I. MEAN-FIELD DYNAMICS OF POPULATIONS WITH DIFFERENT NEURON TYPES

Mathematical models are a necessary tool for understanding brain function and dynamics [1,2]. Due to the vast number of neurons and synapses in the brain, methods from statistical physics and mean-field theory provide a powerful tool for modeling its mesoscopic dynamics [3–5]. Classic mean-field models apply heuristic arguments derived from experimental data to propose equations that govern the evolution of averaged quantities such as population firing rates or mean postsynaptic potentials [6–9]. While these classical models have contributed to our understanding of interacting neural populations within and across brain areas, they do not account for phenomena emerging from spike synchronization, nor do they relate single-cell properties to mean-field dynamics [10,11]. A more recent formulation of mean-field theory derives a set of closed-form mean-field equations from the evolution equations of a set of all-to-all coupled spiking neurons, therefore overcoming these problems [12–14].

Mean-field equations derived from spiking neurons enable the study of the effects of heterogeneously distributed single cell parameters at the mean-field level. Unfortunately, the spiking neural networks for which mean-field equations have been derived so far are defined based on dimensionless state variables, such as the phase on the unit circle or a dimensionless representation of a membrane potential [12,13]. Here we apply this approach to the derivation of mean-field equations for networks of coupled Izhikevich (IK) neurons, which come in two different versions: a dimensionless version and a version with state variables and parameters, incorporates realistic spike resetting conditions, and accounts for heterogeneity in neural spiking thresholds. These features allow for a broad applicability of the model as well as for a direct comparison to experimental data.

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In the remainder of this article, we derive the mean-field equations for networks of all-to-all coupled IK neurons with distributed firing thresholds and analyze how the underlying simplifying assumptions affect the mean-field dynamics of a network of IK neurons. We show that the mean-field model accurately captures a wide range of dynamic regimes and phase transitions of the underlying spiking network. Furthermore, we analyze the conditions under which the mean-field predictions become less accurate. These conditions include (a) strong spike-frequency adaptation at the single cell level, (b) narrow spike reset conditions, and (c) strong neural heterogeneity. We relate these conditions to the simplifying assumptions used in the derivation of the mean-field equations, and show that even in these cases the mean-field predictions capture the qualitative properties of the bifurcation diagrams of the corresponding spiking networks, although the quantitative fit becomes worse. Finally, we provide a correction term that accounts for narrow spike reset conditions.

II. MEAN-FIELD MODELS OF COUPLED IZHIKEVICH NEURONS

A. The spiking neural network

We consider networks of coupled Izhikevich (IK) neurons of the form

\[ C \dot{v}_i = k(v_i - v_r)(v_i - v_t) - u_i + I + g_s(E - v_i), \]

\[ \tau_s \dot{u}_i = -u_i + b(v_i - v_r) + \tau_u \sum_{j \leq i} \delta(t - t_{ij}^s), \]

where \( v_i \) and \( u_i \) represent the membrane potential and the membrane recovery variable of the \( i \)th neuron in a network. This neuron is defined to spike when \( v_i \geq v_p \), where \( v_p \) is the peak membrane potential; when this condition is met, a spike is counted and \( v_i \) is reset to the reset potential \( v_r \). The recovery variable \( u_i \) is driven by two terms. The term \( \kappa \delta(t - t_{ij}^s) \) in the right-hand side of Eq. (2), where \( \delta \) is the Dirac delta function and \( t_{ij}^s \) is the time of the \( j \)th spike of the \( i \)th neuron, represents an increase of \( u_i \) by \( \kappa \) whenever the \( j \)th neuron spikes. This introduces a spike-frequency adaptation mechanism into the neuron model, since \( u_i \) enters into Eq. (1) as a hyperpolarizing variable. The term scaled by \( b \) in the right-hand side of Eq. (2) couples the recovery variable \( u_i \) to the subthreshold membrane potential fluctuations. Additional parameters that control the behavior of the neuron are the cell capacitance \( C \), the leakage parameter \( k \), the resting potential \( v_r \), the spike threshold potential \( v_p \), and the recovery variable time constant \( \tau_u \). Finally, the neuron in Eq. (1) receives two forms of input current: an extrinsic current \( I \), and a synaptic current that depends on the dimensionless synaptic activation \( s \), the maximum synaptic conductance \( g_s \), and the synaptic reversal potential \( E \). We model the synaptic activation \( s \) as the convolution of the mean-field activity of the network with an exponential activation kernel; this can be expressed as a first-order differential equation of the form

