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Review

The effects of synaptic time delay on motifs of chemically coupled Rulkov model neurons

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ABSTRACT

Given the importance of the network motifs, we consider a pair of Rulkov chaotic map neurons, reciprocally coupled via symmetrical chemical synapses with the time delay τ . For the inhibitory and excitatory synapses, the system dynamics is determined by the synaptic weight g_c , synaptic gain parameter k, time delay τ and the external excitation σ . Due to chaotic nature of the map and synaptic model complexity, the appropriately averaged cross-correlation of membrane potentials represents a suitable numerical diagnostics to quantify mutual synchronization. Along with the expected phase and anti-phase synchronization regimes, we find the emergent phenomena that significantly influence the synchronization behavior.

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1. Introduction

A large variety of functions in neural networks is accomplished through cooperative activity of individual neurons or neuronal groups. Synchronization is identified as the most prominent mechanism underlying action coordination, encoding and transmission of information [1], while its precise role in information processing still remains to be determined [2]. Within this context, two broad classes of neuron models are studied: threshold models that display elementary spike

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synchronization, and the models with spiking and/or bursting dynamics, that demonstrate more complex synchronization phenomena (see [3,4] and references therein).

The study of bursting, where the series of spikes emitted in close succession alternate with quiescent phases of quasisteady states, has been motivated by the observed activity modes in hippocampal pyramidal neurons, dopaminergic neurons in midbrain, thalamic neurons and central pattern generators (see [6–8] and references therein). As a complex motion, bursting can be decomposed into two oscillations [9] at widely spread time scales: fast-scale oscillations, corresponding to spikes within the bursts, are superimposed on slow oscillations, that modulate the switches between the bursting phases. Interacting bursting neurons, which are believed to promote more efficient and reliable way of information transfer, as compared to spiking neurons [10–12], may exhibit three types of synchronizing behavior: individual spike synchronization, complete (exact) and burst synchronization [3–5]. In terms of synaptic strength needed, burst synchronization, detected by matching the instants of burst triggering between the neurons [13,14], is easier to achieve than complete synchronization, indicating that burst synchronization may be more commonly represented than complete synchronization mode. Burst synchronization requires the slow oscillations becoming synchronized, whereas the rapid oscillations remain asynchronous, which is reminiscent to coincidence of characteristic time scales in multi-scale motions, as in chaotic phase synchronization [15,16].

An obvious obstacle to studying large networks of bursting neurons lies in the fact that each subunit has to be represented by a strongly nonlinear set of at least three ordinary differential equations [17]. Instead of applying the neurobiological conductance-based models [18,19], one may turn to less complex phenomenological models, that are computationally more efficient and can increase the qualitative understanding of the synchronization phenomena.

While retaining the most important dynamical properties without explicit reference to physiological processes in the membrane, neuron model can be defined in form of a reduced-dimensional system of difference equations, comprising an iterative map. Taking advantage of the bursting decomposition, Rulkov proposed a two-dimensional map-based model, that can be adjusted to replicate a great deal of the experimentally observed regimes [10,20,21] concerning isolated and coupled neurons. This includes the response behavior with related transients [17], spike adaptation [22], routes from silence to tonic spiking or bursting, mediated by subthreshold oscillations [23], emergent bursting [6], as well as phase and anti-phase synchronization with chaos regularization [17,24]. In the present paper, we adopt the chaotic Rulkov map [6,24], which is capable of generating three types of individual neuron dynamics: silence, chaotic spiking and chaotic bursting.

Given the current assumptions about the ways in which complex networks are formed, researchers gained interest in identifying the statistically over-represented micro-circuits, possibly corresponding to network anatomical building blocks (structural motifs) and elementary processing subunits (functional motifs) [25-28]. Motifs can be viewed as minimal networks, whose behavior may offer important insights, or at least provide a starting point for the study of large neural networks. We analyze a pair of reciprocally coupled Rulkov model neurons, that, in light of motifs, may be termed a twonode feedback loop. The neurons are subject to excitatory and inhibitory chemical synapses, since they are by far the more commonly represented than electrical (gap-junction) synapses [12,29,30]. The chemical synapse is modeled to include the sigmoidal threshold function that accounts for the pulse-like release of neurotransmitters [3,31], and, most importantly, implements the transmission delays, inherent to this type of synapses [30,32]. Though the synaptic delay is known to significantly affect the coupled neuron dynamics [21,33,34], to our knowledge, this is the first time it is considered for the Rulkov model neurons. The intention is to examine the possible synchronization regimes arising for different neuron and synaptic parameters. The neuron parameters are taken to be homogeneous, since we focus on the interplay between the effects of synaptic strength and delay under different autonomous neuron regimes, controlled by the amplitude of the external stimulus current. For most of the parameter space we find intermittent, rather than stationary synchronization regimes, that are described by the appropriately averaged membrane potential cross-correlation. Along with the phase and anti-phase burst synchronization, we encounter the synchronization behavior that is substantially influenced by the emergent phenomena (oscillation death preceded by periodic windows, hyperpolarization and depolarization of membrane potential).

