

Phase transformation and synchrony for a network of coupled Izhikevich neurons

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(Received 30 July 2024; accepted 9 December 2025; published 24 December 2025)

Recent studies have used the Lorentzian ansatz to obtain mean-field reductions of Izhikevich neuron networks, but these approaches stop short of connecting microscopic dynamics to synchrony measures. In this work, we construct an equivalent phase model for the Izhikevich neuron and apply the Ott-Antonsen ansatz to derive mean-field equations in terms of the Kuramoto order parameter. This provides governing equations for the evolution of synchrony in networks of Izhikevich neurons. We further demonstrate that the conformal mapping originally derived for quadratic integrate-and-fire neurons remains valid for the Izhikevich model, thereby extending the link between voltage–firing-rate and phase-based descriptions to a broader and more biologically realistic neuron class. Using this mapping, we challenge the common assumption that high firing rates indicate strong synchrony, and instead show that high-amplitude oscillations in firing rate are the true signature of synchrony. This result holds across excitatory and inhibitory populations and suggests that EEG and MEG spectral power could serve as noninvasive biomarkers of underlying neuronal synchrony. Together, these findings extend mean-field reduction techniques to richer neuronal models, strengthen the bridge between mathematical theory and experimental observables, and provide new insight into the relationship between firing activity and collective neural dynamics.

DOI: [10.1103/2qrm-xvhg](https://doi.org/10.1103/2qrm-xvhg)

I. INTRODUCTION

Given the immense complexity of the brain, it is only natural that theoreticians seek reduced simplified models of neural dynamics. Classical neural mass models, such as the Wilson-Cowan model [1], describe average firing rates but neglect the role of synchrony within populations. However, synchronization is known to play a fundamental role in healthy cognition and in a wide range of neurological disorders. For example, excessive synchrony characterizes epileptic seizures and motor symptoms in Parkinson’s disease [2,3]. To understand such phenomena, it is critical to develop mesoscopic models that link neuronal firing rates, voltages, and synchrony in a unified theoretical framework.

A key breakthrough for mesoscopic modeling came with the Ott-Antonsen ansatz, which provided an exact reduction for networks of Kuramoto oscillators [4]. Luke *et al.* subsequently showed that this framework could be extended to networks of theta-neurons [5], and Montbrió *et al.* later introduced a related Lorentzian ansatz to derive a mean-field description for quadratic integrate-and-fire (QIF) neurons [6], which are formally equivalent to the theta-neuron. Together, these advances established the so-called next generation neural mass models [7], which capture firing rate, mean

membrane potential, and synchrony within a unified low-dimensional system.

More recently, attention has turned to extending these approaches to the Izhikevich model [8], which can reproduce a wide variety of cortical firing patterns while remaining computationally efficient. Chen and Campbell applied the Lorentzian ansatz to Izhikevich networks with weak spike-frequency adaptation, showing that the reduction accurately tracks population firing rate and mean voltage [9]. Gast, *et al.* subsequently proposed a population-level adaptation rule, demonstrating improved agreement in the strong adaptation regime [10]. These studies confirmed the feasibility of mean-field reductions for Izhikevich neurons but, importantly, did not provide a link to the Kuramoto order parameter and therefore these models cannot directly quantify synchrony at the mesoscopic scale.

The absence of an explicit synchrony variable limits the interpretability of such models. In particular, changes in mean firing rate cannot reveal whether population activity is asynchronous or synchronized. This distinction is crucial, as synchrony underpins the generation of network rhythms and their alteration in disease [11]. Bridging this gap requires an alternative reduction that describes the dynamics of the Kuramoto order parameter at the mesoscopic scale and a link between these dynamics and the voltage and firing-rate variables.

In this work, we derive an equivalent phase description of the Izhikevich neuron model and apply the Ott-Antonsen ansatz to obtain a reduced model, which describes the dynamics of the Kuramoto order parameter, coupled to the population-averaged synaptic activity and level of adaptation. This provides a synchrony-tracking mean-field model for a network of Izhikevich neurons, thereby extending next

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generation neural mass models to a broader class of dynamics. We further demonstrate that the conformal mapping originally introduced by Montbrió *et al.* [6] for QIF neurons between the phase-based and voltage–firing-rate descriptions also holds for the Izhikevich model, with a suitably modified order parameter. This establishes a direct correspondence between synchrony and mesoscopic observables in a richer class of neuron models.