\[ \tau_s \dot{s} = -s + \frac{J}{\tau_s} \sum_{j=1}^{N} \sum_{k/t_{ij}^s} \delta(t - t_{ij}^s), \]

where \( \tau_s \) is a decay time constant, \( J \) is a global coupling constant, and the double sum in the right-hand side of the equation represents the average firing rate \( r(t) \) of the entire population at time \( t \). Thus, Eq. (3) represents the synaptic activation of each neuron in an all-to-all coupled network of \( N \) neurons.

It has been shown that the population dynamics of certain families of spiking neural networks are fully captured by their average firing rate and average membrane potential, and that their mean-field equations can be derived via the Ott-Antonsen ansatz or the equivalent Lorentzian ansatz. Most recently, a study has shown that the mean-field equations for a system of abstract, dimensionless IK neurons can be derived using a similar approximation. We will follow the latter approach to derive the mean-field equations for the heterogeneous spiking neural network given by Eqs. (1)–(3).

B. Incorporating neural heterogeneity into mean-field models

One important aspect of the spiking neural network considered here is that it allows for heterogeneity across neurons in the network. Typically, dimensionless mean-field models incorporate spiking heterogeneity by treating the input variable \( I = \eta_i + I_{ext}(t) \) as a distributed quantity, with neuron-specific background input \( \eta_i \) and global extrinsic input \( I_{ext}(t) \). The spike threshold \( v_p \) has also been related to single cell heterogeneity. While the input \( I \) enters Eq. (1) as an isolated term, the threshold \( v_p \) is multiplied by the state variable \( v_i \); a distribution of values of \( v_p \) in the population thus couples nonlinearly to the membrane potential dynamics of the neuron. So, while distributions over \( I \) can represent heterogeneity in the tonic drive to a population, distributions over \( v_p \) represent heterogeneity of the electrophysiological properties across cells within a population. Another important difference between these two sources of neural heterogeneity is their experimental accessibility. Spike thresholds can be measured in single cells via patch-clamp recordings and slow input current ramps. The form of the distributions of spike thresholds across cells can be chosen to capture the results of such recordings. On the other hand, background current distributions are a lumped representation of all input currents to a cell that are not explicitly incorporated in the model and are thus much harder to infer from neural recordings.

For these reasons, we focus on \( v_p \) as the heterogeneity parameter. The values of \( v_{p,j} \) in the network model are assumed to be neuron specific and drawn from a probability distribution \( p(v_p) \).

C. Derivation of the mean-field equations

We consider the system given by Eqs. (1)–(3) in the thermodynamic limit, i.e., when \( N \to \infty \). In this limit, the state of the system can be defined via a density function \( \rho(v, u, v_p, t) \). For a given neuron, this quantity represents the joint probability density of its spike threshold \( v_p \), membrane potential \( v \), and recovery variable \( u \) at time \( t \). The conservation of the number of neurons implies that the probability density \( \rho(v, u, v_p, t) \)
must satisfy a continuity equation
\[
\frac{\partial}{\partial t} \rho(v, u, v_{th}, t) = -\frac{\partial}{\partial v} [\rho(v, u, v_{th}, t) G^v(v, u, s, v_{th})] \]
\[
-\frac{\partial}{\partial u} \rho(v, u, v_{th}, t) G^u(v, u),
\]
where the right-hand side of Eq. (4) represents the probability flux given by the vector field
\[
G^v = \frac{1}{C} [k(v - v_r)(v - v_0) - u + I + g_E(E - v)],
\]
\[
G^u = \frac{1}{\tau_u} [b(v - v_r) - u] + \kappa \sum_{k|v|<\delta} \delta(t - t^k).
\]