The paper is organized as follows. In the next section, we outline the details of neuron dynamics: first the isolated neuron behavior is analyzed in the phase plane and then the coupled neurons are introduced, with emphasis on synaptic modeling and the means to determine mutual synchronization regimes. In the Section 3, the effects of time delay and increasing external excitation are presented for low and moderate weights of excitatory and inhibitory synapses. In Section 4, we summarize the obtained results and put them into context by comparison with conductance-based neuron models.

2. Model

The dynamics of an isolated neuron follows the chaotic Rulkov map

$$\begin{aligned} x_{n+1} &= \frac{\alpha}{1+x_n^2} + y_n, \\ y_{n+1} &= y_n - \mu(x_n - \sigma), \end{aligned}$$
(1)

that couples the fast variable x_n to the slow variable y_n , whose evolution rate is low due to very small parameter $0 < \mu \ll 1$, set to $\mu = 0.001$. x_n corresponds to membrane potential (more precisely transmembrane voltage) and can be rescaled to allow exact comparison [22] to Hodgkin–Huxley model, whereas y_n is not explicitly derived from any biological framework, though some analogy to gating variables may be drawn [8,35]. The parameters α and σ , both being $\mathcal{O}(1)$, take part in evoking specific



Fig. 1. (a) Fast submap of the chaotic Rulkov neuron model. The rate of y_i evolution is slow enough to consider it as parameter γ of the fast variable dynamics. The map is pushed up (down) as γ increases (decreases), which effectively alters the number and position of fixed points. For $\gamma < \gamma_{sn}$ (solid line) the map has three fixed points, at $\gamma = \gamma_{sn}$ (dashed line) two, while above γ_{sn} (dotted line) a single fixed points remains. Only the two fixed points to the left are relevant to bursting behavior. (b) Bistability region of the fast submap. The fast variable nullclines form a parabola with the stable (N_s) and unstable (N_u) branches. The bursting region is limited by the hard boundary at $\gamma = \gamma_{sn}$ and the soft boundary at $\gamma = \gamma_{cn}$. Bursting occurs if the slow variable nullcline s are lies above the parabola vertex. The bursting amplitudes are determined by the curves of the minimal (Ξ_{min}) and maximal (Ξ_{max}) iterates of the fast submap.

neuron dynamical regimes, with σ emulating the action of an external dc bias current. As the fast submap alone is sufficient to replicate silent and tonic spiking regimes [22], coupling to the slow submap is required for triggering and terminating the bursts, as well as modulating the transients between bursting phases [6,22]. Due to very slow drifting of the recovery variable y_n [13,36], analysis of neuron dynamics may be reduced to the fast submap, where y_n is treated as a control parameter γ [24]. As γ increases (decreases), the fast submap (Fig. 1a) gets shifted up (down), which affects the number and position of its fixed points. The x_n dynamics exhibits three fixed points at most, two of which are complex conjugates that coincide and eventually disappear through the saddle-node bifurcation, as γ surpasses the value $\gamma = \gamma_{sn}$.

Precisely the stability of these two fixed points (the third fixed point will later be related to certain emergent phenomena) is relevant to bursting. This becomes more obvious if the dependence of the fast variable nullclines on γ is plotted in the phase plane (Fig. 1b). The fast variable nullclines form branches of a parabola that merge at $\gamma = \gamma_{sn}$, with the fixed points on the lower N_s (upper N_u) branch being always stable (unstable).

