Kuramoto model for populations of quadratic integrate-and-fire neurons with chemical and electrical coupling

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We derive the Kuramoto model (KM) corresponding to a population of weakly coupled, nearly identical quadratic integrate-and-fire (QIF) neurons with both electrical and chemical coupling. The ratio of chemical to electrical coupling determines the phase lag of the characteristic sine coupling function of the KM, and critically determines the synchronization properties of the network. We apply our results to uncover the presence of chimera states in two coupled populations of identical QIF neurons. We find that the presence of both electrical and chemical coupling is a necessary condition for chimera states to exist. Finally, we numerically demonstrate that chimera states gradually disappear as coupling strengths cease to be weak.

The Kuramoto model (KM) is a minimal mathematical model for investigating the emergence of collective oscillations in populations of heterogeneous, self-sustained oscillators. Though the KM model was not originally intended to describe any specific natural system, an abundant body of work applies it to explore large-scale neuronal oscillations; see, e.g., Refs. [3–22]. Yet, it remains unclear how the parameters of the KM relate to parameters—such as chemical or electrical synaptic strengths—critical for setting up synchronization in biophysically realistic neuronal models. Here, we unveil a mathematical relation between a popular spiking neuron model, the quadratic integrate-and-fire (QIF), with a well-known variant of the KM, and introduce the method to reduce the QIF model to the KM. This provides support in favor of the use of the KM for modeling studies in computational neuroscience and introduces the powerful mathematical framework of the KM for the analysis of the dynamics of QIF networks.

I. INTRODUCTION

Large-scale neuronal oscillations emerge due to the synchronous interplay of ensembles of neurons. These oscillations are successfully replicated by mathematical models of spiking neurons, which also allow for a mechanistic understanding of neuronal rhythmnogenesis. According to these theories, inhibitory synapses play a central role in setting up neuronal synchronization either in isolation or due to their interplay with excitatory neurons. Additionally, inhibitory cells are very often coupled electrically, and this coupling is usually mediated by so-called gap junctions. Such electrical synapses are well-known to largely favor synchrony.

Recently, important efforts have been put forward to model the oscillatory dynamics of so-called whole-brain networks. To facilitate both the analysis and the computational work many studies do not use spiking neuron models, but apply the mathematical framework of the Kuramoto model (KM); see, e.g., Refs. [3–22]. Yet, it remains unclear how to relate the parameters of the KM to bio-physically meaningful parameters, such as synaptic strengths.

In this paper, we aim to theoretically substantiate the use of the KM for neuronal modeling, by providing a mathematical link between the quadratic integrate-and-fire (QIF) model and the KM. We derive a KM for QIF neurons and subsequently justify its validity in two different ways: First, we compare the predictions of the KM with those of an exact mean-field model for QIF neurons—often referred to as firing rate or neural mass model (NMM). Second, we use two populations of identical Kuramoto oscillators to find so-called chimera states. In a chimera state, one of the two homogeneous populations displays in-phase synchrony, and NMMs are useless in this case. However, the KM for QIF neurons is perfectly suited to describe full synchrony, and we exploit this to uncover the existence of chimera states in two-population networks of QIF neurons.

Our derivation of the KM for QIF neurons mainly builds on a previous work by Izhikevich [27] and also on Refs. [44] and [45]. In Chap. 10 of Ref. [27] Izhikevich applied perturbation methods to derive a simplified model that approximated the dynamics of two identical QIF neurons, with either chemical or electrical coupling. Here, we extend the work of Izhikevich and derive a model that approximates the dynamics of an ensemble of heterogeneous QIF neurons with both chemical and electrical coupling. The approximated model turns out to be a well-known version of the KM [28–29] and is valid when both heterogeneities and coupling strengths are weak.

This paper is organized as follows: In Section II, we introduce the QIF population model, and in Section III, we describe the method to reduce the QIF model to the KM. In Section IV, we analyze the dynamics of the KM and demonstrate that it correctly describes the collective dynamics of populations of nearly identical QIF neurons, with weak electrical and chemical synapses. In Section V, we exploit the KM to uncover the presence of chimera states in coupled populations.
of identical QIF neurons. Finally, in Section IV, we briefly discuss and summarize our results.

