How synaptic function controls critical transitions in spiking neuron networks: insight from a Kuramoto model reduction

Lev A. Smirnov, Vyacheslav O. Munyayev, Maxim I. Bolotov, Grigory V. Osipov, and Igor Belykh

Department of Control Theory, Lobachevsky State University of Nizhny Novgorod, 23 Gagarin Avenue, Nizhny Novgorod, 603022, Russia

Correspondence*:
ibelykh@gsu.edu

ABSTRACT

The dynamics of synaptic interactions within spiking neuron networks play a fundamental role in shaping emergent collective behavior. This paper studies a finite-size network of quadratic integrate-and-fire neurons interconnected via a general synaptic function that accounts for synaptic dynamics and time delays. Through asymptotic analysis, we transform this integrate-and-fire network into the Kuramoto-Sakaguchi model, whose parameters are explicitly expressed via synaptic function characteristics. This reduction yields analytical conditions on synaptic activation rates and time delays determining whether the synaptic coupling is attractive or repulsive. Our analysis reveals alternating stability regions for synchronous and partially synchronous firing, dependent on slow synaptic activation and time delay. We also demonstrate that the reduced microscopic model predicts the emergence of synchronization, weakly stable cyclops states, and non-stationary regimes remarkably well in the original integrate-and-fire network and its theta-neuron counterpart. Our reduction approach promises to open the door to rigorous analysis of rhythmogenesis in networks with synaptic adaptation and plasticity.

1 INTRODUCTION

Cooperative rhythms play a pivotal role in brain functioning. Fully or partially synchronized oscillations, observed across various frequency bands, underlie fundamental processes such as perception, cognition, and motor control Churchland and Sejnowski (1992); Mizuseki and Buzsaki (2014); Kopell and Ermentrout (2000). Extensive research has focused on the emergence of cooperative rhythms in networks of spiking and bursting neurons, encompassing synchronization Kopell and Ermentrout (2000); Brunel (2000); Börgers and Kopell (2003); Somers and Kopell (1993); Izhikevich (2007); Belykh et al. (2005); Ermentrout and Terman (2010), partial and cluster synchronization Achuthan and Canavier (2009); Shilnikov et al. (2008); Belykh and Hasler (2011); Schöll (2016);
Networks of spiking neurons with fast synaptic connections are often modeled via pulsatile on-off coupling, which sharply activates upon the arrival of a spike from a pre-synaptic cell. Such interactions are conveniently represented by networks of quadratic integrate-and-fire (QIF) models, particularly suitable for large-scale simulations and analysis of cooperative dynamics \cite{Gerstner2002}. The macroscopic dynamics of QIF networks have received extensive attention through the reduction to low-dimensional model descriptions, especially in the thermodynamic limit of infinite-dimensional networks \cite{Montbrió2015, Pazó2016, Devalle2017, Esnaola-Acebes2017, Devalle2018, Schmidt2018, Pietras2019, Montbrió2020, Clusella2022, Clusella2024, Ratas2018, Pyragas2022, Pyragas2023, Coombes2023}. Notably, \cite{Montbrió2015} derived exact macroscopic equations for QIF networks, uncovering an effective coupling between firing rate and mean membrane potential governing network dynamics. \cite{Pietras2023} offered an analytical description of QIF network macroscopic dynamics, extending beyond the Ott-Antonsen ansatz \cite{Ott2008} and exploring various fast synaptic pulse profile choices. The impact of synaptic time delay on the collective dynamics of integrate-and-fire networks with sharply activated synaptic coupling, modeled by the Dirac delta function, was also extensively explored \cite{Ernst1995, Devalle2018, Ratas2018, Pyragas2022, Pyragas2023}. In particular, \cite{Devalle2018} reduced a QIF model with synaptic delay to a set of firing rate equations to analyze the existence and stability of partially synchronous states. \cite{Ratas2018} employed a Lorenzian ansatz to characterize macroscopic oscillations of a QIF network with heterogeneous time-delayed delta function synapses. However, there is a lack of analytical studies on the role of slower synaptic activation, potentially in the presence of time delays, in controlling critical phase transitions in QIF networks. Nevertheless, since the seminal paper by \cite{VanVreeswijk1994}, it has been recognized that slow inhibitory and excitatory synapses can reverse their roles, with slow inhibition favoring synchronization \cite{Golomb1993, Terman1998, Elson2002}. While predicting the exact rates of synaptic activation inducing such critical transitions in conductance-based spiking models may be challenging, analytically tractable QIF networks offer promising avenues for such exploration.

Toward this goal, this paper investigates a finite-size network of QIF neurons globally connected via a general kernel function that governs both synaptic activation and synaptic time delay. We analytically illustrate how the shape of the kernel function impacts neuron interaction, significantly altering the microscopic and macroscopic behavior of QIF networks representing Type I neuron populations. This is achieved by reducing QIF networks and their phase analog, theta neuron networks, to the Kuramoto-Sakaguchi (KS) model. Here, oscillator frequencies, coupling strength, and the Sakaguchi phase lag parameter are determined by the pulse profile’s first and second terms in the Fourier expansion. We conduct this reduction under the weak coupling assumption, utilizing the intermediate step of representing the QIF network as a generalized Winfree model, subsequently reduced to the KS model.

In our recent study \cite{Munyayev2023}, we elucidated the qualitative connection between the dynamics of QIF networks incorporating synaptic dynamics and neuronal refractoriness, and the second-order Kuramoto model with high-order mode coupling. Here, we employ multi-scale analysis to derive exact relationships between the QIF network with an arbitrary synaptic activation function...
and the KS model. Specifically, we establish explicit conditions on the parameters of the general kernel function that lead to critical transitions, determining whether the coupling is attractive or repulsive. Consequently, these conditions dictate the emergence of stable synchronization or nonstationary generalized splay states Berner et al. (2021b) and cyclops states Munyayev et al. (2023). Our analysis reveals alternating stability regions for network synchronization, dependent on both the (slow) synaptic activation and time delay. With some important caveats, this finding can be interpreted as an analogous stability criterion for synchronization in time-delayed phase oscillator networks Earl and Strogatz (2003).