The existence of bistability region is essential to bursting: within a certain γ interval, along with the stable fixed point, there may also be a chaotic or periodic attractor between the curves of maximal and minimal iterates of the fast submap, Ξ_{max} and Ξ_{min} , respectively. For each γ , points on curves Ξ_{max} and Ξ_{min} are obtained as the first and second map iterates of zero, respectively. The region of coexisting attractors emerges if the curve of minimal iterates lies above the parabola vertex. Since the relative positions of Ξ_{min} and the parabola vertex are determined by the parameter α , there is a boundary value $\alpha = 4$, above which bursting is enabled. The γ interval corresponding to bursting is limited by the hard boundary $\gamma = \gamma_{sn}$ to the right, and the softer boundary $\gamma = \gamma_{cr}$ to the left, where Ξ_{min} intersects the unstable branch of fixed points N_u . While the onset of bursts is precisely determined by the local maxima of γ , termination of bursts may be delayed beyond $\gamma = \gamma_{cr}$ due to chaotic nature of the attractor around that point. The higher α , the more chaotic bursting becomes: enhancing α , the $\gamma_{sn} - \gamma_{cr}$ difference shrinks, making the effects of chaotic delays at γ_{cr} more visible [6]. Diminishing of $\gamma_{sn} - \gamma_{cr}$ presents an upper boundary on α , $\alpha = \alpha_c$, where bursting gives way to chaotic spiking.

If bursting is enabled ($4 < \alpha < \alpha_c$), the neuron activity regime is determined by the position of the slow variable nullcline $x_n = \sigma$ with respect to the vertex of the fast nullcline parabola. This means that the neuron state ultimately depends on the magnitude of the external bias current σ . If the line $x_n = \sigma$ intersects the stable branch of the fast nullcline N_s , there is a global fixed point of the two-dimensional map, corresponding to the resting state. At the threshold depolarization $\sigma = \sigma_{th}$, the fixed point loses stability through Neimark–Sacker bifurcation [11], closely related to the saddle-node bifurcation of the fast submap. Above σ_{th} the slow nullcline intersects N_u , and the map yields chaotic series of bursts. One can trace the typical bursting orbit of the phase point, which shows hysteresis between the resting and chaotic attractor states. When $x_n < \sigma$ (inter-burst intervals), the phase point moves in direction of slow variable increase, traversing multiple quasi-steady states μ -close to N_s . For $x_n > \sigma$ (bursts), the phase point exhibits rapid oscillations corresponding to spikes within a burst, as it drifts in the direction of the slow variable decrease toward $\gamma = \gamma_{cr}$.

After analyzing the isolated neuron behavior, we consider the equations describing a pair of Rulkov map neurons coupled via reciprocal chemical synapses:

$$\begin{aligned} x_{i,n+1} &= \frac{\alpha}{1+x_{i,n}^2} + y_{i,n} - g_c(x_{i,n} - \nu) \frac{1}{1 + exp(-k(x_{j,n-\tau} - \theta))}, \\ y_{i,n+1} &= y_{i,n} - \mu(x_{i,n} - \sigma), \end{aligned}$$
(2)

where the indexes i, j = 1, 2 ($i \neq j$) specify the postsynaptic and presynaptic neurons, respectively. For simplicity, the synapses are taken to be symmetrical so that no index dependence of parameters g_c , v, k, θ and τ is indicated. The synaptic current enters only the fast submap and follows the fast threshold modulation model [3,31,35], whose synaptic dynamics, though introduced in continuous time models, can easily be adapted to interacting Rulkov map neurons [22]. The synaptic weight g_c corresponds to maximum aggregate conductance of postsynaptic ion channels, while the reversal potential v determines the character of the synapse. If v is smaller (larger) than postsynaptic membrane potential values, the synaptic current has hyperpolarizing (depolarizing) effect, making the synapse inhibitory (excitatory). Most importantly, the effects of processes in synaptic transmission are included by the presynaptic potential dependence $x_{j, n-\tau}$ on time delay τ , that is expressed in units of map iteration steps.

Instead of hard-threshold Heaviside function, as in previous papers on interacting Rulkov model neurons [8,35], we adopt a softer sigmoid function, with the gain parameter k and θ defining the threshold behavior. Taking after a possible way of interpreting the role of k within biological context [37], we consider the gain parameter in limits of low and high values. The $k \sim 1$ case yields the graded synaptic transmission model [38], appropriate for the description of central pattern generators, while for $k \gg 1$ one obtains the fast threshold modulation model [31], common for the majority of chemical synapses in the brain [29,30]. Though in the limit of large $k, k \gg 1$, sigmoid approaches the Heaviside function, there is an important distinction between them. For the sigmoid, the presynaptic potential affects the amplitude of synaptic current, thereby explicitly influencing the postsynaptic potential $x_{i,n}$, as opposed to Heaviside function, where presynaptic potential determines the instant of synaptic activation, but the changes in $x_{i,n}$ are left to self-interaction.