II. POPULATION OF QIF NEURONS WITH ELECTRICAL AND CHEMICAL SYNAPSES

We investigate a population of $N$ quadratic integrate-and-fire (QIF) neurons $i = 1, \ldots, N$ interacting all-to-all via both electrical and chemical synapses:

$$\tau V_i = V_i^2 + \eta_i + \varepsilon I_{syn}(t), \quad \text{if } V_i > V_p, \text{ then } V_i \leftarrow V_r,$$  \hspace{1cm} (1)

where $V_i$ is the membrane potential of neuron $i$, $\tau$ is the membrane time constant of the neurons, and $\eta_i$ represents an external current, which varies from cell to cell. Due to the quadratic nonlinearity of the QIF model, the membrane potential blows up in finite time, and a resetting rule is needed: When the neurons reach the peak value $V_p$, they emit a spike and the voltage is reset to $V_r$. We assume symmetric spike resetting, $V_p = -V_r$ and $V_p \to \infty$, so that the QIF model is equivalent to the so-called theta-neuron model. In addition, we consider $\eta_i > 0$ and hence, in the absence of synaptic inputs ($I_{syn} = 0$), QIF neurons are self-sustained oscillators. Finally, synaptic inputs (whose total strength is controlled by the small parameter $\varepsilon \geq 0$) are composed of electrical and chemical synapses,

$$I_{syn}(t) = g(v(t) - V_i) + J \tau r(t).$$  \hspace{1cm} (2)

Specifically, electrical synapses (of strength $\varepsilon g \geq 0$) diffusively couple each neuron with the mean membrane potential

$$v(t) = \frac{1}{N} \sum_{j=1}^{N} V_j(t).$$  \hspace{1cm} (3)

Electrical synapses mostly connect inhibitory neurons, and hence the chemical synaptic strength, $J$, is thought of as a negative parameter thereafter. Finally, chemical synapses (of strength $\varepsilon J$) are mediated by the mean firing rate

$$r(t) = \frac{1}{N} \sum_{j=1}^{N} \sum_{k} \delta \left( t - t_j^{(k)} \right),$$  \hspace{1cm} (4)

where $t_j^{(k)}$ is the time of the $k$th spike of the $j$th neuron and $\delta(t)$ is the Dirac delta function.

III. DERIVATION OF THE KURAMOTO MODEL FOR POPULATIONS OF QIF NEURONS

In the following we derive the Kuramoto model corresponding to Eq. (1). The derivation exploits well-known mathematical methods that are reviewed for example in Refs. [27, 39] and [50].

We perform the derivation of the KM as follows: First, we obtain the phase resetting curve (PRC) of the QIF model. Second, invoking weak coupling, we derive the so-called Winfree model corresponding to the QIF model. Finally, we assume weak heterogeneity and apply the method of averaging to obtain the Kuramoto model corresponding to Eq. (1).

A. Phase resetting curve (PRC) of a QIF neuron

We first consider an isolated, regularly spiking QIF neuron; i.e., $I_{syn} = 0$ and $\eta_i > 0$. The solution of the QIF model immediately after a spike, $V_i(0) = -\infty$, is

$$V_i(t) = \sqrt{\eta_i} \tan \left(t \sqrt{\eta_i} / \pi - \pi / 2\right).$$  \hspace{1cm} (5)

The frequency of the oscillations is

$$\Omega = 2\sqrt{\eta_i} / \pi,$$  \hspace{1cm} (6)

and a phase variable $\theta_i$ can be defined in the interval $[0, 2\pi]$ as

$$\theta_i = \Omega t = 2 \arctan(V_i / \sqrt{\eta_i}) + \pi.$$  \hspace{1cm} (7)

Next, we assume that the neuron is perturbed so that its membrane potential instantaneously changes from $V_i$ to $V_i + \delta V$. Then, the new phase after the perturbation is $\theta_{i, new} = 2 \arctan((V_i + \delta V) / \sqrt{\eta_i}) + \pi$.

The PRC measures the phase shift produced by the perturbation, i.e., PRC$ = \theta_{i, new} - \theta_i$. Hence, for the QIF model, the PRC is

$$\text{PRC}(\theta, \delta V) = 2 \arctan(\delta V / \sqrt{\eta_i} - \cot(\theta / 2)) + \pi - \theta.$$  \hspace{1cm} (8)

This function depends on both the strength of the perturbation and the phase of the neuron at the instant of the perturbation. The PRC Eq. (5) is always positive, indicating that positive/negative perturbations only produce positive/negative phase shifts. This characterizes the so-called Class 1 neuronal oscillator.

B. Weak coupling approximation and the Winfree model

The PRC Eq. (5) exactly characterizes the phase response of the QIF neuron to a perturbation. Next, we invoke weak coupling, which allows for deriving a new phase model—called the Winfree model—that approximates the network Eq. (1) for $\varepsilon \ll 1$.

