Therefore, it is important to discover the underlying mechanisms of this behavior to understand living systems and adapt these principles to engineering systems [20].

The synchronization characteristics of QIF neurons have already been investigated in a number of studies and the link between the Kuramoto Model [21] and the QIF model has been established [22], voltage dependence of synchronization for electrical synapses has been explored [23] and a mean field approximation for networks of QIF neurons has been proposed [24]. Recently, it has been shown that spike timing dependent plasticity allows QIF neurons to form large clusters that generate synchronous oscillations [25]. Earlier work has shown that synchronization can be achieved both with the coordinated action of inhibitory and excitatory inputs [26] or with inhibition alone [27]. A number of works have reported that inhibitory synapses increase the synchronization capability of neuronal networks [28,29]. And although electrical synapses are thought to promote synchronization in general [30], it has also been observed that anti-synchronous activity can also emerge from weak electrical coupling [31]. Thus, the joint role of electrical and inhibitory chemical coupling is still elusive. Based on these findings, in this study we particularly aim at revealing the complementary roles of inhibitory chemical synapses and electrical synapses in the formation synchronous activity in a network of QIF neurons. In the Materials and Methods, we explain the basic temporal dynamics of a single QIF neuron and the relationship between its membrane voltage and phase. This enables us to describe the neuron as an oscillator. After investigating the phase sensitivity of a neuron, we extend our analysis to a network of QIF neurons and investigate its synchronization properties under different coupling conditions. After presenting the results with discussion we conclude the paper with a summary of our results and a description of future work.

2. Materials and Methods

We first consider a single isolated neuron subject to a constant external current, but with no synaptic input. The QIF model for this neuron reads (2.1),

$$\tau \dot{v} = v^2 + \eta, \quad \text{if } v > v_p, \quad \text{then } v \leftarrow v_r \tag{2.1}$$

where, v is the membrane potential, τ is the membrane time constant and η is external current noise. Since membrane voltage increases quadratically, a resetting rule is needed, which is given as the if statement in the equation. When $\eta < 0$, the model has one stable equilibrium, which corresponds to the resting state of the neuron, i.e., $V_r = -\sqrt{\eta}$, whereas the other equilibrium point is unstable and acts as the threshold voltage for spike generation, i.e., $V_p = +\sqrt{\eta}$. In this study, we will consider the case $\eta > 0$, which corresponds to the situation where there is no equilibrium point and the neuron acts as a self-sustained oscillator. If we set $v(t = 0) = v_r$, then the time evolution of the membrane voltage may be determined by a simple separation of variables (2.2):

$$\int_{v_r}^{v(t)} \frac{1}{v'^2+\eta} dv' = \frac{1}{\tau}\int_0^t dt' \tag{2.2}$$

which gives (2.3),

$$v(t) = \sqrt{n}\tan\left(\frac{\sqrt{n}}{\tau}t + \tan^{-1}\frac{V_r}{\sqrt{n}}\right) \tag{2.3}$$

The period of oscillation, T, may be determined by calculating the time at which $v(t) = V_p$, and is given by (2.4),

$$T = \frac{\tau}{\sqrt{n}}\left(\tan^{-1}\frac{V_p}{\sqrt{n}} - \tan^{-1}\frac{V_r}{\sqrt{n}}\right) \tag{2.4}$$

A symmetric spike resetting is generally assumed, i.e., $V_p = -V_r$, in which case the period becomes (2.5):

$$T = 2\frac{\tau}{\sqrt{n}}\tan^{-1}\frac{V_p}{\sqrt{n}} \tag{2.5}$$

Generally, for mathematical analysis, V_p is assumed to go to infinity, and this makes the period of spike generation a simple function of membrane time constant and external current (2.6):

$$T = \frac{\pi\tau}{\sqrt{n}} \tag{2.6}$$

In practical cases, we can set $V_p \gg \sqrt{n}$ for the above equation to hold. Thence, writing $\omega = \frac{2\pi}{T} = \frac{2\sqrt{\eta}}{\tau}$, a phase variable may be introduced: $\phi = \omega t = \left(\frac{2\sqrt{\eta}}{\tau}\right)t$, which may be derived from (2.3) as (2.7),

$$\phi = 2\arctan\frac{v}{\sqrt{n}} + \pi \tag{2.7}$$

This function may be seen as a mapping between membrane voltage and phase, which turns the neuron into a rotating oscillator on the unit circle that emits spikes when $\phi = 2\pi$.