Due to synaptic model complexity (nontrivial dependence on presynaptic neuron state and the time delay), we cannot follow the line of analysis from [35], that reduces the coupling effects to geometrical changes in the phase plane of a non-interacting neuron. Likewise, the additional irregularity in series of both the fast and slow variables also prevents us from



Fig. 2. Time series of the fast variables x_1 and x_2 for the two interacting neurons, with synaptic parameters $g_c = 0.2$, v = -1.8, and $\sigma = -0.9$. The chaotic evolution makes the fast variable cross-correlation a plausible measure of mutual synchronization behavior.

introducing the proper phase variable in analogy to the one presented in earlier papers, where almost regular slow variable saw-tooth oscillations [13] or the nearly regular oscillation cycles of the averaged potential in a larger network [39] were observed. The chaotic map dynamics and the emergent phenomena keep the neurons from establishing (or maintaining) any particular synchronization regime for most of the intrinsic and synaptic parameter values under arbitrary initial conditions, as confirmed by the typical time series of the neuron pair, shown in Fig. 2. Therefore, we proceed by determining the cross-correlation of membrane potentials R_{x_1,x_2} :

$$R_{x_1,x_2} = \frac{[\langle \mathbf{x}_1 \mathbf{x}_2 \rangle] - [\langle \mathbf{x}_1 \rangle] [\langle \mathbf{x}_2 \rangle]}{\sqrt{\left([\langle \mathbf{x}_1^2 \rangle] - [\langle \mathbf{x}_1 \rangle^2]\right) \left([\langle \mathbf{x}_2^2 \rangle] - \left[\langle \mathbf{x}_2 \rangle^2\right]\right)}},\tag{3}$$

where the angled brackets denote averages over long time series of 50,000 map iterations, and the squared brackets refer to averaging over an ensemble of 200 trials. R_{x_1,x_2} may be considered as an appropriate numerical diagnostics for mutual synchronization, if the trials cover a sufficiently large set of neuron initial conditions [35].

For the reasons provided in Introduction, the neurons are taken to be identical, with $\alpha = 4.15$, so the autonomous dynamics would yield chaotic series of bursts. The neurons receive the same amount of excitation σ , confined to interval $\sigma \in [-1.6, -0.6]$, with the values close to the upper boundary bringing the neurons at the onset of tonic spiking. Throughout the paper, the synaptic parameter θ is set to $\theta = -1.4$, a value easily reached by the bursting neurons. The values of *k* characterizing the soft and hard-threshold-like synaptic behavior are taken to be k = 5 and k = 50, respectively. For each of the motifs we choose a pair of synaptic weights at which principal differences between coupled neuron dynamics regimes may be found. The cross-correlation dependence on σ at fixed g_c is explored for both the excitatory and inhibitory synapses, characterized by v = -1.4 and v = -1.8, respectively. Though the joined effects of g_c , v and σ may effectively alter the character of synapse [35], we rely here on a simple "phenomenological" rule for choosing v: the typical excitatory (inhibitory) synapse should at low σ and g_c favor the prevailing phase (anti-phase) burst synchronization of coupled neurons. Finally, we consider the time delays ranging from 1 to approximately 50 iteration time steps.

In the next section, we present the results of numerical analysis with the described set of neuron and synaptic parameters applied, especially highlighting the mechanisms behind synchronization behavior that cannot be attributed to autonomous neuron dynamics, but solely to the effects of coupling.

3. Results

We examine different synchronization regimes arising with respect to variation of the intrinsic and coupling neuron parameters. The occurrence of and transitions between different synchronization regimes can be characterized by the dependence of the fast variable x_i cross-correlation R_{x_1,x_2} on the external bias current σ .

The selected parameter interval $\sigma \in [-1.6, -0.6]$ admits various bursting modes, ranging from the very short bursts, separated by long inter-burst intervals, to very long bursts, at the onset of tonic spiking. We consider the motifs with reciprocal inhibitory or excitatory synapses, depending on the values of the synaptic reversal potential v. For both types of synapses, the shape of the cross-correlation function strongly depends on synaptic weight g_c . We noticed that the influence of synaptic time delay τ increases with g_c , which is demonstrated by determining the families of cross-correlation curves for different values of τ . Finally, it is also asserted how the form of the obtained families of curves depends on the choice of the synaptic gain parameter k.