The order parameters for which we wish to derive mean-field equations are the average firing rate \(r(t)\), the average membrane potential \(v(t)\), and the average recovery variable \(u(t)\), where averages are evaluated across neurons. These order parameters can be defined in terms of Eq. (4) via the following integrals:
\[
r(t) = \int_{v_0}^{\infty} \int_{u_0}^{\infty} G^v(v, u, s, v_{th})\rho(v, u, v_{th}, t) dudv_{th},
\]
\[
v(t) = \int_{v_0}^{\infty} \int_{u_0}^{\infty} v\rho(v, u, v_{th}, t) dudv_{th},
\]
\[
u(t) = \int_{v_0}^{\infty} \int_{u_0}^{\infty} u\rho(v, u, v_{th}, t) dudv_{th}.
\]

While Eqs. (8) and (9) are simply the expected values of \(u\) and \(v\), Eq. (7) represents the probability flux at \(v = v_p\) (that is, the proportion of neurons emitting a spike at time \(t\)) under the assumption that \(v_p \to \infty\) and \(v_0 \to -\infty\). We evaluate Eq. (9) by following the approach outlined in [19], which critically assumes that \(u \gg \kappa\) for any \(v_0\), that is, for any neuron in the population. This regime amounts to assuming that spike-frequency adaptation [the term in Eq. (22) that scales with \(v\)] in the model is small. Under this assumption, the dynamics of \(u(t)\) can be approximated by replacing the spike train of the individual neuron \(\sum_{k|v|<\delta} \delta(t - t^k)\) with the average firing rate across neurons \(r(t) = \frac{1}{\pi} \sum_{j=1}^{N} \sum_{k|v|<\delta} \delta(t - t^k)\); \(\tau_u \dot{u} = b(v - v_r) - u + \tau_u \kappa r\).

With this approximation, the continuity equation (4) can be integrated with respect to \(u\) to yield
\[
\frac{\partial}{\partial t} \rho(v, t|v_{th}) = -\frac{\partial}{\partial v} [G^v(v, u, v_{th})\rho(v, t|v_{th})],
\]
where we additionally used that \(\rho(v, v_{th}, t) = \rho(v, t|v_{th}) \rho(t|v_{th})\). For a more detailed description of the derivation outlined above, see [19].

To obtain expressions for \(r\) and \(v\), we apply the Lorentzian ansatz outlined in [13]. We assume that the distribution over \(v\) can be fully captured at any time \(t\) by a Lorentzian probability distribution
\[
\rho(v, t|v_{th}) = \frac{1}{\pi} \frac{x(t, v_{th})}{(v - y(t, v_{th}))^2 + x(t, v_{th})^2},
\]
centered at \(y\) and with half-width-at-half-maximum \(x\). As shown in [13], these two parameters of the Lorentzian distribution are inherently related to \(r\) and \(v\) via
\[
C \frac{\partial}{\partial t} r(t) = \int_{v_0}^{\infty} x(t, v_{th}) \rho(v_{th}) dv_{th} = x(t),
\]
\[
\frac{\partial}{\partial t} v(t) = \int_{v_0}^{\infty} y(t, v_{th}) \rho(v_{th}) dv_{th} = y(t).
\]

By plugging Eq. (12) into Eq. (11) and taking the limit of \(\tau = 0\), we find that the system dynamics can be described by a single complex variable \((z(t, v_{th}) = x(t, v_{th}) + iy(t, v_{th})\), the dynamics of which obey
\[
C \frac{\partial}{\partial t} z(t, v_{th}) = i[-kz(t, v_{th})^2 + i\alpha z(t, v_{th}) + \beta],
\]
where \(\alpha\) and \(\beta\) are defined as
\[
\alpha = k(v_r + v_0) + g_S,
\]
\[
\beta = kv_r v_0 + g_E u - u + I.
\]