Finally, we revisit the widely held assumption that high firing rates imply strong synchrony. Using the mean-field model and the conformal mapping, we show instead that high-amplitude oscillations in the firing rate are the true signature of synchrony. This finding holds across both excitatory and inhibitory populations, as well as in coupled networks, indicating that it generalizes beyond a single parameter regime. Moreover, the result has direct experimental implications: high-amplitude oscillations manifest as increased spectral power in EEG and MEG recordings. Our framework therefore not only extends the theoretical foundation of mean-field reductions, but also provides predictions for the interpretation of noninvasive measurements of neural population dynamics.

II. PHASE DESCRIPTION FOR THE IZHIKEVICH MODEL

The Izhikevich neuron model is a simplified two-dimensional description of neural dynamics, which tracks the neuron’s membrane potential v and a phenomenological recovery variable u [8]. When the membrane potential reaches a predefined threshold v_{th} it is said to have spiked and the voltage is reset to a reset value v_{reset} . When the neuron spikes the recovery variable is also increased by a set amount u_{jump} to account for the recovery time post spiking. The dynamics of the Izhikevich neuron model are as follows:

$$C_m \frac{dv}{dt} = k(v - v_\theta)(v - v_r) - u + \eta, \quad (1)$$

$$\frac{du}{dt} = a(b(v - v_r) - u) \quad (2)$$

with the condition $v(t) \rightarrow v_{\text{reset}}$ and $u(t) \rightarrow u(t) + u_{\text{jump}}$ when $v(t) > v_{\text{th}}$. Here, C_m is the membrane capacitance, k is the leakage parameter, v_θ and v_r are the threshold and resting potentials, respectively, η is the background drive, a is the recovery time constant, and b relates to the sensitivity of the recovery variable to the subthreshold fluctuations in the membrane potential.

In the absence of adaptation, the Izhikevich neuron model is simply a transformation of the quadratic integrate-and-fire (QIF) model,

$$C_m \frac{dv}{dt} = v^2 + \eta, \quad (3)$$

with the condition $v(t) \rightarrow v_{\text{reset}}$ when $v(t) > v_{\text{th}}$. The QIF model is formally equivalent to the θ -neuron model under the transformation $v = \tan \frac{\theta}{2}$, subject to the conditions $v_{\text{reset}} \rightarrow -\infty$ and $v_{\text{th}} \rightarrow \infty$. Note that v_{reset} and v_{th} are different to the threshold and resting potentials v_θ and v_r .

The general solution to the QIF model is given as $v_{\text{QIF}}(t) = \frac{1}{2} C_m \omega \tan \left[\frac{\omega}{2} (t + c) \right]$, while the general solution to the Izhikevich

neuron model (in the absence of adaptation) is given as

$$v_{\text{IZH}}(t) = \frac{1}{2k} C_m \omega \tan \left[\frac{\omega}{2} (t + c) \right] + \frac{v_\theta + v_r}{2}, \quad (4)$$

where ω is the frequency of oscillations and c is the constant of integration (depends on initial conditions). See Supplemental Material [12], section Izhikevich neuron model solution for the derivation of this solution. To obtain the phase transformation, we note that

$$v_{\text{IZH}}(t|\omega, d) = \frac{1}{k} v_{\text{QIF}}(t|\omega, c) + \frac{v_\theta + v_r}{2}.$$

Given that $v_{\text{QIF}} = \tan \frac{\theta}{2}$, we have

$$v_{\text{IZH}}(\theta) = \frac{1}{k} \tan \frac{\theta}{2} + \frac{v_\theta + v_r}{2}.$$

Hence, the equivalent phase model for the Izhikevich neuron is given as

$$C_m \frac{d\theta}{dt} = 1 - \cos \theta + k(1 + \cos \theta) \left[\eta - u - \frac{k}{4} (v_\theta - v_r)^2 \right],$$

where the phase variable is coupled to the adaptation variable as follows:

$$\frac{du}{dt} = a \left[b \left(\frac{1}{k} \tan \frac{\theta}{2} + \frac{v_\theta - v_r}{2} \right) - u \right].$$

When $\theta(t)$ increases through π the neuron is said to spike and $u(t) \rightarrow u(t) + u_{\text{jump}}$ (spike-frequency adaptation).