Figure 1 shows the time course of membrane voltage and phase for a QIF neuron. Membrane voltage obeys (2.1) and the phase is calculated for each voltage value from (2.7). It may be seen that the spiking period is close to *10 s*, which may be confirmed from (2.6). The membrane voltage of the neuron has fast dynamics immediately after a spike and just before the spike and a slow increase governs the rest of its temporal dynamics. On the other hand, please note the linear increase in the phase of the neuron between the spikes. Actually, we had derived the angular velocity above, and thus, the mapping given in (2.7) converts the non-linear temporal dynamics of membrane voltage to a linear phase system. We can also verify this using the chain rule (2.8):

$$\frac{d\phi}{dt} = \frac{d\phi}{dv}\frac{dv}{dt} = \frac{2}{\sqrt{\eta}\left(1+\frac{v^2}{\eta}\right)}\frac{(v^2+\eta)}{\tau} = 2\frac{\sqrt{\eta}}{\tau} \tag{2.8}$$

This analysis shows that phase temporally depends on the external current and membrane time constant, and if these two parameters are constant then the phase of the neuron changes linearly between the spikes.

In our simulations, we will consider nearly identical neurons, in the sense that their membrane time constants will be identical but the external noise they are exposed to will be stochastic with a definite mean and variance.

Thus, the instantaneous change of phase will be dependent on the instantaneous noise component but can be assumed to be constant over a long time which is determined by the mean of the external noise, η.

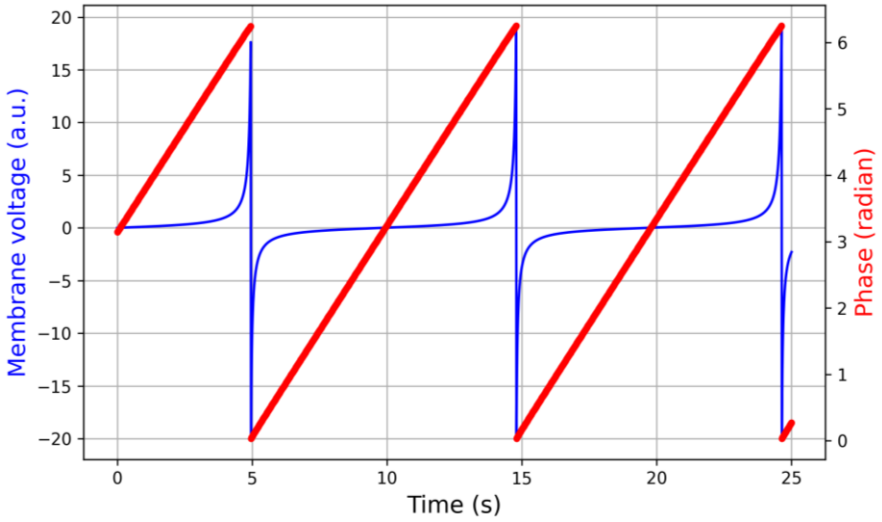


Figure 1. The time course of membrane voltage, v, for a QIF neuron with $V_p = -V_r = 20$, $\tau = 1$, and $\eta = 0.1$

2.1. Phase Sensitivity of a Neuron

The phase of a QIF neuron is a function of its membrane voltage and perturbations in this voltage cause an alteration in the phase of the neuron. In (2.7), we have noted the derivative of phase with respect to voltage (2.9):

$$\frac{d\phi}{dv} = \frac{2}{\sqrt{\eta}\left(1 + \frac{v^2}{\eta}\right)} \tag{2.9}$$

This equation tells us that the phase sensitivity depends both on the corresponding voltage and on the external noise component, and the sensitivity is maximum when $v = 0$. It is also possible to derive the sensitivity of the phase to the noise component, η, and can be shown that phase sensitivity is maximum (for a constant voltage) at $\eta = v^2$.