Weak perturbations produce small changes in the membrane potential, $|\delta V| \ll 1$. Then the PRC scales linearly with the strength of the perturbation,

$$\text{PRC}(\theta, \delta V) \approx Z(\theta) \delta V,$$

where $Z(\theta)_{\varepsilon}$ is called phase sensitivity function or infinitesimal phase resetting curve (iPRC). For the QIF model, the iPRC is

$$Z(\theta) = \frac{\partial \text{PRC}(\theta, \delta V)}{\partial \delta V} \bigg|_{\delta V = 0} = 1 - \cos \theta.$$  \hspace{1cm} (9)

When weak perturbations are described by a continuous function $P(t)$ with $|P(t)| \ll 1$, the infinitesimal change in the phase due to the perturbations is $d\theta = Z(\theta) P(t) dt$. Accordingly, assuming weak coupling $\varepsilon \ll 1$, the population of QIF neurons Eq. (1) is well approximated by the Winfree model,

$$\dot{\theta}_i = \Omega_i + \varepsilon \left( 1 - \cos \theta_i \right) \sum_{j=1}^{N} P(\theta_i, \theta_j),$$  \hspace{1cm} (10)
where perturbations to neuron $i$ are due to synaptic inputs from neuron $j$ and can be written in terms of the phase variables as

$$P(\theta_i, \theta_j) = \frac{g}{N} \left( \cot(\theta_j/2) - \sqrt{\frac{\eta_j}{\eta}} \cot(\theta_j/2) \right) + \frac{2J}{N} \sqrt{\frac{\eta_j}{\eta}} \delta(\theta_j).$$  \hspace{1cm} (11)

Recall that $\theta_j \in [0, 2\pi)$ so that the Dirac delta function in Eq. (11) has argument zero whenever neuron $j$ fires a spike.

C. Weak heterogeneity and the averaging approximation

The Winfree model can be further simplified using the method of averaging. We consider the external currents in Eq. (1) as a common current $\eta$ plus a weakly distributed parameter as

$$\eta_i = \bar{\eta} + \varepsilon \chi_i.$$  \hspace{1cm} (12)

In the derivation of the Winfree model, we already assumed weak coupling, $\varepsilon \ll 1$. Therefore, the smallness of parameter $\varepsilon$ implies now the smallness of both coupling terms and the level of heterogeneity. This assumption allows for a separation of time scales so that the phases $\theta_i$ can be written as

$$\theta_i = \Phi + \phi_i,$$  \hspace{1cm} (13)

where $\Phi$ describes the fast, free-running oscillation of period

$$T = \tau \pi / \sqrt{\eta},$$

whereas the phases $\phi_i$ describe slow phase drifts produced by weak heterogeneities and synaptic inputs. Substituting Eq. (13) into the Winfree model Eqs. (10,11) and collecting terms of order $\varepsilon$, we find the evolution equation for the slow phases

$$\dot{\phi}_i = \frac{\varepsilon \chi_i}{\tau \sqrt{\eta}} + \left[ 1 - \cos(\Phi + \phi_i) \right] \frac{\varepsilon}{\tau N} \sum_{j=1}^{N} p(\Phi + \phi_i, \Phi + \phi_j + \Delta_{ji}).$$  \hspace{1cm} (14)

Here, we defined pairwise phase differences as $\Delta_{ji} = \phi_j - \phi_i$ and a function describing synaptic perturbations as

$$p(x,y) = g \left[ \cot(x/2) - \cot(y/2) \right] + 2J \delta(y).$$

To apply the method of averaging to Eq. (14), we consider that in one period of the fast oscillation, $T$, the slow phases $\phi_i$ can be assumed constant. Then, Eq. (14) reduces to

$$\dot{\phi}_i = \frac{\varepsilon}{\tau N} \sum_{j=1}^{N} \Gamma(\Delta_{ji}),$$  \hspace{1cm} (15)

where the coupling function $\Gamma$ is obtained by averaging the r.h.s. of Eq. (14) over one period $T$. This involves the evaluation of four integrals that can be explicitly computed and yields the phase interaction function

$$\Gamma(\Delta_{ji}) = \frac{\chi_i}{\sqrt{\eta}} + g \sin \Delta_{ji} + \frac{J}{\pi} (1 - \cos \Delta_{ji})$$  \hspace{1cm} (16)

D. Kuramoto model for populations of QIF neurons

Substituting Eq. (10) into Eq. (15) and expressing the result in terms of the original phases Eq. (12), we find the Kuramoto model

$$\dot{\theta}_i = \omega_i + \frac{\varepsilon}{\tau N} \sum_{j=1}^{N} \left[ g \sin(\theta_j - \theta_i) - \frac{J}{\pi} \cos(\theta_j - \theta_i) \right] + \frac{\varepsilon J}{\tau \pi},$$  \hspace{1cm} (17)

with natural frequencies

$$\omega_i = \frac{2\sqrt{\eta}}{\tau} + \frac{\varepsilon \chi_i}{\tau \sqrt{\eta}}.$$  \hspace{1cm} (18)

In the absence of electrical synapses $g = 0$, Eqs. (17) essentially reduces to the Kuramoto model with chemical synapses derived in Refs. [44] and [45]. The KM for QIF neurons Eq. (17) generalizes the results in Refs. [44] and [45] to networks with both electrical and chemical coupling, and it is our main result.