Fig. 3. Families of cross-correlation curves over σ in case of the double inhibitory synapses for different values of τ . (a) At $g_c = 0.2$, the cross-correlation remains below zero. (b) At $g_c = 0.5$ there is an interval of σ where cross-correlation rises sharply.



Fig. 4. The fast variable time series of the two neurons (the consecutive map iterates are connected by black and red lines, respectively) coupled via inhibitory synapses with the time delay $\tau = 10$. The upper row corresponds to $g_c = 0.2$, and the lower row to $g_c = 0.5$. (a) At $\sigma = -1.4$ there is only partial antiphase synchronization. (b) At $\sigma = -0.8$ anti-phase synchronization inflection point ($\sigma = -0.9$), we observe simultaneous oscillation deaths as the emergent phenomena. Oscillation deaths give rise to mutual synchronization. (d) At higher σ ($\sigma = -0.6$), sequences of linked oscillation deaths begin to occur, accompanied by another emergent phenomenon: one of the neurons may enter the hyperpolarized state, while the other may not.

A more detailed analysis is presented for the inhibitory motif, as it is neurobiologically more important [4,8] than the excitatory one. In particular, for the inhibitory synapses we consider the cases of weak and moderate synaptic weights, $g_c = 0.2$ and $g_c = 0.5$, with the corresponding dynamical regimes conditionally termed weak and strong coupling regimes, respectively. In the σ -interval up to the inflection point in the cross-correlation graphs (see Fig. 3a and b), the system behavior is dominated by the effects independent of g_c and weakly dependent on τ .

At small values of σ , burst intervals are short compared to the inter-burst intervals. Though the beginning of one neuron's burst matches the beginning of the silent phase of the other, one can not accomplish the full anti-phase synchronization in this dynamical regime. Due to the overlap of silent intervals, the cross-correlation takes negative, but small values. The increase of stimulus current σ gives rise to the number of spikes within a single burst, which leads to the lengthening of burst intervals and the subsequent shrinking of inter-burst intervals. It is noticeable that the cross-correlation value decreases, since the overlap between the silent intervals diminishes with increasing σ . This case corresponds to the fast variable time series of the neuron pair shown in Fig. 4a. Traversing the σ interval to the inflection point from the left, the neurons enter the regime of complete anti-phase synchronization. From the inflection point onwards, with the increase of σ , there arise significant differences between the cases $g_c = 0.2$ and $g_c = 0.5$.

For $g_c = 0.2$, at larger σ the anti-phase synchronization is suppressed due to lengthening of the burst intervals. The lengthening is related to the spike adding phenomenon, that causes the increasing number of overlaps between bursting phases in the time series (Fig. 4b) of the two neurons. The intermittent break-up of anti-phase burst synchronization may be viewed as manifestation of phase slipping [13,35,40]. At the upper end of the considered σ -interval, the neurons are brought to the onset of tonic spiking. Then it becomes impossible to achieve anti-phase synchronization, but the coincident spikes within the prolonged bursts also fail to produce positive cross-correlation.

At larger values of g_c , the sudden increase of cross-correlation above the inflection point is influenced by the emergent phenomena, that cannot be readily predicted from the dynamics of isolated neurons. In particular, in the time series of the neuron pair we observe the simultaneous oscillation deaths [36,41,42], whereby neurons undergo transition from the bursting regime to the unstable excitable states (Fig. 4c). One may ask about the factors influencing the position of the potential in the state corresponding to oscillation death relative to the resting state level. For the models where the transition from the spiking activity mode to oscillation death is considered, the death state and the resting state potentials have been found to match [30]. However, a different picture emerges for the neurons undergoing transition from bursting to oscillation death. Namely, it is for the Hindmarsh-Rose bursting neurons, presenting a more realistic dynamics as compared to the model of Rulkov map, that the depolarized oscillation death state, akin to the one in this paper, has already been observed [42]. The other factor behind the depolarization may be model-specific, in relation to multi-stability in its phase space, since the different attractors regulate the switching between bursting and resting (the two fixed points in the left of Fig. 1a), and the transition to oscillation death (the third fixed point, located in the right of Fig. 1a). Leaving the excitable states may take place under two different scenarios: either by the neurons settling together in quasi-steady silent states or by one of the neurons ending up in the hyperpolarized state. The hyperpolarized states are distinguished from the resting states by the substantially lower values of the fast variable x_i . Along with the oscillation deaths, the occurrence of hyperpolarized states is the second emergent phenomenon characterizing the dynamics of coupled neurons.