Phase sensitivity may be empirically determined by applying small perturbations to the membrane voltage at different phases, $\phi = \frac{t_s}{T_o}$, where T_o is the intrinsic period of the neuron and t_s is the time between the last action potential and the perturbation. We can then measure the corresponding change in the period, $\frac{(T_o - T_p)}{T_o}$, where T_p is the period in the perturbed interval. This function, relating phase to voltage perturbation is also called the phase resetting curve (PRC) of a neuron [32]. It summarizes the responses of a neuron to external inputs, from neighboring neurons or other noise components.

Figure 2 shows the analytical and numerical PRC of a QIF neuron. The two curves are in close agreement with each other, and they show that the neuron is rather insensitive to external perturbations immediately after and just before a spike, and the sensitivity is comparably much higher during the interval between these two ends. Another point to note is that phase sensitivity is always non-negative. Thus, positive perturbations will shorten the spike interval, whereas negative perturbations will cause a delay in the next spike time.

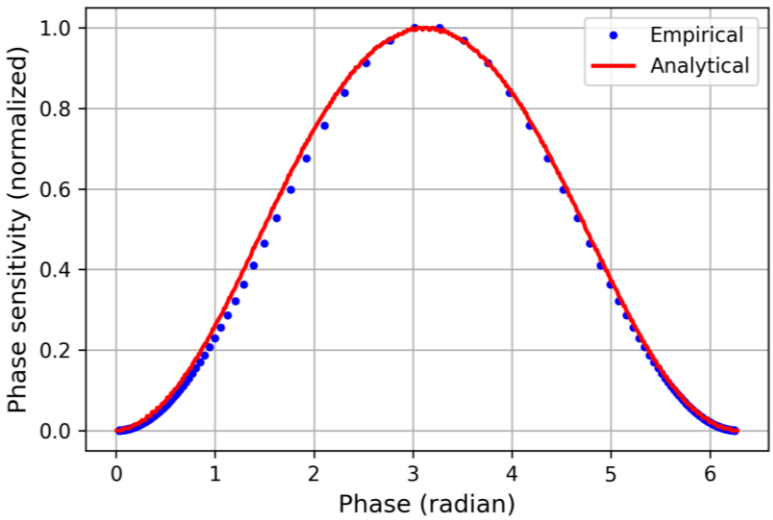


Figure 2. Analytical and empirical phase sensitivity curves for a QIF neuron. These curves show the normalized increase (or decrease) in the spiking period with an infinitesimal perturbation in the membrane voltage

2.2. A Simple Network of QIF Neurons

Neurons do not function in an isolated manner, and our main concern is the relationship among the interacting neurons. Equipped with a general understanding of phase sensitivity of a single neuron, we will now consider a network of QIF neurons, whose individual membrane voltages are evolving according to (2.10),

$$\tau \dot{v}_i = v_i^2 + \eta_i + S_i(t), \quad \text{if } v_i > v_p, \quad \text{then } v_i \leftarrow v_r \tag{2.10}$$

where $i \in \{1,2,\cdots,N\}$ indexes all-to-all coupled N neurons. $S_i(t)$, is the total time dependent synaptic input to the neuron and has two components: chemical and electrical synapses. Electrical synapses are gap junctions which permit the connected neurons to conduct electricity. In our model, electrical synapses are coupled to the neurons by the mean membrane voltage (2.11):

$$S_i^e(t) = g(\mu_V(t) - v_i), \quad \mu_v(t) = \sum_{j=1}^{N} v_j(t) \tag{2.10}$$

where $\mu_v(t)$ is the mean membrane voltage of the neuron population and g is a constant denoting the strength of the synaptic coupling which may be seen as the average conductance of the synaptic channels. Chemical synapses, on the other hand, are biological junctions that act through neurotransmitters, and they allow the transmission of presynaptic signals to postsynaptic neurons.

In the current model, chemical synapses are mediated by mean firing rate (2.12):

$$S_i^c(t) = \frac{C}{N} \sum_{j=1}^{N} \sum_{k} \delta\left(t - t_j^k\right) \tag{2.10}$$

where C is a constant denoting the strength of chemical couplings, t_j^k is the k^{th} spike of the j^{th} neuron, and $\delta(\cdot)$ is the *Dirac delta function*.