Eq. (18) is the linear approximation of Eq. (6) for weak heterogeneity—see also Eq. (12). The last term of Eq. (17) describes the deviation of the natural frequencies due to synaptic coupling, which exclusively depends on chemical coupling. Excitatory coupling ($J > 0$) speeds up the frequencies of the oscillators, and inhibition ($J < 0$) slows them down. These frequency shifts do not qualitatively affect the collective dynamics of Eq. (17), but they may become relevant if the oscillators are not all-to-all coupled [28,31] or in the case of interacting excitatory and inhibitory populations [29].

Alternatively, Eq. (17) can be cast in the more transparent form [28,29]

$$\dot{\theta}_i = \omega_i + \frac{K}{N} \sum_{j=1}^{N} \left[ \sin(\theta_j - \theta_i - \alpha) + \sin \alpha \right],$$  \hspace{1cm} (19)

with the coupling constant

$$K = \frac{\varepsilon}{\tau} \sqrt{\left( \frac{J}{\pi} \right)^2 + g^2},$$  \hspace{1cm} (20)

and the phase lag parameter

$$\alpha = \arctan \left( \frac{J}{\pi g} \right).$$  \hspace{1cm} (21)

The coupling parameters $K$ and $\alpha$ satisfy a simple geometric relation with the coupling parameters of the QIF model Eq. (1), illustrated in Fig. [1]. Given a particular choice of the QIF coupling parameters, Fig. [1] shows that electrical coupling and chemical coupling (divided by a factor $\pi$) contribute equally to the overall coupling strength $K$ of the KM, and that $K$ is insensitive to the sign of the chemical coupling—in Fig. [1] we consider an inhibitory network; i.e., $J < 0$.

To lighten the notation, we consider $\varepsilon = 1$ thereafter. Hence, for the KM Eq. (19) to be a good approximation of Eq. (1), in the following, the synaptic weights $J$ and $g$ need to be regarded as small quantities.
IV. ANALYSIS OF THE KURAMOTO MODEL FOR QUADRATIC INTEGRATE-AND-FIRE NEURONS

Using Fig. 1—or, equivalently, Eq. (21)—we may infer how chemical and electrical synapses contribute to synchronization, using well-known results for the KM. For example, the phase constant $\alpha$ critically determines the synchronization behavior of Eq. (19). In the absence of electrical coupling, $g = 0$, we find $\alpha = +\pi/2$ for excitatory coupling and $\alpha = -\pi/2$ for inhibitory coupling. This indicates that collective synchronization is unreachable—consistent with the well-known fact that instantaneous chemical coupling is unable to synchronize type 1 neuronal oscillators. In contrast, in the absence of chemical coupling, one finds $\alpha = 0$, and Eq. (19) reduces to the standard KM, in which collective synchronization is achieved at a critical degree of heterogeneity $\Delta = \Delta_c(K)$ that depends on the coupling strength. Between these two extreme cases, that is in networks with both electrical and chemical synapses, we find the phase lag parameter $|\alpha| \in (0, \pi/2)$, and synchronization generally depends on both $\alpha$ and the overall shape of the distribution of natural frequencies.

To validate the KM for QIF neurons, in Section IV A, we obtain the mean-field model corresponding to Eqs. (19) and compare its predictions with those of the mean-field model derived in Ref. In the limit $N \to \infty$. The mathematical approach to obtain the mean-field equations corresponding to Eqs. (19) is a standard procedure. Here, we skip the mathematical details and refer the reader to, for example Ref. where the mean-field model corresponding to Eq. (19) was derived in detail. Accordingly, using Eqs. (18,20,21), we obtain the mean-field equations

\[
\begin{align*}
\dot{R} &= \frac{R}{2\pi} \left( -\frac{2\Delta}{\sqrt{\eta}} + g(1 - R^2) \right), \quad (23a) \\
\dot{\psi} &= \frac{2\sqrt{\eta}}{\tau} + \frac{J}{2\pi} (1 - R^2), \quad (23b)
\end{align*}
\]

which approximate the dynamics of the QIF model Eqs. (1) for small $g$ and $J$. The radial equation Eq. (23a) shows that the incoherent state ($R = 0$) is a stable fixed point above the critical width,

\[
\Delta_c = g\sqrt{\eta}/2, \quad (24)
\]

which is independent of chemical coupling, $J$. At $\Delta = \Delta_c$ a stable nontrivial solution—corresponding to a partially synchronized state—bifurcates from incoherence with

\[
R = \sqrt{(\Delta_c - \Delta)/\Delta_c},
\]

and frequency

\[
\Omega = \frac{2\sqrt{\eta}}{\tau} + \frac{\Delta}{\tau\pi\sqrt{\eta}} J.
\]