Our approach serves as a connecting link between two alternative methodologies for describing macroscopic dynamics: QIF networks and theta neurons, and Winfree-type models Pazó and Montbrió (2014); Gallego et al. (2017); Montbrió and Pazó (2018); Pazó et al. (2019); Pazó and Gallego (2020); Manoranjani et al. (2021); Bick et al. (2020). Our KS model reduction of the generalized Winfree model with a general synaptic activation function can be seen as an extension of the work Montbrió and Pazó (2018), where a two-population Kuramoto model was derived from a network of Winfree oscillators featuring a feedback loop between fast excitation and slow inhibition.

The structure of this paper is outlined as follows. Section 2 presents the QIF network model, its theta-neuron equivalent, and the general synaptic activation function. Section 3 details transforming the theta-neuron model into the generalized Winfree model. We expand the pulse profile as a Fourier series and further simplify the model to the KS model using weak coupling-enabled averaging techniques. Section 4 focuses on a specific example of synaptic activation, presenting a class of kernel functions. We establish exact conditions determining whether the synaptic coupling is attractive, promoting synchronization, or repulsive, favoring splay and cyclops states. Section 5 offers numerical validation of the derived conditions and presents a comparison between the dynamics of the QIF network, the theta-neuron model, and the reduced KS model. We demonstrate that the KS model accurately predicts firing rates and times, capturing the emergence of synchronization, weakly stable cyclops states, and non-stationary regimes. Section 6 contains concluding remarks and discussions.

2 THE GENERAL QIF NETWORK AND ITS THETA-NEURON REPRESENTATION

Physiologically, excitable neurons are commonly categorized into two types. We focus here on Type I neurons, a group encompassing cortical excitatory pyramidal neurons. When subjected to a sufficiently large input stimulus, these neurons exhibit action potentials at an arbitrarily low rate, signaling the disappearance of a resting state through a saddle-node bifurcation. The canonical model used to describe Type I neurons is the QIF neuron model, which characterizes neurons’ dynamics near the spiking threshold Izhikevich (2007).

This study investigates a globally coupled network of $N$ QIF neurons interacting through chemical synapses. Each neuron’s microscopic state is characterized by its individual membrane potential $v_n$, governed by the following ordinary differential equation Izhikevich (2007):

$$\begin{align*}
\dot{v}_n &= v_n^2 + \eta_n + \kappa S(t) & \text{if } v_n < v_{th}, \\
v_n &= v_r & \text{if } v_n \geq v_{th}.
\end{align*}$$

(1)

Here, $\eta_n$, $n = 1, 2, \ldots, N$ represents external constant currents applied to neurons, $\kappa$ is a common synaptic weight controlling the total strength of synaptic inputs, and $S(t)$ is a time-varying input...
drive. When the membrane potential $v_n$ of the $n$th neuron reaches the threshold value $v_{th}$, the neuron generates a spike, and its voltage resets to $v_r$. In the absence of the input drive ($S(t) = 0$), the intrinsic applied current $\eta_n = 0$ places the corresponding $n$th neuron at a saddle-node bifurcation, marking the onset of periodic firing. Thus, if $\eta_n < 0$, the neuron is in the excitable regime, while if $\eta_n > 0$, it is in the oscillatory regime.

The last term on the right-hand side of (1) represents synaptic interactions characterized by the coupling strength $\kappa$ of the global synaptic drive. In general, the mean population synaptic activity $S(t)$ can be expressed by the following recurrent input equation:

$$S(t) = \frac{1}{N} \sum_{n'=1}^{N} \sum_{m \nonumber \leq n' < t} \int_{-\infty}^{t} d\tilde{t} G((t - \tilde{t}) / \tau) \delta (\tilde{t} - t_{n'}^m).$$ (2)

This equation accounts for relaxation processes and describes a specific type of neuron activation and its sensitivity to stimuli from other cells, including signal duration and post-spike latency. Here, $t_{n'}^m$ denotes the time of the $m$th spike of the $n'$th neuron, $\delta(t)$ represents the Dirac delta function, and $G(t/\tau)$ is the normalized synaptic activation caused by a single presynaptic spike with a time scale $\tau$. Notably, the integral transformation with the kernel $G(t/\tau)$ acts as a low-pass filter.

The QIF-neuron model (1) describes the membrane potential $v_n$ and operates as a hybrid dynamical system, incorporating instantaneous resets to a base value $v_r$ upon spike emission. While this formulation provides a direct physical interpretation, discontinuities can pose challenges for certain applications. Fortunately, a smooth change of coordinates exists, transforming the QIF-neuron dynamics into a space where the membrane potential $v_n$ is represented by a phase variable $\theta_n$ on the unit circle. This representation captures nonlinear spike-generating mechanisms of Type I neurons, ensuring smooth solutions within a compact domain. In the limit $v_{th} = -v_r \to \infty$, the transformation $v_n(t) = \tan (\theta_n(t)/2)$ [Ermentrout and Kopell, 1986] converts the membrane potential description of the QIF-neuron model (1) into a canonical theta-neuron model of a population of Type I neurons coupled by an excitatory or inhibitory synaptic drive. In this case, each neuron’s dynamics is governed by the following equation:

$$\frac{d\theta_n}{dt} = (1 - \cos \theta_n) + \eta_n (1 + \cos \theta_n) + \kappa (1 + \cos \theta_n) S(t),$$ (3)

where $n = 1, \ldots, N$ represents the index of the $n$th neuron, and its state is characterized by the phase angle $\theta_n$. We assume that the constant excitability parameters $\eta_n$, akin to fixed input currents, have slightly different values across the network elements. Moreover, we consider $\eta_n > 0$, placing each neuron in an oscillatory regime indicative of periodic spiking. A neuron is deemed to spike or produce an action potential when $\theta_n$ crosses $\pi$ while increasing. Consequently, in addition to a common external input $\eta_n$, each cell receives stimulation from other cells. Thus, the neurons are recurrently coupled via synaptic current $S(t)$.