III. OTT-ANTONSEN ANSATZ

We now consider a network of N globally coupled conductance-based Izhikevich neurons. For simplicity, we consider first-order synaptic coupling of the form,

$$\tau_s \frac{ds_j}{dt} = -s_j + \frac{\kappa}{N} \sum_{l=1}^N \sum_m \delta(t - t_l^m), \quad (5)$$

where $j = 1, \dots, N$, t_l^m is the m th spike time of the l th neuron and κ is the synaptic coupling strength. The results of this study could easily be extended to higher-order synaptic coupling, such as second-order alpha-type synapses.

Using the phase transformation (4), we arrive at the following equations:

$$\begin{aligned} \frac{d\theta_j}{dt} = & 1 - \cos \theta_j + k(1 + \cos \theta_j) \left[-\frac{k}{4} (v_\theta - v_r)^2 - u_j \right. \\ & \left. + \eta_j + s_j \left(v_{\text{syn}} + \frac{v_\theta + v_r}{2} \right) \right] - s_j \sin \theta_j, \end{aligned} \quad (6)$$

$$\frac{du_j}{dt} = a \left[b \left(\frac{1}{k} \tan \frac{\theta_j}{2} + \frac{v_\theta - v_r}{2} \right) - u_j \right], \quad (7)$$

for $j = 1, \dots, N$, and $u_j(t) \rightarrow u_j(t) + u_{\text{jump}}$ when $\theta_j(t)$ increases through π . To describe coordinated activity in populations of phase oscillators, such as (6), it is typical to use the Kuramoto order parameter $Z = \frac{1}{N} \sum_j e^{i\theta_j} = R(t) e^{i\phi(t)}$. This measure encapsulates the average phase of the neurons, ϕ , and the phase coherence, $|Z| = R$, i.e., the level of synchrony.

To derive a low-dimensional description of the network we make use of the Ott-Antonsen ansatz [4], which can be applied

to networks of phase oscillators whose individual dynamics take the following form:

$$\frac{d\theta}{dt} = f e^{i\theta} + h + \bar{f} e^{i\theta}. \quad (8)$$

The reduced model takes the form

$$\frac{d\alpha}{dt} = -i[\alpha^2 f + \alpha h + \bar{f}], \quad (9)$$

where $\alpha(\eta, t)$ is related to the Kuramoto order parameter as follows:

$$\bar{Z}(t) = \int_{-\infty}^{\infty} d\eta \mathcal{L}(\eta) \alpha(\eta, t) = \alpha(\eta_0 + i\Delta, t), \quad (10)$$

where $\mathcal{L}(\eta)$ is a Lorentzian distribution, with center η_0 and full width at half-maximum Δ , from which the background drives η_j are drawn.

For our Izhikevich network (6) we have

$$f = \frac{1}{2C_m}(-1 + \Pi + iks), \quad h = \frac{1}{C_m}(1 + \Pi),$$

where $\Pi = k(-\frac{k}{4}(v_\theta - v_r)^2 - u + \eta + s[v_{\text{syn}} + \frac{v_\theta + v_r}{2}])$. Subbing this into (9) and evaluating at $\eta = \eta_0 + i\Delta$, allows us define the dynamics of the Kuramoto order parameter,

$$\begin{aligned} C_m \frac{dZ}{dt} = & -i \frac{(Z-1)^2}{2} + k \frac{(Z+1)^2}{2} \left(i \left[-\frac{k}{4}(v_\theta - v_r)^2 \right. \right. \\ & \left. \left. - U + \eta_0 + s \left(v_{\text{syn}} + \frac{v_\theta + v_r}{2} \right) \right] - \Delta \right) \\ & - S \frac{Z^2 - 1}{2}, \end{aligned} \quad (11)$$

where $U = \frac{1}{N} \sum_j u_j$ is the mean adaptation,

$$\frac{dU}{dt} = a \{ b[V(Z) - v_r] - U \} + u_{\text{jump}} f(Z), \quad (12)$$

and $S = \frac{1}{N} \sum_j s_j$ is the mean synaptic conductance,

$$\tau_S \frac{dS}{dt} = -S + \kappa f(Z). \quad (13)$$

The functions $f(Z)$ and $V(Z)$ are given as

$$\begin{aligned} f(Z) &= \frac{1}{\pi C_m} \frac{1 - |Z|^2}{1 + Z + \bar{Z} + |Z|^2}, \\ V(Z) &= \frac{1}{k} \frac{\text{Im}(Z)}{1 + Z + \bar{Z} + |Z|^2} + \frac{v_\theta + v_r}{2}. \end{aligned}$$

In deriving the mean adaptation variable, we can either use the adiabatic assumption of Chen and Campbell [9] or assume that the spike-frequency adaptation is applied at the population level and use the approach of Gast *et al.* [10].