The solid line in Fig. 2 corresponds to the critical boundary Eq. (24), while dashed lines correspond to the exact synchronization boundaries of the QIF network for various degrees of inhibitory coupling—see Eq. (7) in Ref. Note that for weak electrical coupling and/or weak heterogeneity, all boundaries approach Eq. (24). Furthermore, for weak heterogeneity, the frequency of the synchronized cluster Eq. (25)
agrees with Eq. (8) in Ref. [37], which describes the frequency of the oscillations near their onset. In sum, these results confirm the validity of the Kuramoto model Eq. (19) as an approximation of a population of heterogeneous QIF neurons with electrical and chemical coupling, Eq. (1).

V. CHIMERA STATES IN COUPLED HOMOGENEOUS POPULATIONS OF QIF NEURONS

To further illustrate the appropriateness of the KM to investigate the dynamics of QIF networks, we investigate the presence of chimera states in populations of QIF neurons. Our motivation is threefold:

1. Chimera states were originally uncovered in a nonlocally coupled network of identical Kuramoto oscillators.[38] Given that the phase dynamics Eqs. (19) are an approximation of Eqs. (1) valid for weak heterogeneity and weak coupling, we expect QIF networks to display similar chimera states, at least for weak coupling.

2. Several papers have been devoted to investigate chimera states in networks of spiking neurons; see, e.g., Refs. [59–69] Some of them provide numerical evidence that the presence of both chemical and electrical synapses favors the emergence of chimera states.[33–35] Yet, the relation between chimera states in spiking neuron networks with the original chimera states uncovered in the KM[38,39] is lacking.[40–42]

3. Recently, exact mean-field models for large populations of QIF neurons (often called neural mass models, NMMs) with electrical and chemical synapses have been put forward.[31–38] However, such NMMs have an important limitation when neurons are—as in a chimera state—identical and fully synchronized since both the mean membrane potential and the mean firing rate diverge at the instant of collective firing.[24] This divergence is avoided using the averaging approximation, and hence the KM for QIF neurons, Eq. (17), becomes singularly suited to study collective behavior where neurons are fully synchronized.

Chimera states were originally uncovered in a ring of identical Kuramoto oscillators with nonlocal coupling when $\alpha \lesssim \pi / 2$. Shortly after their discovery, chimera states were also found in a simpler setup, consisting of two populations of identical Kuramoto oscillators.[30–41] Here, to investigate chimera states in networks of QIF neurons, we adopt the two-population setup of Refs. [40 and 41].

Specifically, we analyze the dynamics of two identical populations (labeled $\sigma \in \{1, 2\}$) of $n = N/2$ identical QIF neurons, interacting all-to-all via both chemical and electrical synapses

$$\tau \dot{V}_i^\sigma = (V_i^\sigma)^2 + \eta + I_{i,\text{syn},s}^\sigma + I_{i,\text{syn},c}^\sigma,$$  \hspace{1cm} (26)

with the resetting rule of Eq. (1). Synaptic inputs have a contribution $I_{i,\text{syn},s}^\sigma$ due to self-interactions within each population $\sigma$ and another contribution $I_{i,\text{syn},c}^\sigma$ due to cross-interactions of population $\sigma = \{1, 2\}$ with population $\sigma' = \{2, 1\}$,

$$I_{i,\text{syn},s}^\sigma = g_s (V_i^\sigma - V_s^\sigma) + J_s \tau r^\sigma,$$

$$I_{i,\text{syn},c}^\sigma = g_c (V_i^\sigma - V_c^\sigma) + J_c \tau r^\sigma.$$  \hspace{1cm} (27)