The last term on the right-hand side of (3) accounts for chemical interactions among neurons. The coupling strength $\kappa$ is assumed to be uniform across all neurons. We model the synaptic activity
$S(t)$ acting on a neuron with the following expression:

$$S(t) = \int_{-\infty}^{t} dt' G\left(\frac{t - t'}{\tau}\right) \left[\frac{1}{N} \sum_{n=1}^{N} P_\nu (\theta_n(t'))\right], \quad (4)$$

where the function

$$P_\nu(\theta_n) = p_\nu \left(1 - \cos \theta_n\right), \quad \nu \in \mathbb{N} \quad (5)$$
determines the shape of the pulsatile chemical synapse. The positive integer parameter $\nu$ controls the sharpness of $P_\nu(\theta_n)$, with higher values yielding sharper peaks. Note that as $\nu \to \infty$, the smooth profile $P_\nu(\theta_n)$ converges to $P_\infty(\theta_n)$, effectively representing $\delta$-pulses so that

$$P_\nu(\theta_n) \xrightarrow{\nu \to \infty} P_\infty(\theta_n) = 2 C_T(2 \pi (\theta_n - \pi)) = 2 \sum_{k=-\infty}^{+\infty} \delta(\theta_n - \pi - 2\pi k), \quad (6)$$

where $C_T(\cdot)$ denotes the Dirac comb with period $T$. In this limit and under the assumption $v_{\text{th}} = -v_r \to \infty$, the theta-neuron model (3),(4) fully aligns with the QIF-neuron model (1), (2). It is noteworthy that these models exhibit an unconventional characteristic: in contrast to conductance-based models, inhibitory $\delta$-pulse coupling ($\kappa < 0$) promotes synchronization, thus being considered attractive, while excitatory $\delta$-pulse coupling ($\kappa > 0$) is repulsive Izhikevich (2007).

In this work, we primarily focus on the pulse shape defined by (5), originally proposed in Ariaratnam and Strogatz (2001) and widely adopted in recent studies of pulse-coupled phase oscillators O’Keeffe and Strogatz (2016); Pazó and Montbrió (2014); Gallego et al. (2017); Pazó et al. (2019); Bick et al. (2020) and populations of theta neurons Luke et al. (2013); So et al. (2014); Laing (2014, 2015); Montbrió et al. (2015); Pazó and Montbrió (2016); Chandra et al. (2017); Goel and Ermentrout (2002); Bick et al. (2020); Pietras et al. (2023). However, our approach is directly applicable to alternative pulse shapes satisfying common properties such as unimodality, normalization, symmetry, and localization around $\theta_n = \pi$ and considered in previous studies Gallego et al. (2017); Pietras et al. (2023).

In the following, we explore how the shape of the kernel function $G(t/\tau)$, governing synaptic activation, impacts the network’s collective behavior. To tackle this analytically, we will demonstrate that the model (3),(4) consideration can be reduced to a more analytically-tractable KS model. The process involves several key steps: firstly, leveraging the assumption $\eta_n > 0$, we introduce an alternative phase representation for (3),(4). Subsequently, we will derive the KS model of phase oscillators by employing multiple time scale analyses in weak chemical synaptic coupling scenarios.

### 3 DERIVING THE KS MODEL MODEL FROM THE THETA-NEURON MODEL: AN ASYMPTOTIC ANALYSIS

We begin by assuming that each neuron operates within an oscillatory regime in the absence of interaction, i.e., $\eta_n > 0$. Hence, we can employ a dynamical variable transformation:

$$2 \tan \left(\frac{\theta_n}{2}\right) = \Omega \tan \left(\varphi_n/2\right), \quad (7)$$
The coefficients where with respect to hypergeometric function. Specifically, for the first two Fourier series coefficients \( C \), where
derivation and technical intricacies are presented below. Further elaboration on this approach’s representation(8), (9) proves more convenient for subsequent analysis. Thus, we derive the governing model (3),(4), all cells receive constant external inputs \( \eta_n > 0 \), and all units operate within the oscillatory regime, each phase \( \varphi_n \) uniformly rotates in the absence of network interaction. Hence, the representation(8), (9) proves more convenient for subsequent analysis. Thus, we derive the governing equation for the introduced dynamical variables \( \varphi_n \) that may be viewed as the generalized Winfree model, which, in turn, can be reduced to the KS model. Further elaboration on this approach’s derivation and technical intricacies are presented below.