IV. MEAN-FIELD VALIDATION

The two most common, and widely studied, neurons in the human cortex are regular spiking (RS) excitatory neurons and fast spiking (FS) inhibitory neurons. For FS neurons, where spike-frequency adaptation is typically not included ($u_{\text{jump}} = 0$), the mean-field equations (11)–(14) accurately describe the evolution of the Kuramoto order parameter, the firing rate,

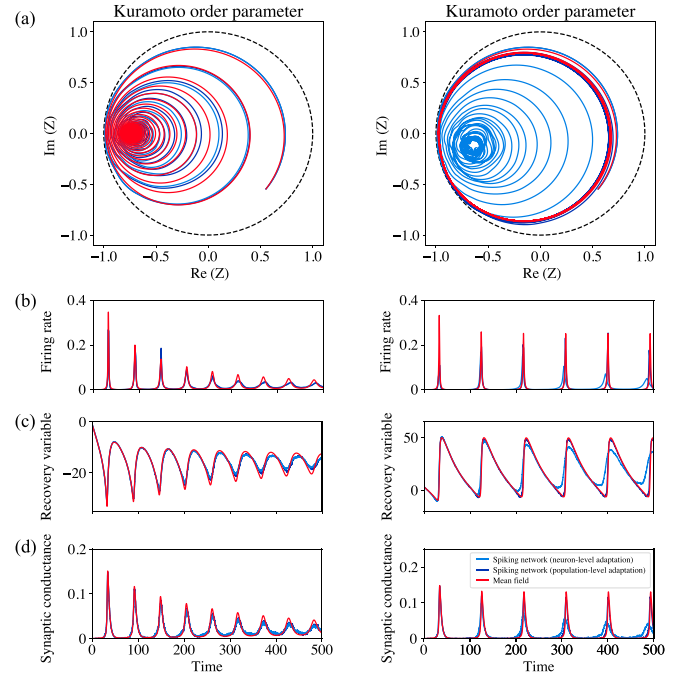


FIG. 1. Mean-field validation. Comparison of the (a) Kuramoto order parameter, (b) firing rate, (c) recovery variable, and (d) synaptic conductance for the reduced mean field model (red) and a simulation of a network of 500 regular spiking Izhikevich neurons with single neuron-level spike-frequency adaptation (light blue) and with population-level spike-frequency adaptation (dark blue). Left: Weak spike-frequency adaptation ($u_{\text{jump}} = 10$). Right: Strong spike-frequency adaptation ($u_{\text{jump}} = 100$). Parameter values: $\eta_0 = 100$, $\Delta = 1$, $C_m = 100$, $k = 0.7$, $v_\theta = -40$, $v_r = -60$, $v_{\text{syn}} = 0$, $\kappa = 10$, $\tau_S = 3$, $a = 0.03$, $b = -2$.

the population average of the recovery variable and the average synaptic conductance, respectively, for populations of, as little as, 500 Izhikevich neurons (5)–(7) (see Supplemental Material [12] Fig. S1). For RS neurons, however, the accuracy of mean-field model decreases as spike-frequency adaptation ($u_{\text{jump}} \neq 0$) is introduced. For weak spike-frequency adaptation ($u_{\text{jump}} = 10$), the mean-field dynamics (red) closely match the spiking network dynamics for the standard neuron-level adaptation, $u_j \rightarrow u_j + u_{\text{jump}}$ when $v_j(t) > v_{\text{th}}$ (light blue) [Fig. 1(a)]. While for strong spike-frequency adaptation ($u_{\text{jump}} = 100$), the mean-field dynamics no longer match the spiking network dynamics for standard neuron-level adaptation [Fig. 1(b)]. This discrepancy is a result of the mean-field description treating the adaptation as a population-level effect, i.e., the adaptation is applied to all neurons in the population rather than just the one that has spiked.

If we instead consider the rate-based adaptation of Gast *et al.* [10] in the network simulations, i.e., replace the condition $u_j \rightarrow u_j + u_{\text{jump}}$ when $v_j(t) > v_{\text{th}}$, with $u_k \rightarrow u_k + u_{\text{jump}}/N \forall k$ (for all neurons) when $v_j(t) > v_{\text{th}}$, the mean-field equations accurately describe the evolution of the average quantities for network simulations, even for strong spike-frequency adaptation. In this population-level adaptation regime, all neurons undergo spike-frequency adaptation when one neuron spikes, which is more closely related to the mean-driven adaptation employed in the mean-field model.