The actual transition from the oscillatory regime to excitable state follows the sequence of inverse Hopf bifurcations that will be analyzed elsewhere. Here, we note that the increase of cross-correlation at larger σ is caused by the neurons residing together in excitable and resting states, as their phase points move along the stable branch of the parabola in Fig. 1b. This effectively relates the increase of cross-correlation with the enhancing number of simultaneous oscillation deaths in the time series of the two neurons. For instance, observing the sudden rise of cross-correlation at $\tau = 10$, for $\sigma = -1.1$, we found one oscillation death, and for $\sigma = -0.8$, there are 21 in the time series comprising 50,000 iteration steps. Further increasing σ , the cross-correlation value remains approximately constant, since the number of oscillation deaths does not rise, whereas the sequences of linked oscillation deaths emerge (Fig. 4d).

We noticed that with increasing τ (the family of curves in Fig. 3b) at fixed σ , the cross-correlation reduces due to two effects. Firstly, the number of deaths becomes lower as τ increases. Secondly, larger τ causes the differences in respective times of entering the excitable and resting states to appear. This discrepancy is related to different ways in which the two neurons undergo transition to excitable states. Both of the before mentioned effects are especially manifested at $\tau = 40$. There the increase of cross-correlation is very mild and is accompanied by moderate rise in number of oscillation deaths.

In case of the double-excitatory synapses, we also consider the two values of synaptic weight ($g_c = 0.35$ and $g_c = 0.5$), for which the families of cross-correlation curves display substantial differences. At $g_c = 0.35$ (Fig. 5a), the general trend of decreasing cross-correlation with the increase of σ is observed. The underlying mechanism is analogous to the one described for the inhibitory motif up to the inflection point of the cross-correlation graphs.

At low σ , it is easy to achieve synchronization, since the burst intervals are shorter. As the burst intervals increase, occasional desynchronization between them begins to appear, which may be interpreted as phase slipping. The main difference between the cases of small and large τ stems from the fact that larger delays cause the alternation between phase and antiphase burst synchronization in the time series of two neurons. At $\tau = 40$, even the negative values of cross-correlation occur in the interval $\sigma \in (-1, -0.6)$. The trend of gradual increase of cross-correlation in the interval $\sigma \in (-1, -0.6)$ corresponds to lengthening of burst intervals and the arrival at the onset of tonic spiking.



Fig. 5. Families of cross-correlation curves over σ for excitatory coupling with different synaptic time delays τ . (a) For $g_c = 0.35$, the cross-correlation remains positive at small τ . (b) For $g_c = 0.5$, we observe the boundary value $\tau = \tau_{mR}$. Below it, cross-correlation rises at each σ , while above it decreases for small σ and increases for the large σ .

For $g_c = 0.5$ (Fig. 5b), we split the analysis in two parts, according to the behavior of cross-correlation with increasing τ at fixed values of σ . We identified two diverse trends, that correspond to bias current intervals $\sigma \in (-1.6, -0.9)$ and $\sigma \in (-0.9, -0.6)$. In the first interval, the cross-correlation rises till $\tau_{mR} \approx 19$, and declines afterwards. The existence of τ_{mR} is the clear result of coupling, since it is absent at low values of synaptic weight, whereas it appears beyond $g_c \approx 0.4$. Enhancing τ till $\tau = \tau_{mR}$ gives rise to the number of simultaneous oscillation deaths in the neurons' time series. Above τ_{mR} ($\tau > \tau_{mR}$) cross-correlation reduces on two grounds. On one hand, the number of oscillation deaths decreases, while on the other hand, significant desynchronization emerges as neurons leave the resting or hyperpolarized states. The number of hyperpolarized states increases with τ due to the enhanced number of sequences with linked oscillation deaths, whereby one neuron undergoes transition to oscillation death, and the other settles in the hyperpolarized state (this may be viewed in analogy with behavior presented in Fig. 4d). In the second interval $\sigma \in (-0.9, -0.6)$, at fixed σ , we observe the steady increase of cross-correlation with larger τ . As time delay increases at given σ , the number of hyperpolarized states reduces, while the number of oscillation deaths remains approximately the same. On the other hand, at moderate τ , the number of oscillation deaths reduces of time delay ($\tau = 40$) the increase of σ is accompanied by the rise of the number of simultaneous oscillation deaths. This is similar to the earlier observed behavior of the inhibitory neuron motif at $\tau = 40$.