Here, $V_i^\sigma$ and $r^\sigma$ are the mean membrane voltage and mean firing rate of population $\sigma$, respectively. Using Eqs. (19,20,21), it is straightforward to write the KM corresponding to Eqs. (26) as

$$\dot{\theta}_i^\sigma = \omega + \frac{K_s}{n} \sum_{j=1}^n \left[ \sin (\theta_j^\sigma - \theta_i^\sigma - \alpha) + \sin \alpha \right]$$

$$+ \frac{K_c}{n} \sum_{j=1}^n \left[ \sin (\theta_j^\sigma - \theta_i^\sigma - \alpha) + \sin \alpha \right],$$  \hspace{1cm} (27)

with $\omega = 2 \sqrt{\pi} / \tau$ and

$$K_{s,c} = \frac{1}{\tau} \sqrt{(J_{s,c} / \pi)^2 + (g_{s,c})^2},$$  \hspace{1cm} (28)

$$\alpha_{s,c} = \arctan \left( \frac{J_{s,c} / \pi}{g_{s,c}} \right).$$  \hspace{1cm} (29)

The KM Eqs. (27) is slightly more general than the model originally investigated in Refs.[40–42] which considered $\alpha_s = \alpha_c$. In the QIF network, this equality of the phase lag parameters implies that the ratios of chemical to electrical coupling

$$\rho_s = \frac{J_s / \pi}{g_s}, \quad \rho_c = \frac{J_c / \pi}{g_c},$$  \hspace{1cm} (30)

are identical, $\rho_s = \rho_c$. Recent work has also considered the dynamics of chimera states in populations of Kuramoto oscillators with distributed phase lag.[43,75] Specifically, Martens et al.[33] investigated chimera states in the two-population model Eqs. (27).

A. Mean-field model

As we discussed previously, in the thermodynamic limit ($n = N/2 \rightarrow \infty$) the KM can be exactly reduced to a low-dimensional mean-field model using the OA ansatz. In the case of the homogeneous, two-population Kuramoto model Eqs. (27), the dynamics reduces to six ordinary differential equations using the Watanabe-Strogatz ansatz.[27] Assuming a particular set of initial conditions for the phases, the system further reduces to four differential equations and it is described by the OA ansatz.[14–16] Such mean-field equations describe the evolution of the complex Kuramoto order parameters of the two populations,

$$Z_\sigma = R_\sigma e^{i \theta_\sigma} = \frac{1}{n} \sum_{j=1}^n e^{i \theta_j^\sigma}.$$  \hspace{1cm} (31)

Using the mean-field analysis in Refs. [43] and [78] and Eqs. (28,29,30), the mean-field equations for the complex Kuramoto order parameters can be further reduced (by virtue of
Chimera states in two-population Kuramoto networks correspond to symmetry-broken states where one of the populations is fully synchronized (i.e. $R_2 = 1$), while the other remains only partially synchronized ($R_2 < 1$). In addition, chimera states in two-population networks of identical Kuramoto oscillators coexist with the fully synchronized state, $R_1 = R_2 = 1$.23,24

To obtain the phase diagrams depicted in Fig. 3, we set $R_2 = 1$ in Eqs. (26) and numerically continued chimera states using initial conditions in their basin of attraction—see Fig. 4(a) and Ref. 41. The diagrams show the regions where steady (shaded) and unsteady (hatched) chimera states are stable for two different values of the ratio $\rho_s$; see Eqs. (30). These regions lie between a saddle-node (red) and a Homoclinic (green) bifurcation lines, which—together with a Hopf (blue) bifurcation line separating steady and unsteady chimera states—meet at two Takens-Bogdanov (TB) points. For decreasing $|\rho_s|$, the region of chimera states shrinks and eventually disappears when the two TB points collide.

The phase diagrams in Fig. 3 are qualitatively identical to that of Fig. 4a in Ref. 43 but here the regions of chimeras are represented in the parameter space of the QIF model. This allows us to determine three necessary conditions for the existence of chimera states in two-population networks of QIF neurons:

1. Chimera states only exist in the presence of both chemical and electrical coupling.

2. Self-chemical coupling needs to be much larger than self-electrical coupling, $|J_c| > g_s$, or, equivalently, $|\rho_s| > 1$. Using Eq. (29), this implies that $\alpha_s$ is close to $\pm \pi/2$ in correspondence with previous work.10,11,13

3. The modulus of self-chemical coupling needs to be larger than that of cross-chemical coupling, $|J_c| > |J_s|$.

These three conditions are not sufficient conditions to have chimera states in Eqs. (26) though. Indeed, Eq. (27) and the corresponding mean-field Eqs. (32) are an approximation of the QIF Eqs. (26) for weak coupling, but it remains to be seen whether chimera states persist in QIF networks when coupling strengths become stronger. We numerically explore this issue in the next sections.

C. Chimera states in populations of QIF neurons

In the following, we numerically investigate the presence of chimera states in the spiking neuron network model of QIF neurons Eqs. (26). First, we confirm that, for weak coupling, chimeras are present in QIF networks and they exist in the parameter range predicted by the phase diagrams of the KM, Fig. 3. However, then we show that the basin of attraction of chimera states shrinks as synaptic coupling strengths become stronger.