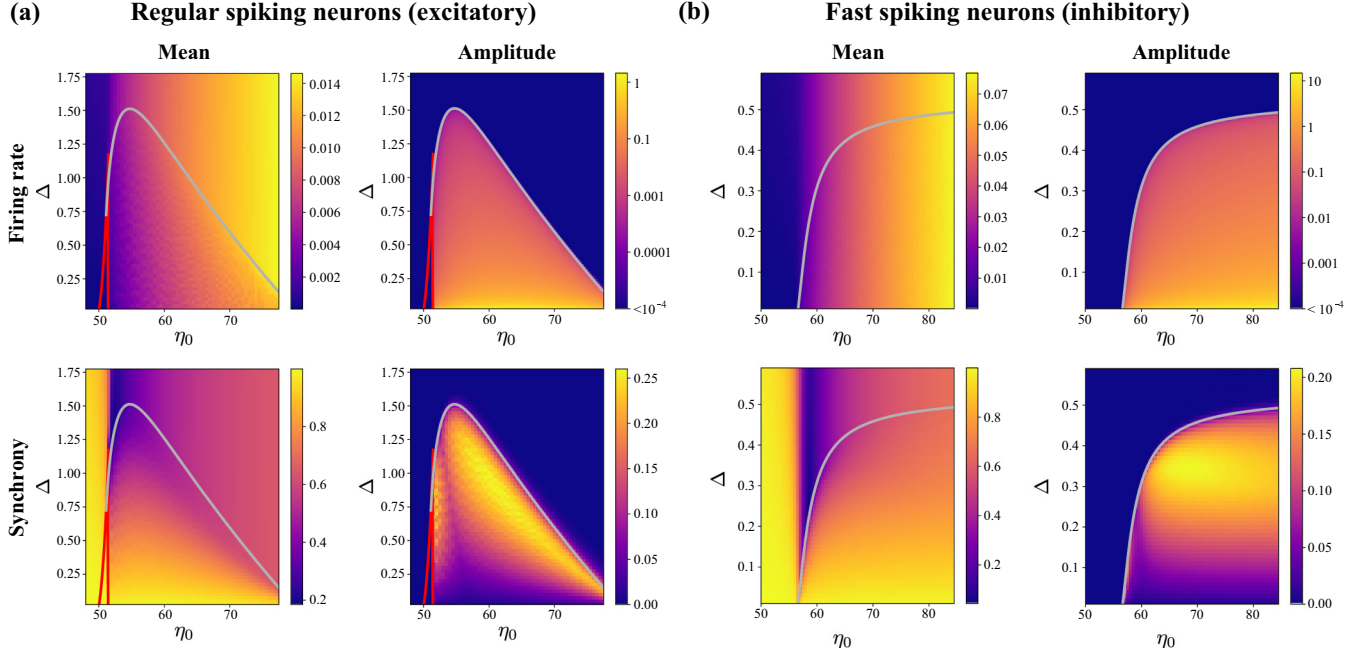


FIG. 2. Uncoupled populations of (a) regular spiking and (b) fast spiking neurons. Mean and amplitude of oscillation of the firing rate r (top) and within-population synchrony $|Z|$ (bottom) as a function of the mean background drive η_0 and heterogeneity of the drive Δ . Hopf curves are shown in gray and saddle-node curves in red. Both populations exhibit similar behaviors, the mean firing rate increases with η_0 , while the mean synchrony level decreases as Δ increases. Note that the color bar for the amplitude of the oscillations in firing rate uses a log scale, while the other panels use a linear scale. Parameters: $C_m^E = 100$, $k^E = 0.7$, $v_\theta^E = -40$, $v_r^E = -60$, $v_{\text{syn}}^E = 0$, $\kappa^E = 10$, $\tau_s^E = 3$, $a^E = 0.03$, $b^E = -2$, $u_{\text{jump}}^E = 10$, $C_m^I = 20$, $k^I = 1$, $v_\theta^I = -40$, $v_r^I = -55$, $v_{\text{syn}}^I = -65$, $\kappa^I = 4$, $\tau_s^I = 10$, $a^I = 0.2$, $b^I = 0.025$, $u_{\text{jump}}^I = 0$.