For further analysis, it is convenient to expand the symmetric pulse \( Q_\nu (\varphi_n) \) into a Fourier series with respect to \( \varphi_n \). This expansion takes the following form:

\[
Q_\nu (\varphi_n) = \sum_{\ell=0}^{\infty} Q_{\nu \ell} e^{i \varphi_n}, \quad Q_{\nu \ell} = \frac{1}{2\pi} \int_{-\pi}^{+\pi} d\varphi e^{-i\ell \varphi} Q_\nu (\varphi), \quad Q_{\nu,-\ell} = Q_{\nu \ell} \in \mathbb{R}.
\]  

The coefficients \( Q_{\nu \ell} \) in this series can be expressed analytically as follows:

\[
Q_{\nu \ell} = \frac{\nu \Gamma(\nu + m + \frac{1}{2})}{\Gamma(\nu + \ell + 1)} \left[ \left( \frac{\Omega}{2} \right)^{2(\ell-m)+1} \times 2F_1 \left( \ell + 1, \ell - m + \frac{1}{2}; \nu + \ell + 1; 1 - \frac{\Omega^2}{4} \right) \right],
\]

where \( C_{2\ell}^{2m} \) represents combinations, \( \Gamma(\cdot) \) denotes the gamma function, and \( 2F_1(\cdot, \cdot; \cdot; \cdot) \) is the hypergeometric function. Specifically, for the first two Fourier series coefficients \( Q_0 \) and \( Q_1 \), we obtain:

\[
Q_{\nu 0} = \frac{|\Omega|}{2\pi} 2F_1 \left( 1, \frac{1}{2}; \nu + 1; 1 - \frac{\Omega^2}{4} \right),
\]

\[
Q_{\nu 1} = \frac{4\pi (\nu + 1)}{2F_1} \left[ \frac{\Omega^2}{4} 2F_1 \left( 1, 2; \nu + 2; 1 - \frac{\Omega^2}{4} \right) - (2\nu + 1) 2F_1 \left( 2, \frac{1}{2}; \nu + 2; 1 - \frac{\Omega^2}{4} \right) \right].
\]
Noteworthy, in the limit $\nu \to \infty$, i.e., for $Q_{\infty}(\varphi_n)$, all coefficients in the Fourier series converge to the same value $Q_{\infty} = (-1)^{\ell} |\Omega| / 2\pi$.

We proceed by assuming that the synaptic coupling is weak, allowing us to express it as $2\kappa/\Omega = \varepsilon \kappa$, where $\varepsilon \ll 1$ is a small parameter. Similarly, we assume that the deviations of external inputs $\eta_n$ from the value $\Omega^2 / 4$ are small, i.e., $\eta_n - \Omega^2 / 4 = \varepsilon \Omega \sigma_n / 2$.

These assumptions enable a multiple-time scale analysis. To facilitate this analysis, we introduce a separation of time scales:

$$t_k = \varepsilon^k t, \quad k = 0, 1, \ldots, \infty$$

and represent each phase variable, $\varphi_n(t)$, as an asymptotic series with respect to the small parameter $\varepsilon$:

$$\varphi_n(t) = \sum_{k=0}^{\infty} \varepsilon^k \varphi_n^{(k)} (t_0, t_1, t_2, \ldots).$$

Substituting the series and the Fourier series into (8), (9), and considering the zeroth order in $\varepsilon$, we obtain for $\varphi_n^{(0)}(t_0, t_1, t_2, \ldots)$:

$$\varphi_n^{(0)} (t_0, t_1, t_2, \ldots) = \Omega t_0 + \phi_n (t_1, t_2, \ldots)$$

and, taking into account (16), for $S(t_0, t_1, t_2, \ldots)$ we arrive at

$$S(t_0, t_1, t_2, \ldots) = \tau \int_0^{+\infty} d\xi G(\xi) \left[ \frac{1}{N} \sum_{n'=1}^{N} Q_{n'} \left( \Omega t_0 - \Omega \tau \xi + \phi_{n'} (t_1 - \varepsilon \tau \xi, t_2 - \varepsilon^2 \tau \xi, \ldots) \right) \right. + \left. \varepsilon \varphi_{n'}^{(1)} (t_0 - \tau \xi, t_1 - \varepsilon \tau \xi, \ldots) + \ldots \right] \approx \frac{1}{N} \sum_{n'=1}^{N} \sum_{\ell=-\infty}^{+\infty} G_{\ell} Q_{n'} e^{i\ell \Omega t_0 + i\ell \phi_{n'}(t_1, t_2, \ldots)},$$

where each corresponding complex coefficient $G_{\ell}$ is determined as follows:

$$G_{\ell} = \tau \int_0^{+\infty} d\xi G(\xi) e^{-i\ell \Omega \tau \xi}.$$ 

In (16), the first term $\Omega t_0$ describes the fast, free-running rotation of period $2\pi/\Omega$, while the slow phase drifts induced by synaptic interaction are characterized by the set of slow variables $\phi_n (t_1, t_2, \ldots)$. Hence, $\phi_n (t_1, t_2, \ldots)$ can be considered constant over time scales comparable to the period of the corresponding fast rotation. Consequently, the standard averaging method can be applied to derive the KS model corresponding to (3), (4). Notably, in this case, the Sakaguchi phase shift emerges naturally due to the complex nature of the coefficient $G_{\ell}$.

In accordance with the averaging procedure, the next step of our asymptotic approach involves considering all terms that are $o(\varepsilon)$ and, in the first order in $\varepsilon$, we obtain a set of equations for $\varphi_n^{(1)}(t_0, t_1, \ldots)$. 