Electrical and chemical synapses play complementary roles in neuronal network synchronization [33]. The strength of coupling needed for synchronization depends on the relative contributions of chemical and

electrical synapses [34]. Additionally, chemical synapses may be inhibitory or excitatory. Excitatory synapses increase the activity of the postsynaptic neuron, whereas inhibitory synapses work in the reverse direction.

It has been shown that inhibitory synapses increase the synchronization capability of neuronal networks [28]. However, the exact roles of electrical and inhibitory coupling in the formation of synchronous activity is still under debate. Therefore, in this study we will only consider inhibitory chemical synapses and in order to investigate the effect of both types of coupling, we analyse the network in two different settings: 1) Only electrical coupling, 2) Inhibitory chemical coupling and electrical coupling.

3. Results and Discussion

Here, we present the results for a network of $N = 256$ all-to-all coupled neurons. Since the membrane time constant, τ, acts as an inverse multiplier of integration time in our simulations, *w*ithout loss of generality, we will assume that $\tau = 1$. To keep the heterogeneity among the neurons at a low level we impose the constraint that the mean of the Gaussian noise is much larger than the standard deviation. This is also needed to satisfy the weak coupling regime between the QIF neurons. Accordingly, the external noise has a Gaussian distribution with a mean of 0.1 and a standard deviation of 0.01.

The QIF model is not directly related to physiological voltage levels. Nevertheless, to keep the voltage levels at reasonable ranges, the neurons were randomly initialized uniformly between -1 and 1 V and time courses of their membrane voltages were determined by Euler integration with time steps of *0.01 s*.

3.1. Only Electrical Synapses

We first set $C = 0$ and let the network interact only through electrical synapses. Beginning from a g value of 0.01, we incrementally increased g with steps of 0.01 and check for synchronization. To quantify the synchronization, we calculated the *index of dispersion* (*ratio of variance to mean*) of spike intervals of the neurons.

In Figure 3, it is observable that dispersion index drops close to zero beginning from $g = 0.04$. Thus, we deducted that synchronization is achievable beginning from this value of g.

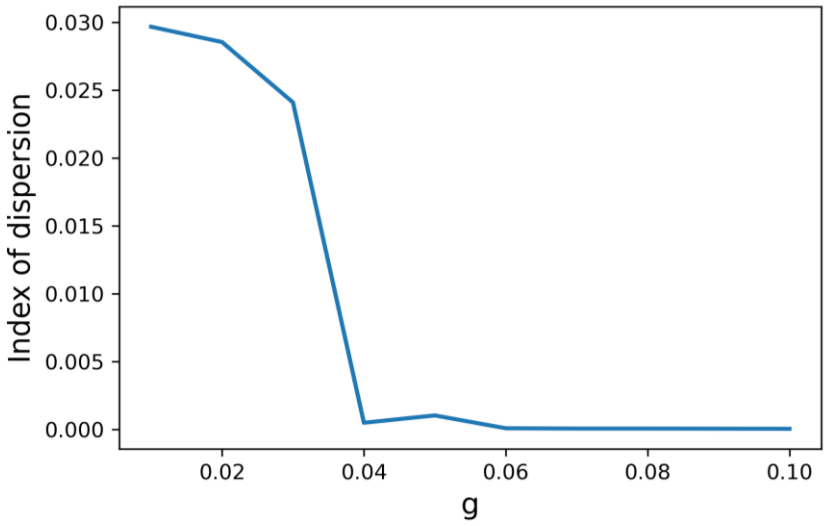


Figure 3. Index of dispersion of spiking intervals with varying electrical coupling strength. Decrease in dispersion points to the emergence of synchronization