Returning to Fig. 1, we see that the spiking network dynamics with population-level adaptation (dark blue) closely matches the mean-field dynamics for both weak adaptation [Fig. 1(a)] and strong adaptation [Fig. 1(b)].

V. MAPPING TO VOLTAGE-FIRING RATE REPRESENTATION

For a network of QIF neurons, there is a direct correspondence between the Kuramoto order parameter and the macroscopic order parameter found by applying the Lorentzian ansatz to the QIF network [6]. This conformal mapping is given as

$$Z = \frac{1 - \bar{W}}{1 - W}, \quad (14)$$

where $W = \pi C_m r + iV$, where r is the population firing rate and V is the average membrane potential. The mapping (15) remains valid for the Izhikevich neuron model. However, the definition of W differs.

As the inflection point for the voltage trace is no longer at $v = 0$, the Lorentzian ansatz must be altered to account for the rescaling of v

$$\rho(t, \tilde{v}|\eta) = \frac{1}{\pi} \frac{x(t, \eta)}{[\tilde{v} - y(t, \eta)]^2 + x^2(t, \eta)}, \quad (15)$$

where $\tilde{v} = kv - \frac{1}{2}k(v_\theta + v_r)$. The macroscopic order parameter is then defined as

$$W = x + iy = C_m \pi r + ik[v - \frac{1}{2}(v_\theta + v_r)].$$

Using the modified Lorentzian ansatz (15) together with the continuity equation we obtain the following dynamics for W :

$$\frac{dW}{dt} = -iW^2 - \Delta + ik \left[\eta_0 + I_{\text{syn}} - U - \frac{k}{4}(v_\theta - v_r)^2 \right],$$

where $I_{\text{syn}} = S(v_{\text{syn}} - V)$. Although this equation differs for that proposed by Chen and Campbell [9], Gast *et al.* [10] and Guerreiro *et al.* [13], the alternative definitions for W mean that we arrive at the same equations for r and v ,

$$C_m \frac{dr}{dt} = 2krv - k(v_\theta + v_r)r + \frac{k\Delta}{C_m \pi}$$

$$C_m \frac{dv}{dt} = k(v - v_\theta)(v - v_r) + \eta_0 + I_{\text{syn}} - U - \frac{(C_m \pi)^2}{k} r^2,$$

with

$$\frac{dU}{dt} = a(b(v - v_r) - U) + u_{\text{jump}}r, \quad \tau_s \frac{dS}{dt} = -S + \kappa r.$$

VI. RELATIONSHIP BETWEEN FIRING RATE AND SYNCHRONY

A common misconception for mean-field models of neural activity is that a high firing rate implies high synchrony. Using the conformal mapping between the Kuramoto order parameter and the macroscopic firing rate and mean voltage variables, we find that high amplitude oscillations in the firing rate are instead a signature of high synchrony, where synchrony is defined as the magnitude of the Kuramoto order parameter $|Z|$ (Fig. 2). We also see high synchrony when the firing rate is low, i.e., when the majority of the neurons are synchronized