Frontiers
To eliminate the secular terms that grow without bounds as $t_0 \to \infty$, we impose the conditions

$$\frac{\partial \phi_n}{\partial t_1} = \sigma_n + \kappa Q_{\nu 0} + \frac{\kappa |G_1| Q_{\nu 1}}{N} \sum_{n' = 1}^{N} \cos \left( \phi_{n'} - \phi_n + \arg (G_1) \right).$$

(19)

This yields a solution for $\varphi^{(1)}_n(t_0, t_1, \ldots)$ without the secular terms. Note that (19) measures the rate of change of $\phi_n(t_1, t_2, \ldots)$ with respect to the slow time scale $t_1$. Finally, taking into account the relation $d\phi_n/dt \approx \varepsilon \partial \phi_n/\partial t_1$, we find that the dynamics of the slow phases $\phi_n(t)$ is approximately described by the KS model:

$$\frac{d\phi_n}{dt} = \omega_n + K \sum_{n' = 1}^{N} \sin \left( \phi_{n'} - \phi_n - \alpha \right),$$

(20)

where

$$\omega_n = \varepsilon \sigma_n + \varepsilon \kappa Q_{\nu 0} = 2 \left( \eta_n - \Omega^2/4 + \kappa Q_{\nu 0} \right)/\Omega,$$

(21a)

$$K = \varepsilon \kappa |G_1| Q_{\nu 1} = 2 \kappa |G_1| Q_{\nu 1}/\Omega,$$

(21b)

$$\alpha = - \arg (G_1) - \pi/2.$$  

(21c)

To determine the unknown parameter $\Omega$, we set the mean value of the intrinsic frequencies $\omega_n$ in the KS model (20) to zero. Hence, the value of $\Omega$ can be found by solving the nonlinear algebraic equation:

$$\langle \eta_n \rangle - \Omega^2/4 + \kappa Q_{\nu 0} (\Omega) = 0.$$  

(22)

This choice of the optimal value of parameter $\Omega$ yields a better quantitative match between the numerical simulation results of the theta-neuron model (3), (4) and the KS model (20), compared to the conventional choice of $\Omega = 2 \sqrt{\langle \eta_n \rangle}$. The numerical comparison between the simulation results of the QIF, theta-neuron, and KS models is given in Sec. 5.

Note that oscillator frequencies $\omega_n$, coupling strength $K$, and the Sakaguchi phase lag parameter $\alpha$ in the KS model (20) are explicitly defined through by the pulse profile’s first and second Fourier series terms $Q_{\nu 0}$ and $Q_{\nu 1}$. This direct dependence on the properties of the synaptic activation enables a straightforward assessment of the role of synaptic interactions in critical phase transitions and dynamics. Specifically, the coupling strength $K$ directly reflects the impact of synaptic activation on critical phase transitions and the synchronization behavior of the neuron population. In particular, determining whether the coupling in the theta-neuron model (3), (4) is attractive or repulsive presents a challenge due to the complexity of the system, whereas the KS model (20) makes this process straightforward. In the subsequent section, we delve into this process, focusing on a particular synaptic activation profile.
4 THE ROLE OF SYNAPTIC PROFILE: A COMBINED EFFECT OF ACTIVATION, DEACTIVATION, AND TIME DELAYS

To demonstrate the important effects arising from the specific selection of the shape of a “low-pass filter” in synaptic activation, we examine the following class of kernel functions:

\[
G\left(\frac{t}{\tau}\right) = \frac{(t/\tau)^q e^{-t/\tau}}{q! \tau} H\left(\frac{t}{\tau}\right),
\]

(23)

where \(H(t/\tau)\) is the Heaviside step function and \(q\) represents an integer parameter pivotal to the network dynamics, as will become evident subsequently. The chosen kernel corresponds to the Green’s function for a non-homogeneous linear differential equation of order \((q + 1)\). In this case, the equation for the mean synaptic activity \(S(t)\) is generated by the \((q + 1)\)th order linear differential operator \(\hat{L} = (\tau d/dt + 1)^{q+1}\) and contains an external signal that represents an average profile of spike pulses:

\[
\left(\tau \frac{d}{dt} + 1\right)^{q+1} S(t) = \begin{cases} 
\frac{1}{N} \sum_{n'=1}^{N} \sum_{m\backslash t_{m'n'}^n < t} \delta(t - t_{m'n'}^n), \\
\frac{1}{N} \sum_{n'=1}^{N} P_{\nu} (\theta_{n'}(t)), \\
\frac{1}{N} \sum_{n'=1}^{N} Q_{\nu} (\varphi_{n'}(t)).
\end{cases}
\]

(24)

The source term on the right-hand side of (24) is presented in three interchangeable forms corresponding to the QIF model (1), (2), theta-neuron (3), (4), and their averaged representation through the KS model (20). This term can be interpreted as the population firing rate, which induces a post-synaptic current in response to the arrival of spikes. In the limit \(\tau \to 0\), one might assume that the interaction between neurons becomes instantaneous. However, if the characteristic time scale \(\tau\) of a post-synaptic response is not negligibly small, it becomes imperative to take into account the individual adaptation dynamics of the synaptic variable \(S(t)\), encompassing its activation, deactivation, and time delay.

To describe the adaption dynamics of \(S(t)\), it is common to assume that the synaptic variable \(S(t)\) follows the first-order Devalle et al. (2017); Bick et al. (2020); Afifurrahman et al. (2020, 2021); Pietras et al. (2023) or second-order ordinary differential equation Mohanty and Politi (2006); Zillmer et al. (2007); Bolotov et al. (2016); Chen et al. (2017), corresponding to \(q = 0\) and \(q = 1\) in (24), respectively.

In the case \(q = 0\), the mean population synaptic activity \(S(t)\) is governed by the standard relaxation rule. Here, when the \(n'\)th neuron fires at time \(t_{m'n'}^n\), generating the \(m\)th spike in the form of the Dirac delta function, the mean activity \(S(t)\) instantaneously changes and subsequently decays exponentially in the absence of further firings. The parameter \(\tau\) acts as a synaptic time constant.

For \(q = 1\), when the \(n'\)th neuron fires at time \(t_{m'n'}^n\) and the \(m\)th Dirac delta pulse is generated, the variable \(S(t)\) is augmented by the function \(G((t - t_{m'n'}^n)/\tau) / N\) defined by (23) with \(q = 1\), coinciding with the so-called alpha-function pulse Mohanty and Politi (2006); Zillmer et al. (2007); Bolotov...
et al. (2016); Chen et al. (2017). For this alpha-pulse created by a spiking neuron, $\tau$ determines both the signal’s width and the time at which it attains its maximum value (Fig. 1).