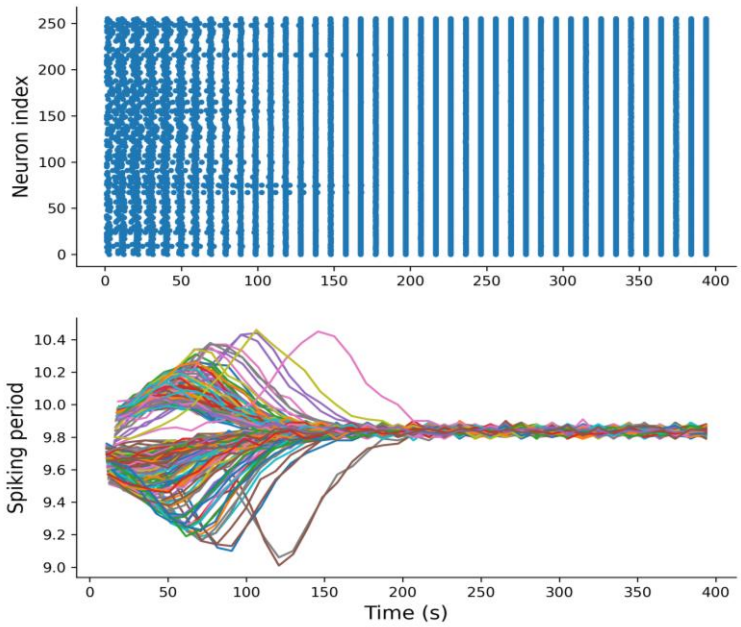


Figure 4. Top: Raster plots of neurons. Each firing of a neuron is represented as a dot at that time point. Synchronization in firings is evident as time progresses. Bottom: Evolution of spiking intervals of neurons. Spiking intervals converge as time progresses

In Figure 4, we present the raster plot and the change in the spiking periods of the neurons for $g = 0.05$. Beginning from an unsynchronized state, the neurons organize themselves towards a synchronized state. During this interval, their spiking periods converge to their intrinsic periods (*~9.8 s*). Since the external current is stochastic small fluctuations continue after convergence. Please note that this is the only achievable synchronized condition for identical neurons with electrical coupling. Since this coupling is mediated by mean membrane voltage, for those neurons whose membrane voltage is lower than the average, electrical coupling acts as an accelerator whereas for the ones that are higher than the average, coupling causes a deceleration. And as time progresses every neuron is pulled towards the mean and begins to trigger a spike at the same time. Interestingly, this pull towards synchronization also decreases the total amount of electrical coupling among the neurons. This phenomenon may be understood within the light of optimality of the brain [35]. Thus, synchronization is not achieved per se but is a state that comes out of a more general principle of energy minimization.

3.2. Inhibitory Chemical Coupling and Electrical Coupling

After investigating the role of electrical synapses on the synchronization of the neuronal network, we proceed with analyzing the combined effects of inhibitory chemical synapses and electrical synapses. Cortical circuits display a layer-specific excitation-inhibition balance in generating synchronization between neuronal assemblies [36]. In the sequel, we conjectured that a certain amount of inhibitory coupling would corroborate the synchronization capacity of the network. To be able to test this conjecture we set an electrical coupling constant of $g = 0.02$ (which is below the full synchronization level of the network). Then, we increased the inhibitory chemical coupling and checked the synchronization. We observed that the synchronization capacity increased after a certain level of inhibition, but began to decrease with further increase, which points to an optimum exhibition/inhibition ratio. In Figure 5, it may be observed that the network reaches synchronization with a larger time delay. As a further test, we lowered g and found that inhibitory coupling should also be lowered in the same amounts for synchronization (Figure 6).