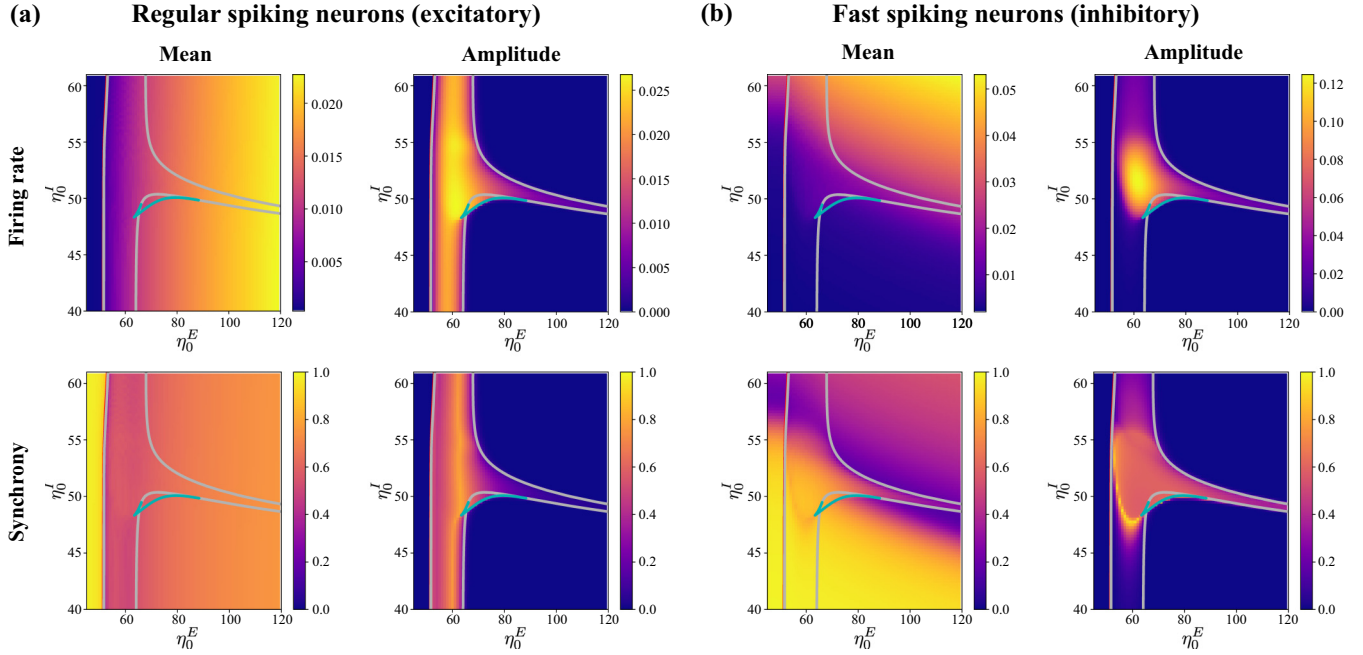


FIG. 3. Network of coupled (a) regular spiking and (b) fast spiking neurons. Mean and amplitude of oscillation of the firing rate r_m (top) and within-population synchrony $|Z_m|$ (bottom) as a function of the background drives η_0^E and η_0^I , for $m \in \{E, I\}$. Hopf curves are shown in gray, saddle-node curves in red, and the saddle node of periodic orbits in cyan. The oscillation amplitude is maximal when the fast spiking population is highly synchronised with a nonzero firing rate. Parameter values: $\Delta_E = \Delta_I = 1$, $\kappa_{EE} = 10$, $\kappa_{IE} = 4$, $\kappa_{EI} = 10$, $\kappa_{II} = 4$, all other parameters are as in Fig. 2.

at rest. Increases in synchrony are also seen as the mean firing rate increases. However, maximal synchrony is seen for parameter values that result in high amplitude oscillations in the firing rate. As expected, increasing the mean background drive (η_0) increases the firing rate and increasing the level of heterogeneity (Δ) decreases the overall level of synchrony. Interestingly, the level of heterogeneity has little effect on the mean firing rate, other than the abrupt decrease in synchrony where the firing rate becomes nonzero, the mean background drive has little effect on the synchrony. These relationships hold true for isolated populations of both excitatory RS neurons [Fig. 2(a)] and inhibitory FS neurons [Fig. 2(b)].

Also shown in Fig. 2 are the bifurcation curves. The gray curve is a Hopf curve, where oscillations are observed for parameter values under the curve. The red curve in Fig. 2(a) is a saddle node curve, with three fixed points existing between the two lines. Although the saddle node bifurcation is not present for the FS neuron parameter set, the ghost of a saddle node bifurcation can be seen in the sharp increase in firing rate and decrease in synchrony at $\eta_0 \approx 55$. Details on the numerical simulations and how the bifurcation curves were computed are given in Appendix A.

Next we look at coupled populations of RS and FS neurons (see Supplemental Material [12] for equations and parameter values). Again we investigate the relationship between firing rate and synchrony, this time as a function of the mean excitatory background drive (η_0^E) and the mean inhibitory background drive (η_0^I) (Fig. 3). For the inhibitory FS neurons, there is a clear association between high amplitude oscillations in the firing rate and high synchrony. However, the excitatory RS neurons are moderately to highly synchronized for all values of η_0^E and η_0^I , making it difficult to determine

a relationship between firing rate and synchrony in this case. For the RS neurons, changes in η_0^I have little to no effect on the mean firing rate and synchrony. Whereas, increase to η_0^E and η_0^I lead to increases in the mean firing rate for the FS neurons. The amplitude of oscillation is maximal when the FS population is highly synchronized and the firing rate of the FS is nonzero, suggesting that inhibitory neurons plays a pivotal role in generating oscillations. As in Fig. 2, the bifurcation diagram is superimposed on the color maps. The gray curves represents the Hopf bifurcations and the cyan curve is a saddle node of periodic orbits bifurcation. There is also a set of saddle node curves (red curves) close to the leftmost Hopf curve, but the window of bistability is very small and the two curves are indistinguishable.