So far the analysis was concerned with neurons coupled via the soft-threshold synapses, characterized by the gain parameter k = 5. As indicated in the beginning of this section, we also address the dynamics of the observed motifs in the limit of hard-threshold synapses and point to differences as compared to the soft-threshold ones. The families of curves $R(\sigma)$ for different values of τ in cases of weak and strong couplings are presented for the gain parameter k = 50.

In the weak coupling regime of the inhibitory motif (Fig. 6a), one notes that at small σ enhancing k results in the significant decrease of R. This effect is related to bursting periodicity, established at both of the neurons, bringing down



Fig. 6. Families of cross-correlation curves $R_{x_1,x_2}(\sigma)$ for different values of τ in the case of the hard-threshold synaptic behavior (k = 50). The upper row presents the results for the inhibitory, and the lower row for the excitatory motif. (a) For $g_c = 0.2$ the form of the curves remains similar to the one with k = 5, though the values at small σ decrease. (b) For $g_c = 0.5$, compared to the motif with soft-threshold synapses, the strong dependence of R on τ at large σ is lost. (c) At $g_c = 0.35$, the occurrence of oscillation deaths causes the dynamics of the excitatory motif to become significantly altered in comparison to the one for small k. (d) At $g_c = 0.5$ the increase of k has little effect on the cross-correlation curves, except for the large τ at small σ .

the possibility of the accidental burst overlaps. Thus, increasing *k* at low σ in this case mainly contributes the anti-phase burst synchronization to be more easily achieved. For the stronger coupling ($g_c = 0.5$), similar behavior at small σ may be found (see Fig. 6b). However, in contrast to the motif with the soft-threshold synapses, for k = 50 the influence of τ on the cross-correlation value diminishes when σ is increased. It turns out that enhancing *k* causes the aforementioned discrepancy between the respective times of the neurons entering the excitable or the resting state to become reduced.

For the excitatory motif at $g_c = 0.35$, unlike the case with the soft-threshold synapses, families of cross-correlation curves in the hard-threshold limit (Fig. 6c) for $\tau > 0$ lie above the curve $\tau = 0$. Such dependence may be explained by the fact that large k = 50 allows the time delay to bring about the oscillation deaths, even at small synaptic weights. The k and τ interplay for the hard-threshold synapses causes greater similarity between system behaviors at weak and strong coupling (Fig. 6c and d, respectively) regimes than for k = 5. With this in mind, the comparison of Figs. 5a and 6c also suggests that increasing keffects more significantly the synchronization regimes at small, than at large g_c (compare Figs. 5b and 6d). For the stronger synapses, however, the only difference worth mentioning occurs for the high values of τ and small σ , as in the hard-threshold limit the increase of τ does not lead to the reduction in the number of oscillation deaths and the subsequent decrease of R.

4. Summary and discussion

We have studied a pair of identical Rulkov map neurons coupled via reciprocal chemical synapses. The Rulkov map parameters have been selected to set the neurons in the bursting dynamical mode. The parameters of the chemical synapses are taken to be symmetrical, with the threshold behavior controlled by the sigmoid form function. The implemented threshold function can be modified in such a way to include both the soft-threshold-like behavior similar to graded synaptic transmission, and the hard-threshold-like behavior, related to fast threshold modulation model. In supplement to earlier considerations, we took into account the synaptic time delay, that is inherent to chemical synapses.

With the map being chaotic in its own right, we numerically determine the cross-correlation of fast variables, where the appropriate ensemble averaging eliminates the dependence on initial neuron states. The cross-correlation is used to characterize the synchronization states of the neuron pair with respect to external current σ , controlling the autonomous neuron dynamics, as well as the synaptic weight g_c , time delay τ and gain parameter k. For both the inhibitory and excitatory synapses, we observe that the families of cross-correlation curves over σ for different values of τ significantly differ at small and moderate g_c . However, at each of the chosen g_c values, for low σ , the phase (anti-phase) burst synchronization is achieved in cases of excitatory (inhibitory) synapses.