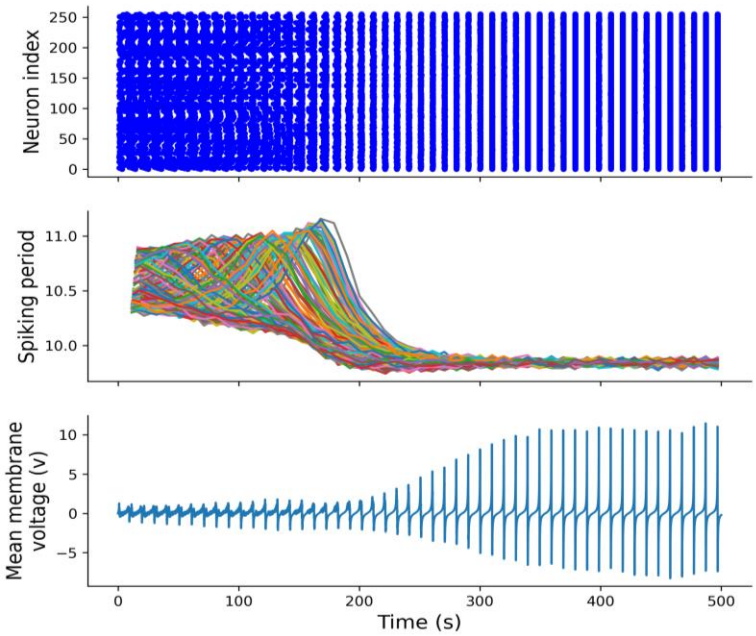


Figure 5. The definitions of the firts two plots are as in Figure 4. At the bottom we included the mean membrane voltage of the population. Electrical coupling constant g is 0.02, inhibitory coupling constant $C = -0.06$. Please note that simulation time is 500 seconds

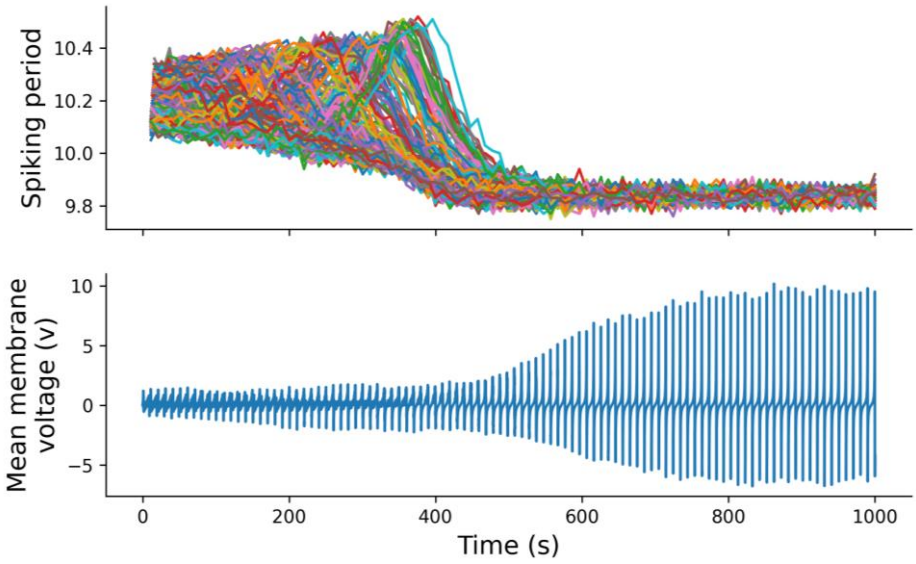


Figure 6. The definitions of the plots are as in Figure 4. Electrical coupling constant g is 0.01, inhibitory coup-ling constant $C = -0.12$. Please note that simulation time is 1000 seconds

4. Conclusion

In this study, we provide further evidence for the complementary roles of inhibitory chemical synapses and electrical synapses in the formation of synchronized behavior in networks of coupled neurons. First, we showed that the network may become synchronized with only electrical coupling. This synchronization takes place at a certain amount of coupling, and we also made the observation that synchronized activity also decreased the total amount of coupling among the neurons. We related this observation to the optimality of the brain hypothesis since every interaction between the neurons needs energy consumption [37]. Second, we showed that with an injection of inhibitory coupling, it may become possible for the network to arrive at full

synchronization with less electrical coupling but with a larger time delay. It has been claimed that electrical coupling had increased the robustness of inhibition induced synchronization [38]. Our results do not directly relate to this conjecture but provide an alternative look at the interplay between inhibitory and electrical coupling. Interestingly, we also observed that further increases in inhibition causes a loss in the synchronization. This point is of note since it has been reported that for a total amount of coupling either electrical coupling only or inhibition only is better at arriving neural synchrony [39]. Please note that in our simulations only electrical coupling is more efficient for driving the network towards synchrony and with the addition of inhibition this electrical coupling should be decreased. And after the network reaches synchronization, further increase in inhibition deteriorates synchrony.