1. Dynamics of chimera states

Using the OA ansatz, the dynamics of the two-population model Eqs. (27) with $N \to \infty$ can be exactly reduced to the three-dimensional system Eqs. (32). In a chimera state we may set $R_2 = 1$ so that Eqs. (32) further reduce to a planar system with variables $R_1$ and $\psi$. Figures 4a,e show the basins of attraction (dashed lines) of (a) a steady chimera state (solid diamond symbol) and of (e) an unsteady chimera state (red limit cycle), with parameters corresponding to $+$ symbols in...
As mentioned previously, chimera states coexist with the stable fully synchronized solution (solid dot symbols), $Z_1 = R_1 = 1$, $\Psi = 0$. The basin of attraction of the chimera state is defined by the stable manifold of a saddle point (open diamond symbols).

To set initial conditions leading to the chimera state of Fig. 3(a) in the network of QIF neurons, we considered the initial condition $Z_2 = 1$ and $Z_1 = R_1 e^{i\Psi}$—with $R_1$ and $\Psi$ such that the system is in the basin of attraction of the steady chimera state. Then we used the conformal map

$$\pi r_\sigma - i v_\sigma = \frac{1-Z_\sigma}{1+Z_\sigma},$$

(33)

to transform the mean-field coordinates $Z_2$ and $Z_1$ into the mean firing rate $r$ and the mean membrane potential $v$ of the populations of QIF neurons—for population 2 we find $\pi r_2 + i v_2 = 0$. Then, we initialized the membrane voltages of the populations according to the formula $V_i(t) = v + (\pi r_i)\tan[\pi/(2(2i-n-1)/(n+1))], \text{for } i = 1,\ldots,n$.\(^{15}\)

In Fig. 5 we show the results of a numerical simulation of the QIF network ($g_s = 0.1$). The raster plot in Fig. 5(a) clearly shows the signature of a chimera state: Neurons in population 1 (blue) are only partially synchronized, while neurons in population 2 remain fully synchronized. The time evolution of the firing rate $r_1$ and the mean-membrane potential $v_1$ for the incoherent group are displayed in Figs. 5(b) and (c), respectively. These collective variables indicate a periodic evolution of the incoherent population, with fluctuations caused by the finite resetting of the QIF neurons and by finite-size effects. Finally, Fig. 5(d) shows the Kuramoto order parameter $R_1$ (blue) obtained using the time series $r_1(t)$ and $v_1(t)$ and the conformal map Eq. (33). In contrast with the steady chimera state in the mean-field Eqs. (32) (black dotted line), the chimera state in the network of QIF neurons is not stationary but oscillates periodically in time. The same unsteady chimeras arise in two-population networks of Winfree oscillators\(^{12}\) and are the consequence of the lack of rotational symmetry in the Winfree and QIF models.

In Fig. 6 we also explored how the unsteady chimera states in the Kuramoto model Eqs. (27) translate to networks of QIF oscillators. To this aim we set the parameters of the QIF model in the hatched region of the bifurcation diagram in Fig. 3(b) ($\times$ symbol), and used the same initial conditions as in the previous simulation. Here, we find a more complex chimera state that seems to display macroscopic quasiperiodic dynamics,\(^{83}\) both the firing rate and the mean-membrane potential of the incoherent population oscillate with two characteristic frequencies as can be appreciated in Figs. 6(b,c). Again, the quasiperiodic chimera state in the QIF network corresponds to a periodic chimera state in the Kuramoto model. In Fig. 6(d)
we used Eq. (33) to represent the Kuramoto order parameter for the QIF network (blue), which roughly approximates the periodic dynamics of the corresponding Eqs. (32).

2. **Chimeras for strong coupling**

The derivation of Eqs. (27) from the QIF network Eqs. (26) has been made under the assumption of weak coupling. Yet, are chimeras states in QIF networks robust against stronger levels of coupling?

To investigate this issue, we used the conformal map Eq. (33) to express the boundary of the basin of attraction of Fig. 4(a) in terms of \((r_1, v_1)\). This transformed boundary is represented as a dashed line in Figs. 4(b-d). Then, we performed three sets of numerical simulations of the QIF network for increasing values of \(g_s\) while keeping the ratios \(g_r/g_s\), \(J_r/J_s\), and \(\rho_s\) constant—note that this implies increasing all the other coupling parameters.