Figure 1. Synaptic dynamics $S(t)$, defined by (4), induced by presynaptic spikes $p(t)$, taking the form of: (a,b) $P_\infty(t)$ as defined by (6) and (c,d) $P_\nu(t)$ as defined by (5) with $\nu = 40$ and linearly increasing phase $\theta_1(t) = t$ ($N = 1$). The parameter $\tau = 0.18$ is used for all cases.

We extend the argument to arbitrary $q$ and $\tau$ that make the model of synaptic adaptation encompass a broad spectrum of realistic biophysical scenarios, ranging from fast non-delayed to slow delayed activation. These scenarios include neurotransmitter release in the synaptic cleft and the opening/closing of postsynaptic ion channels, characterized by distinct time scales such as latency, rise, and decay times, as reflected in the kernel function (23).

Our choice of the kernel function $G(t/\tau)$ for synaptic activation aligns with the gamma distribution, a continuous probability distribution characterized by two parameters: $\tau > 0$, the scale parameter, and $q > 0$, the shape parameter. This distribution reaches its maximum value at $t_{max} = q\tau$, with mean value $t_{\text{mean}} = (q + 1)\tau$, variance $\sigma_t^2 = (q + 1)\tau^2$, and skewness $\gamma_t = 2/\sqrt{q + 1}$. The skewness, reflecting the symmetry of the distribution about its mean, is maximal for the exponential case $q = 0$ and diminishes for larger values of $q$, indicating increased symmetry.

While both $q$ and $\tau$ influence synaptic dynamics, $q$ has a more significant impact on synaptic time delay than $\tau$, whereas $\tau$ predominantly shapes the synaptic activation profile. Figures 1 and 2 demonstrate how the parameters $\tau$ and $q$ determine the time profile of the post-synaptic response and its characteristics.

Towards our objective of deriving explicit conditions for the attractiveness or repulsiveness of synaptic coupling governed by (18) with the kernel function (23), we calculate the complex coefficient...
Figure 2. Characteristics of the synaptic dynamic profile for $S(t)$ as a function of $q$. (a): Peak latency, $t_m$, (b): maximum value, $S_m$, and (c): full width at half maximum (FWHM), induced by spikes $P_\nu(t)$ with $\tau = 0.1$ and different $\nu$ (cyan markers – $\nu = 10$, red markers – $\nu = 100$, orange markers – $\nu = 1000$).

$G_1$ and its modulus and argument from (18) as follows:

$$G_1 = \frac{1}{(1 + i\Omega \tau)^{q+1}}, \quad |G_1| = \frac{1}{\left(1 + (\Omega \tau)^2\right)^{(q+1)/2}}, \quad \arg (G_1) = -(q + 1) \arctan (\Omega \tau),$$

(25)

where $\Omega$ is determined from (22). While the hypergeometric function $Q_{\nu1}$, a factor defining the coupling strength $K$ in the KS model (20), cannot be expressed via elementary functions, it is evident that $Q_{\nu1} \leq 0$ for $\nu \geq 0$. Consequently, the coupling strength $K = 2\kappa|G_1| Q_{\nu1}/\Omega$ in the KS model (20) for $\Omega > 0$ and the coupling strength $\kappa$ in the QIF model (1) have opposite signs.

Therefore, for $\kappa < 0$, a positive coupling strength $K$ corresponds to attractive coupling, provided that the Sakaguchi phase lag parameter $\alpha < \pi/2$. According to (21), $\cos \alpha > 0$ if

$$\sin[(q + 1) \arctan (\tau \Omega)] > 0.$$

(26)

Solving this inequality, we obtain the following $\lfloor q/2 \rfloor + 1$ intervals of parameters that correspond to the attractive coupling in the KS model (20) and therefore in the QIF (1,2) and theta-neuron.
models \((3), (4)\) with \(\kappa < 0\):

\[
\tau \Omega \in D_0 \cup \left( \tan \left( \frac{|q/2|}{q+1} \pi \right), +\infty \right), \quad \text{for even } [q/2],
\]

\[
\tau \Omega \in \left( -\infty, -\tan \left( \frac{|q/2|}{q+1} \pi \right) \right) \cup D_0, \quad \text{for odd } [q/2],
\]

where \(D_0 = \left[ \frac{(q-2)/4}{\tan \left( \frac{2n}{q+1} \pi \right), \tan \left( \frac{2n+1}{q+1} \pi \right)} \right] \).

Figure 3 displays the regions, as defined by (27), where the coupling in the KS model is attractive (blue) or repulsive (red). These regions exhibit an alternating pattern as functions of the synaptic time constant \(\tau\) and the common external input \(\theta\) for a fixed \(q\). Interestingly, increasing the parameter \(q\), which primarily controls the time delay, renders the synaptic coupling more sensitive and results in a greater number of alternating zones. This alternating pattern of attractive and repulsive coupling resembles the stability criterion for synchronization in time-delayed phase oscillator networks, where the sign of the derivative of the periodic coupling function is controlled by the time delay and alternates [Earl and Strogatz (2003)]

\[\text{Figure 3. Regions of attractive (blue) and repulsive (red) coupling in the theta-neuron model (3), (4), corresponding to the KS model regions defined by (27). The colors represent the coupling strength } K \text{ sign as a function of the synaptic time constant } \tau \text{ and the common external input } \theta \text{ for a fixed } q. \text{ (a): } q = 2, \text{ (b): } q = 4, \text{ and (c): } q = 12. \text{ Other parameters: } \kappa = -0.2\pi, \nu = 20, \eta_1 = \eta_2 = \cdots = \eta_N = \eta. \text{ The yellow points } A, B, C, \text{ and } D \text{ indicate the parameter values used for numerical simulations of Figs. 5-7.}\]

In the following, we offer additional evidence supporting the predictive power of the derived KS model. We show numerically that it effectively captures the emergence of robust dynamical regimes like synchronization and more intricate partially synchronized dynamics such as weakly stable cyclops states and non-stationary generalized splay states in both the QIF and theta-neuron models.