This study analyzed quadratic integrate and fire neurons as use cases, however similar approaches can be extended to other neuron models. Physiologically inspired neuronal models and network structures may be used to explore the synchronization phenomenon in biologically more plausible settings.

Author Contributions

The author read and approved the final version of the paper.

Conflicts of Interest

The author declares no conflict of interest.

References

[1] A. L. Hodgkin, A. F. Huxley, *A quantitative description of membrane current and its application to conduction and excitation in nerve*, The Journal of Physiology 117 (4) (1952) 500.

[2] R. Jolivet, T. J. Lewis, W. Gerstner, *Generalized integrate-and-fire models of neuronal activity approximate spike trains of a detailed model to a high degree of accuracy*, Journal of Neurophysiology 92 (2) (2004) 959–976.

[3] N. Brunel, M. C. Van Rossum, *Lapicque's 1907 paper: From frogs to integrate-and-fire*, Biological Cybernetics 97 (5–6) (2007) 337–339.

[4] A. N. Burkitt, *A review of the integrate-and-fire neuron model: I. Homogeneous synaptic input*, Biological Cybernetics 95 (2006). 1–19.

[5] E. M. Izhikevich, Dynamical systems in neuroscience, MIT Press, London, 2007.

[6] D. Mishra, A. Yadav, P. K. Kalra, *Learning with single quadratic integrate-and-fire neuron*, in: J. Wang, Z. Yi, J. M. Zurada, B. L. Lu, H. Yin, (Eds.) Advances in Neural Networks - ISNN 2006, Vol. 3971 of Lecture Notes in Computer Science, Springer, Berlin, 2006, pp. 424–429.

[7] D. Hansel, G. Mato, *Existence and stability of persistent states in large neuronal networks*, Physical Review Letters 86 (18) (2001) 4175–4178.

[8] Á. Byrne, M. J.Brookes, S. Coombes, *A mean field model for movement induced changes in the beta rhythm*, Journal of Computational Neuroscience 43 (2) (2017) 143–158.

[9] G. B. Ermentrout, C. C. Chow, *Modeling neural oscillations*, Physiology and Behavior 77 (4) (2002) 629–633.

[10] S. Keeley, Á. Byrne, A. Fenton, J. Rinzel, *Firing rate models for gamma oscillations*, Journal of Neurophysiology 121(6) (2019) 2181–2190.

[11] G. Buzsaki, A. Draguhn, *Neuronal oscillations in cortical networks*, Science 304 (5679) (2004) 1926–1929.

[12] T. Womelsdorf, J. M. Schoffelen, R. Oostenveld, W. Singer, R. Desimone, A. K. Engel, P. Fries, *Modulation of neuronal interactions through neuronal synchronization*, Science 316 (5831) (2007) 1609–1612.

[13] T. Womelsdorf, P. Fries, *The role of neuronal synchronization in selective attention*, Current Opinion in Neurobiology 17 (2) (2007) 154–160.

[14] N. Axmacher, F. Mormann, G. Fernández, C. E. Elger, J. Fell, *Memory formation by neuronal synchronization*, Brain Research Reviews 52 (1) (2006) 170–182.

[15] J. Van Der Werf, O. Jensen, P. Fries, W. P. Medendorp, *Neuronal synchronization in human posterior parietal cortex during reach planning*, Journal of Neuroscience 30 (4) (2010) 1402–1412.

[16] F. Varela, J. P. Lachaux, E. Rodriguez, J. Martinerie, *The brainweb: Phase synchronization and large-scale integration*, Nature Reviews Neuroscience 2 (4) (2001) 229–239.

[17] M. V. L. Bennett, R. S. Zukin, *Electrical Coupling and Neuronal Synchronization in the Mammalian Brain*, Neuron 41 (4) (2004) 495–511.

[18] S. H. Strogatz, I. Stewart, *Coupled oscillators and biological synchronization*, Scientific American 269 (6) (1993) 102–109.

[19] L. Glass, *Synchronization and rhythmic processes in physiology*, Nature 410 (6825) (2001) 277–284.

[20] X. Li, G. Chen, *Synchronization and desynchronization of complex dynamical networks: An engineering viewpoint*, IEEE Transactions on Circuits and Systems I: Fundamental Theory and Applications 50 (11) (2003) 1381–1390.