Finally, to derive the equations for \(r(t)\) and \(v(t)\) from Eq. (15), we would like to solve the integral
\[
\frac{\partial}{\partial t} z(t, v_{th}) = \int_{v_0}^{\infty} \rho(v_{th}) dv_{th}.
\]
As shown in [13], this integral can be evaluated analytically if \(\rho(v_{th})\), the distribution of the heterogeneous spike threshold, is chosen to be a Lorentzian density function
\[
\rho(v_{th}) = \frac{1}{\pi} \frac{1}{(v_{th} - v_{\bar{v}})^2 + \Delta_v^2},
\]
centered at \(v_{\bar{v}}\) and with half-width-at-half-maximum \(\Delta_v\). For this choice of \(\rho(v_{th})\) we can solve Eq. (18) by evaluating \(\frac{\partial}{\partial t} z(t, v_{th}) = \int_{v_0}^{\infty} \rho(v_{th}) dv_{th}\) at the single pole of the integrand in the upper half of the complex plane \(v_{th} = v_{\bar{v}} + i\Delta_v\). Under further consideration of Eq. (13) and Eq. (14), it holds that \(z(t, v_{\bar{v}} + i\Delta_v) = x(t) + iy(t) = \frac{\pi}{k} r(t) + i u(t)\). By plugging this relationship into Eq. (18) and solving for \(r\) and \(v\), we obtain the following set of coupled ordinary differential equations:
\[
\frac{C r'}{r} = \frac{\Delta_v k^2}{\pi^2} (v - v_r) + r(k(2v - v_{\bar{v}} - v_0) - g_S),
\]
\[
\frac{C v'}{r} = kv(v - v_{\bar{v}}) - \pi Cr \left( \frac{\Delta_v}{\pi} \frac{C}{r} \right) + kv_r v_{\bar{v}} - u + I + g_E u - v,
\]
\[
\tau_u \dot{u} = b(v - v_r) - u + \tau_u \kappa r,
\]
\[
\tau_u \dot{v} = s + \tau_v J r.
\]
Accounting for this state dependence of the choice of the pole where the integral is evaluated, the mean-field equations become

\[
\dot{C}r = \frac{\Delta_r k^2 \sigma_r}{\pi C} (v - v_r) + r(k(2v - v_r - R) - gs), \quad (24)
\]
\[
\dot{C}v = kv(v - v_r - R) - \pi Ckr \left( \Delta_r \sigma_r + \frac{\pi C}{k} r \right) + k(v_r R - u) + I + gs(E - v), \quad (25)
\]
\[
\tau_a \dot{u} = b(v - v_r) - u + \tau_u kr, \quad (26)
\]
\[
\tau_s \dot{s} = -s + \tau_s Jr, \quad (27)
\]

where \( \sigma_r = \text{sgn}(v - v_r) \) is 1 when \( v - v_r < 0 \) and \(-1\) otherwise. Under the assumptions that spike-frequency adaptation is small and that spike peak and reset potentials approach the same value at different spike thresholds and study its effect on the mean-field dynamics of the spiking neural network. This modification on the assumed form of \( p(v_0) \) accounts for the biological fact that spike thresholds are confined to a finite range of potentials, bound by the resting membrane potential from below and the peak membrane potential from above.

Finally, as noted above, Eqs. (20)–(23) are valid only for regimes where \( (v - v_r) > 0 \) and are thus a special case of Eqs. (24)–(27). It is in this condition that spiking occurs in our model, and it is the regime studied in the remainder of this article. For all the parameters of interest studied here, the requirement \( v(t) - v_r > 0 \) is satisfied at all times \( t \). Thus, our comparison between the mean-field dynamics and the dynamics of spiking neural networks involves simulations of Eqs. (20)–(23) and Eqs. (1)–(3).

**D. Form of the recovery variable \( u \)**

We derived the mean-field equations (20)–(23) for a network of IK neurons with neuron-specific recovery variables \( u_i \), as defined by Eq. (2). Following the approach of [19], we showed that the mean-field dynamics of the average recovery variable \( u \) as defined by Eq. (9) are coupled to those of the average membrane potential \( v \) and average firing rate \( r \) of the population [see Eq. (22)]. This result is equivalent to the result obtained in [21,22]. Whereas a steady-state approximation was used in [21] to derive the mean-field equations, the adiabatic approximation used in [22] was based on the assumption that the dynamics of the recovery variables \( u_i \) are slow in comparison to the dynamics of the membrane potentials \( v_i \).