5 DYNAMICAL EQUIVALENCE OF THE MODELS: NUMERICAL VALIDATION

We conduct numerical computations using a widely accepted fifth-order Runge–Kutta method with a fixed time step of 0.01, providing additional validation for our analytical findings and predictions.
To characterize the dynamical regimes, we utilize both microscopic measures (pairwise phase differences and firing times) and macroscopic indicators such as the first- and second-order complex Kuramoto parameters Daido (1992); Skardal et al. (2011):

$$R_{\ell}(t) = \frac{1}{N} \sum_{n'=1}^{N} e^{i\varphi_{n'}} = r_{\ell} e^{i\psi_{\ell}},$$  (28)

where $r_{\ell}$ and $\psi_{\ell}$, $\ell = 1, 2$ define the magnitude and the phase of the $\ell$th moment Kuramoto order parameter $R_{\ell}(t)$, respectively. The first-order scalar parameter $r_{1} = |R_{1}|$ characterizes the degree of phase synchrony with $r_{1} = 1$ corresponding to full phase synchrony. The second-order scalar parameter $r_{2} = |R_{2}|$ determines the degree of cluster synchrony, where $r_{2} = 0$ corresponds to generalized splay states Berner et al. (2021b) and their particular case of cyclops states Munyayev et al. (2023).

To identify the time steps corresponding to neuron spike events in both the QIF-neuron model and the theta-neuron model, we monitored the sign changes of $v_{n}(t) - v_{th}$ and $(\theta_{n}(t) \mod 2\pi) - \pi$, along with their time derivative signs. Subsequently, we determined the spike moment $t_{m}^{n}$ using linear interpolation within each time step.

Figures 4 and 5 illustrate the perfect correspondence between the emergence of full synchronization and non-stationary generalized splay states in the theta-neuron model (3),(4) and the KS model (20) within the range of attractive coupling (point A in Fig. 3) and repulsive coupling (point B in Fig. 3), respectively. In the case of full synchronization (Fig. 4), the first-order and second-order scalar parameters, $|R_{1}|$ and $|R_{2}|$, converge to unity but cannot reach 1 due to intrinsic parameter mismatch in $\eta_{n}$. Likewise, the first-order scalar parameter, $|R_{1}|$, associated with the non-stationary generalized splay state oscillates closely around 0 (Fig. 5).

Figures 6-8 illustrate the remarkable agreement between cooperative dynamics in the QIF and KS models. Specifically, Fig. 6 depicts the onset of full synchronization, as evidenced by synchronized firing rates and times. The slight discrepancy in the firing times between the QIF and KS models may stem from various sources, such as accumulated numerical errors and the approximate calculation of the frequency parameter $\Omega$ derived from (22) for selecting the KS model parameters. Figure 7 provides evidence for the capability of the KS model to perfectly predict even non-stationary, asynchronous firing in the QIF model. Figure 8 illustrates the predictive power of the derived KS model in discerning stable complex cluster patterns like cyclops states in the QIF model. Introduced in Munyayev et al. (2023), cyclops states are formed by two distinct, coherent clusters, and a solitary oscillator reminiscent of the Cyclops’ eye. While detecting stable cyclops states can be challenging in the QIF model, the KS model provides a more convenient and constructive approach.

The numerical results depicted in Figs. 6-8 were obtained using sufficiently large values of $v_{th}$ and $v_{r}$, specifically $v_{th} = -v_{r} = 10^{5}$, ensuring better consistency between the QIF-neuron model and the theta-neuron model. However, additional numerical simulations, not presented here, indicate that reducing these parameters to $v_{th} = -v_{r} = 10^{2}$ does not alter the qualitative depiction of firing times. The synchronous or asynchronous nature of the dynamics and the critical transitions remain unchanged.
Figure 4. Dynamical equivalence between the theta-neuron \( [3], [4] \) and KS models \([20]\), demonstrated via the onset of full synchronization. (a): The evolution of the first \(|R_1|\) (solid curves) and second \(|R_2|\) (dashed curves) order parameters for the theta-neuron (green curves) and KS model (red curves), including the transient period. Initial phases \( \theta_n, n = 1, \ldots, N = 21 \) are uniformly distributed over the interval \([-\pi; \pi]\). (b): The colors depict the phase differences \( \theta_n - \theta_{21} \) in the theta-neuron model converging to imperfect full synchronization, subject to mismatched parameters \( \eta_n \) that are uniformly distributed on the segment \([\bar{\eta} - \delta \eta/2; \bar{\eta} + \delta \eta/2]\), \( \bar{\eta} = 2.0, \delta \eta = 10^{-6} \). (c): Comparison between the dynamics of the phase differences \( \theta_n - \theta_{21} \) for \( n = n_1 = 17 \) (thick red curve) and \( n = n_2 = 20 \) (thick blue curve) in the theta-neuron model and \( \theta_n - \theta_{21} \) recalculated from phases \( \phi_n \) using the relation (7) for \( n = n_1 = 17 \) (thin cyan curve) and \( n = n_2 = 20 \) (thin red curve) in the KS model. Note the perfect alignment of the phase-difference dynamics in the two models. Parameters: \( q = 2, \kappa = -0.2 \pi, \tau = 0.5, \nu = 20, \bar{\eta} = 2.0 \) correspond to point A on Fig. 3.