[21] Y. Kuramoto, *Collective synchronization of pulse-coupled oscillators and excitable units*, Physica D: Nonlinear Phenomena 50 (1) (1991) 15–30.

[22] P. Clusella, B. Pietras, E. Montbrió, *Kuramoto model for populations of quadratic integrate-and-fire neurons with chemical and electrical coupling. Chaos: An interdisciplinary*, Journal of Nonlinear Science 32 (1) (2022) 013105.

[23] M. Jafarian, K. H. Johansson, (2019). Synchronization of quadratic integrate-and-fire spiking neurons: Constant versus voltage-dependent couplings. 2019 IEEE 58th Conference on Decision and Control (CDC), 4711-4716.

[24] W. Ye, *Dynamics of a large-scale spiking neural network with quadratic integrate-and-fire neurons*, Neural Plasticity 2021 (2021) Article ID 6623926 10 pages.

[25] I. Ratas, K. Pyragas, *Interplay of different synchronization modes and synaptic plasticity in a system of class I neurons*, Scientific Reports 12 (1) (2022) Article Number 19631 17 pages.

[26] E. H. Buhl, G. Tamás, A. Fisahn, *Cholinergic activation and tonic excitation induce persistent gamma oscillations in mouse somatosensory cortex in vitro*, The Journal of Physiology 513 (1) (1998) 117–126.

[27] M. Beierlein, J. R. Gibson, B. W. Connors, *A network of electrically coupled interneurons drives synchronized inhibition in neocortex*, Nature Neuroscience 3 (9) (2000) 904–910.

[28] C. Van Vreeswijk, L. F. Abbott, G. Bard Ermentrout, *When inhibition not excitation synchronizes neural firing*, Journal of computational neuroscience 1 (4) (1994) 313–321.

[29] M. A. Whittington, R. D. Traub, N. Kopell, B. Ermentrout, E. H. Buhl, *Inhibition-based rhythms: Experimental and mathematical observations on network dynamics*, International Journal of Psychophysiology 38 (3) (2000) 315–336.

[30] R. D. Traub, *Model of synchronized population bursts in electrically coupled interneurons containing active dendritic conductances*, Journal of Computational Neuroscience 2 (1995) 283–289.

[31] C. C. Chow, N. Kopell, *Dynamics of spiking neurons with electrical coupling*, Neural Computation 12 (7) (2000) 1643–1678.

[32] C. C. Canavier, S. Achuthan, *Pulse coupled oscillators and the phase resetting curve*, Mathematical Biosciences 226 (2) (2010) 77–96.

[33] N. Kopell, B. Ermentrout, *Chemical and electrical synapses perform complementary roles in the synchronization of interneuronal networks*, Proceedings of the National Academy of Sciences 101 (43) (2004) 15482–15487.

[34] M. S. Baptista, F. M. Moukam Kakmeni, C. Grebogi, *Combined effect of chemical and electrical synapses in Hindmarsh-Rose neural networks on synchronization and the rate of information*, Physical Review E 82 (3) (2010) 036203.

[35] L. Koban, A. Ramamoorthy, I. Konvalinka, *Why do we fall into sync with others? Interpersonal synchronization and the brain's optimization principle*, Social Neuroscience 14 (1) (2019) 1–9.

[36] H. Adesnik, *Layer-specific excitation/inhibition balances during neuronal synchronization in the visual cortex*, The Journal of Physiology 596 (9) (2018) 1639–1657.

[37] A. Navarrete, C. P. Van Schaik, K. Isler, *Energetics and the evolution of human brain size*, Nature 480 (7375) (2011).

[38] J. A. White, C. C. Chow, J. Rit, C. Soto-Treviño, N. Kopell, *Synchronization and oscillatory dynamics in heterogeneous, mutually inhibited neurons*, Journal of Computational Neuroscience 5 (1) (1998) 5–16.

[39] T. J. Lewis, J. Rinzel, *Dynamics of spiking neurons connected by both inhibitory and electrical coupling*, Journal of Computational Neuroscience 14 (3) (2003) 283–309.