VII. DISCUSSION

In this work, we derive an equivalent phase model for the Izhikevich neuron and apply the Ott-Antonsen ansatz to obtain a reduced description in terms of the Kuramoto order parameter. This extends the scope of next-generation neural mass models beyond quadratic integrate-and-fire (QIF) neurons to a more versatile and biologically realistic model class. By doing so, we provide a framework in which synchrony, firing rate, and mean voltage can be analyzed simultaneously in networks that reproduce a wide range of cortical firing patterns.

In validating the mean-field equations, we compared two approaches to spike-frequency adaptation previously considered in networks of Izhikevich neurons. Chen and Campbell [9] employed a neuron-level adaptation rule, whereas Gast *et al.* [10] implemented a population-level rule in which adaptation is driven by the population firing rate. Our

analysis shows that for weak spike-frequency adaptation the network dynamics produced by these two approaches are similar and can both be approximated by the same mean-field model. However, the underlying mechanisms they represent are distinct: the neuron-level rule describes intrinsic cellular processes, such as adaptation currents within individual neurons, while the population-level rule describes collective mechanisms, such as shared resource depletion or recovery. As the adaptation strength increases, these differences become more pronounced, and the two network models diverge. In this regime, only the population-level formulation remains consistent with the mean-field description. This suggests that population-level adaptation rules are better suited to mean-field reductions, as neuron-level adaptation can only be accurately described by the mean-field model when the effect of adaptation is weak.

We also demonstrated that the conformal mapping introduced by Montbrió *et al.* for QIF neurons remains valid for the Izhikevich model (once the order parameter is appropriately redefined), extending the link between phase-based and voltage–firing-rate descriptions to a broader class of neurons. This connection enables synchrony, firing rate, and voltage to be interpreted within a single theoretical framework, facilitating the key conceptual result of this study: synchrony is reflected in high-amplitude oscillations of the firing rate rather than in high mean firing rates. This finding holds for both excitatory and inhibitory populations and in coupled networks, suggesting that it generalizes beyond a single parameter set. As the conformal mapping relates the Kuramoto order parameter directly to firing-rate and voltage dynamics, the finding that synchrony relates to the oscillatory amplitude of the firing rate is a general consequence of Ott-Antonsen and Lorentzian reductions. We therefore expect it to extend to more complex networks, including those with heterogeneous excitability thresholds [10], synaptic heterogeneity [14], network substructure [15,16], stochasticity [17–19], and plasticity [20–22].

From an experimental perspective, high-amplitude oscillations in the firing rate correspond to increased spectral power in EEG and MEG recordings, and can thus provide

a direct readout of population synchrony. This identification establishes a theoretical link between mean-field theories and noninvasive brain measurements. In addition to quantifying the degree of underlying synchrony, the model allows us to infer the intrinsic firing frequencies of individual neurons. These intrinsic frequencies typically differ from the network-level oscillations measured via EEG and MEG [23], facilitating the study of how single-neuron spike rates relate to the frequency of coherent network oscillations.

Taken together, these findings extend mean-field reductions of the Izhikevich model, establish a unifying mapping between the complementary mean-field descriptions, and provide a theoretical basis for interpreting population synchrony in terms of experimentally accessible signals. We anticipate that this framework will facilitate future studies of pathological rhythms in neurological disorders, and more generally, will broaden the applicability of next-generation neural mass models to biologically realistic neuronal networks.

DATA AVAILABILITY

The data that support the findings of this article are openly available [25].

APPENDIX: NUMERICAL SIMULATIONS

All numerical simulations were performed in python. The built-in ODE solver `solve_ivp` (from the `scipy.integrate` sub-package) was used for both the network and the mean-field simulations. Bifurcation curves in Figs. 2 and 3 were computed using XPPAUT [24]. The mean-field model was simulated for a range of parameters within the neighborhood of the bifurcation curves (100×100 equally spaced grid points). The model was simulated for sufficient time to ensure transients had died away (50000 ms). Then the mean firing rate and synchrony, and amplitude of their oscillations, was calculated for the last 250 ms of the simulation. The period of the oscillations were 10–50 ms. Hence, 250 ms ensured that we captured at least five full oscillations.

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