For very small values of \(g_s\), we expect the averaging approximation to hold, and so the stability boundary of chimeras states in the QIF model. The green dotted region in Fig. 4(b) corresponds to the stability boundary of the steady chimera state in the QIF network for \(g_s = 0.025\). The boundary approximately agrees with that of the KM (dashed line), although the region is slightly smaller in the QIF model. Notably, further increases in coupling strength—see Figs. 4(c,d)—lead to a gradual reduction of the chimera’s basin of attraction.

To further investigate the reduction of the stability boundary of chimeras in the QIF model, in Figs. 4(f-h) we computed the basins of attraction of chimeras states in a different parameter regime—corresponding to the + symbol in Fig. 3(a). Here \(|\rho_s|\) is three times larger than in the previous case, and hence, chemical couplings are three times stronger than in Figs. 4(b-d). Correspondingly, the reduction of the basin of attraction

FIG. 5. Periodic chimera state in a two-population network of \(N = 5000\) identical inhibitory QIF neurons \((n = 2500\) neurons in each population). (a) Raster plot of 500 randomly chosen neurons. Neurons in population 1 (blue) are partially synchronized, and neurons on population 2 are in-phase synchronized (red). (b) Time series of the mean firing rate \(r_1\) of population 1, computed averaging the firing rate at each time step in time windows of \(\delta t = 0.05\). (c) Time series of the mean membrane potential \(v_1\) of population 1. (d) Time series of the Kuramoto order parameter of population 1, \(R_1\), obtained from the mean-field quantities \(r_1, v_1\) using the conformal map Eq. (33) (blue lines) and from direct integration of Eqs. (32) (black dots). Parameters as in Fig. 4 (see also + symbol in Fig. 4a): \(J_r/J_s = 0.75\), \(g_r/g_s = 0.5\), \(\rho_s = -40/\pi\), and \(g_s = 0.1\). Numerical simulations performed using the Euler scheme with time step: \(dt = 10^{-4}\) and symmetric resetting: \(V_p = -V_f = 1000\).

FIG. 6. Quasiperiodic chimera state in a two-population network of \(N = 5000\) identical inhibitory QIF neurons. The description of the panels and parameters is as in Fig. 5 except \(J_r/J_s = 0.5\) (see also the \(\times\) symbol in Fig. 4b).
of chimera states in Figs. 3(f–h) is more pronounced than in Figs. 3(b–d). In fact, in Fig. 3(h), we find the complete disappearance of the region of stable chimera states in the QIF network. This suggests that chimera states in QIF networks are only observable for weak coupling.

VI. CONCLUSIONS

In this paper, we have applied a perturbative approach to simplify a weakly heterogeneous population of QIF neurons, with weak all-to-all chemical and electrical coupling Eqs. (1). This approach leads to a classical variant of the Kuramoto model Eq. (19), whose coupling parameters satisfy a simple geometric relation with those of the QIF model Fig. 1.

The approximation of the QIF network by Eq. (19) allows one to use the framework of the KM to investigate the role of chemical and electrical synapses in setting up synchronization. For example, we find that in the absence of electrical coupling the phase lag parameter of the KM is \( \alpha = \pm \pi/2 \), which prohibits synchronization; see also Ref. 48. Moreover, for Lorentzian distributions of currents the synchronization threshold depends only on electrical coupling, Eq. (24), whereas the oscillation frequency Eq. (25) is determined by the ratio of chemical to electrical coupling. These results are in consonance with the exact description provided by so-called neural mass (or firing rate) models for networks of QIF neurons.\(^{28,29}\)

The framework of the KM allows for uncovering and investigating dynamical states that are not reachable using neural mass models for QIF neurons.\(^{37,39,48}\) Here, we analyzed the case of chimera states in two-population networks of identical QIF neurons.\(^{49,51}\) Despite the large number of studies devoted to investigate chimera states in spiking neuron networks—see e.g.\(^{60,69}\)—the relation between such states and the original chimera states uncovered in the KM is lacking.\(^{70,72}\) We showed that chimera states in QIF networks emerge in the presence of both chemical and electrical couplings but only if chemical coupling is much stronger than electrical coupling. However, our numerical results suggest that chimeras in QIF networks are not robust against stronger levels of coupling.

Finally, we introduced a framework for the analysis of QIF networks that can be readily applied to a variety of extensions of Eqs. (1). In particular, the derivation of Eq. (19) does not impose constraints on the structure of the network or the shape of the distribution of heterogeneities. Given that network structure\(^{28,53}\) and heterogeneities\(^{48,49}\) greatly affect the dynamics of the KM, it may be interesting to investigate how this translates to QIF networks.

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DATA AVAILABILITY

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

AUTHOR DECLARATIONS

The authors have no conflicts to disclose.

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