6 CONCLUSIONS

Understanding the influence of synaptic dynamics, including activation rates, deactivation processes, and latency, on collective dynamics in neuronal networks is of significant importance. Considerable advancements have been made in analyzing the role of fast or time-delayed synapses in integrate-and-fire neuron networks. However, there remains a scarcity of analytical studies exploring the influence of slower synaptic dynamics, potentially in the presence of time delays, on controlling critical phase transitions in neuronal networks.

In this paper, we have made substantial contributions to advancing analytical methods in this area. We studied a finite-size network of QIF neurons globally interconnected via a generalized kernel function governing both synaptic activation and time delay. Our analytical exploration demonstrated how the shape of the kernel function profoundly affects neuron interaction, thereby
Figure 5. Dynamical equivalence between the theta-neuron (3), (4) and KS models (20), demonstrated via the onset of non-stationary generalized splay state with an oscillating $|R_1| \approx 0$. Notations are as in Fig. 4. Mismatch parameters $\eta_n$ are chosen from a uniform distribution $[\bar{\eta} - \delta \eta / 2; \bar{\eta} + \delta \eta / 2]$, $\bar{\eta} = 2.0$, $\delta \eta = 10^{-3}$. Other parameters: $N = 21$, $q = 2$, $\kappa = -0.2\pi$, $\tau = 0.8$, and $\nu = 20$ correspond to point B on Fig. 3 and yield the frequency parameter $\Omega \approx 2.639$, calculated from (22) [not shown].

significantly modifying the microscopic and macroscopic behavior of QIF networks. To achieve this, we reduced the QIF and theta neuron network models to the Kuramoto-Sakaguchi model. In this model, oscillator frequencies, coupling strength, and the Sakaguchi phase lag parameter are determined by the Fourier terms of the pulse profile series expansion.

We established exact conditions determining whether synaptic coupling is attractive, fostering synchronization, or repulsive, promoting splay and cyclops states. Furthermore, we demonstrated a remarkable correspondence between the dynamics of the derived KS model and the original QIF and theta-neuron models. Specifically, the KS model accurately predicted firing rates and times, capturing the emergence of synchronization, weakly stable cyclops states, and non-stationary regimes in the QIF model.

Our reduction approach to an analytically tractable Kuramoto model holds promise in facilitating constructive analysis of rhythmogenesis in QIF networks. By utilizing the reduced KS model, a variety of methods and analytical machinery can be applied to the analysis of collective dynamics in QIF models, including the constructive selection of complex patterns, such as chimeras, whose existence and emergence might be easier to deduce from the reduced Kuramoto model description. Furthermore, the reduction approach holds the potential for extensions to incorporate synaptic
Figure 6. Onset of full synchronization in the QIF (1) and KS models (20). (a): Firing rate and (b): firing times of QIF neurons (cyan curves and round markers) and oscillators of the KS model (black curves and cross markers) with the firing times recorded at $\theta_n(t_f) = \pi$. Each row in (b) represents the firing times of a neuron/oscillator. Inset (c) zooms-in on the firing time pattern from (b). Initial conditions are as in Fig. 4. Parameters: $N = 21$, $q = 4$, $\tau = 0.15$, $\kappa = -0.2\pi$, $v_{th} = -v_r = 10^5$, $\bar{\eta} = 2$, $\delta \eta = 6 \times 10^{-3}$, $\nu = 10^5$ correspond to point C in Fig. 3. The firing rates are calculated within a sliding window with a width of $10^{-2}$.

Figure 7. Diagram similar to Fig. 6 showing a nearly perfect match for asynchronous firing rates (a) and firing times (b) in the QIF network (cyan curves and round markers) and the KS model (black curves and cross markers). Parameters: $N = 21$, $q = 4$, $\tau = 0.41$, $\kappa = -0.2\pi$, $v_{th} = -v_r = 10^5$, $\bar{\eta} = 2$, $\delta \eta = 6 \times 10^{-3}$, $\nu = 10^5$ correspond to correspond to point C in Fig. 3. Other notations and settings are as in Fig. 6.
Figure 8. Firing rates (a) and firing times (b) of a three-cluster cyclops state in the QIF network (cyan curves and round markers) and the KS model (black curves and cross markers). (c). Snapshots of the cyclops state phase distributions $\varphi_n$ in the KS model at two time instants. The oscillators’ coloring represents their phase. The cyclops states is formed by a solitary oscillator (red) and two coherent clusters, each composed of 10 oscillators (orange and blue). The initial phases are chosen near a cyclops state. Parameters: $N = 21$, $q = 2$, $\tau = 0.8$, $\zeta = -0.2\pi$, $v_{th} = -v_r = 10^5$, $\bar{\eta} = 2$, $\delta\eta = 0$, $\nu = 10^5$. Other notations and settings are as in Fig. 6.

adaptation, Hebbian learning, and a complex network structure by employing node-degree block approximation.

FUNDING

This work was supported by the Ministry of Science and Higher Education of the Russian Federation under project No. 0729-2020-0036 (to G. V. O. and M. I. B.), the Russian Science Foundation under project No. 22-12-00348 (to V. O. M. and L. A. S.), the Georgia State University Brains & Behavior Program, the National Science Foundation (USA) under Grant No. CMMI-2009329, and the Office of Naval Research under Grant No. N00014-22-1-2200 (to I. B.).
DATA AVAILABILITY STATEMENT

The numerical simulation code and data supporting this study’s findings are available from the corresponding author upon reasonable request.

CONFLICT OF INTEREST STATEMENT

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

REFERENCES