The more specific phenomena are presented first for the inhibitory, and then for the excitatory motif with the soft-threshold synapses (case k = 5). For the former, with enhancing g_c , the differences in cross-correlation behavior above the inflection point (compare Fig. 3a and b) arise due to the prevailing effects of the emergent phenomena. The highly positive cross-correlation at $g_c = 0.5$ is a consequence of the simultaneous oscillation deaths that the neurons leave together, passing through the resting states. The oscillation death events observed here may be put in context with the analogous phenomena of spike death [43], firing death [44] and population death [45], obtained for the more realistic neuron models. The other emergent phenomenon we encounter has the effect of reducing the cross-correlation: coming out of oscillation death, one neuron enters the hyperpolarized state, while the other does not. The hyperpolarized states appear at larger σ and are related to the linked sequences of simultaneous oscillation deaths.

The sharp rise of cross-correlation is a consequence of the increased number of the simultaneous oscillation deaths in the neurons' time series. It is interesting to note that R_{x_1,x_2} "jumps" abruptly once it is crossed from the instantaneous to chemical synapses with the included time delay. Further increasing σ , the number of oscillation deaths remains approximately constant, but they reorganize to longer sequences with more hyperpolarized states. Therefore, at fixed τ and arbitrary σ , there is the constant cross-correlation value, whereas with increasing τ and fixed σ , the cross-correlation R_{x_1,x_2} reduces.

In case of double-excitatory synapses, we observe the more complex interplay of σ and τ parameters than for the inhibitory synapses. At higher σ , increasing τ gives rise to R_{x_1,x_2} , which may be attributed to the larger number of oscillation deaths. At lower σ , there is a boundary value τ_{mR} , below which R_{x_1,x_2} rises, while above it begins to decay. This behavior is related to the increasing number of oscillation deaths for $\tau < \tau_{mR}$, followed by its decrease for $\tau > \tau_{mR}$.

In addition, it is considered how introducing the hard-threshold synapses (case k = 50) effects the behavior of both of the motifs. For the inhibitory motif, in the weak coupling regime the increase of k is found to favor the anti-phase burst synchronization at small σ , while in the strong coupling regime at high σ the effects of τ become suppressed. For the excitatory motif, it is interesting that the transition from soft- to hard-threshold synaptic behavior brings about the increase of synchronization with τ , mostly due to the neurons residing together in states that occur as a consequence of the oscillation death.

We emphasize that the applied model is phenomenological by nature, so that the considered variables, especially the slow one, have not been attributed explicit biological interpretation. However, a possible insight in this context can be gained by comparing some of the presented dynamical regimes with the experimental results. Namely, the time series embedding the oscillation deaths have been observed for neurons with excitability modified by antagonists of the delayed potassium outward current [46,47], by administering cholinergic agonists [48] or otherwise [49]. The first suggests that for describing the neurons with unmodified excitability one should probably look more closely into modeling the hyperpolarization mechanism, including an additional hyperpolarization current [22], or even considering the third independent

variable. Though some authors have recently indicated additional biological implications of the oscillation death phenomenon in hybrid systems [50,51], with the data currently known we find it more appropriate to compare this and the other phenomena discussed with those obtained for the conductance-based models (Hodgkin-Huxley and Hindmarsh-Rose model). The differences in behavior of the respective motifs that stem from the discrete or continuous nature of these models were already pointed to [6], but not for the neurons coupled via chemical synapses with the time delay. To a certain extent, it is possible to draw a parallel between the results for the pair of Hodgkin-Huxley neurons and our results concerning the obtained dynamical regimes, including the oscillations deaths. However, for the Hodgkin-Huxley neurons, the oscillation deaths are confined to smaller domains of the parameter space, as the multi-stability intervals of different dynamical regimes are rare [30,52]. Here such intervals may be broader, since the interplay of map chaoticity and higher synaptic weights enables the neurons to pass beyond the external crisis barrier, entering the multi-stable regime between the upper branch of the parabola in Fig. 1b and the fixed point to the right in Fig. 1a. Greater similarity to the phenomena we report may be found for the Hindmarsh-Rose neurons coupled via chemical synapses with the soft-threshold function [42,50,53,54]. In [42], depending on the coupling strength, three stationary synchronization regimes were observed, including synchronization related to oscillation death and the burst phase synchronization. On the other hand, in our model, the time series of the neuron pair show alternation between the burst phase synchronization and the exact synchronization to states occurring due to the oscillation death.

It would be worth exploring how the parameter inhomogeneity effects the possible synchronization regimes of the considered motifs. Also, the present research could be extended to include larger motifs and their interactions.

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