Strikingly, these different approaches all result in the same mean-field equation for \( u \), which is the mean-field equation derived in [23] for spiking neural networks where all neurons share a single global recovery variable \( u \). The dynamic equations of the spiking neural network considered in [23] are given by

\[
\dot{C}u_i = k(v_i - v_0)(v_i - v_0) - u + I + gs(E - v_i), \quad (28)
\]
\[
\tau_a u = -u + \frac{b}{N} \sum_{j=1}^{N} (v_j - v_i) + \frac{\tau_u k}{N} \sum_{j=1}^{N} \sum_{\ell \in J_i} \delta(t - t^i_{\ell}), \quad (29)
\]

and the dynamics of \( s \) is still controlled by Eq. (3). Although both the spiking network with neuron-specific recovery variables \( u_i \) and the spiking network with a global recovery variable \( u \) produce the same mean-field equations, it is likely that their dynamics are not identical. To examine how spiking neural networks with neuron-specific vs global recovery variables differ in their dynamics, and to determine how both spiking models differ from their mean-field approximation, we compare the dynamics of both the spiking neural network given by Eqs. (1) and (2) and the spiking neural network given by Eqs. (28) and (29) to the dynamics predicted by the mean-field model of Eqs. (20)–(23).

**III. MEAN-FIELD MODELING OF ADAPTATION-INDUCED BURSTING**

Our first assumption in deriving the mean-field model [(20)–(23)] is that spike-frequency adaptation is small, i.e., \( u \gg \kappa \) for any \( v_0 \). Here we examine how well the predictions of the mean-field theory capture the dynamics of the spiking neural network when \( \kappa \) is systematically varied. To this end, we performed a bifurcation analysis of the mean-field model over input current \( I \) and adaptation parameter \( \kappa \), using PyRates [25] and Auto-07p [26], and compared it to numerical approximations of the bifurcation structure of the spiking neural network.

To locate the bifurcation points for the spiking neural network, we performed numerical integration of the network Eqs. (1)–(3) for an all-to-all coupled population of \( N = 10000 \) neurons over a time interval of 20 s. This value is much larger than the time constant of the regular-spiking model neurons, which is \( \tau = 8 \text{ ms} \) [2]. We used the explicit Euler method with an integration step size of 0.001 ms. Over the course of the integration interval, we slowly ramped up the background current \( I(t) \) from 20 pA to 70 pA in the first 10 s and then linearly decreased it back to 20 pA in the second 10 s, resulting in a rate of change of 5.0 pA/s. We used the troughs and peaks of the recovery variable \( \langle u_i \rangle \) averaged over the population to locate fold and Hopf bifurcations, respectively, as a function of the input current \( I \). For similar approaches that used changes in the dynamics of neuronal state variables during slow parameter sweeps to identify phase transitions, see [27–30].

Figures 1(d) and 1(e) depict representative dynamics of the average recovery variable and the background current \( I \).
as used for locating fold [in Fig. 1(d)] and Hopf [in 1(e)] bifurcations. Note that we increase the ramping of the input current $I$ to 20.0pA/s for the plots in in Figs. 1(d) and 1(e), in order to make the transient dynamics of the network easier to see. For simplicity, we use $u$ to refer to the population average recovery variables of each of the three models that we compare in Figs. 1(d) and 1(e), i.e., the global recovery variable of the mean-field model given by Eq. (22), the global recovery variable given by Eq. (29), and the average of the neuron-specific recovery variables $\langle u_i \rangle$ with $u_i$ given by Eq. (2). Note that while fold bifurcations are identified as broad, single troughs in $\langle u_i \rangle$, Hopf bifurcations are located at the onset of intervals in which peaks separate multiple narrow troughs. We repeated this procedure for multiple values of the spike-frequency adaptation strength $\kappa$ to approximate the fold and Hopf bifurcation curves in the 2D parameter plane spanned by $I$ and $\kappa$. All other model parameters were set to the values reported in Table I.

Figure 1(a) shows that $\kappa$ controls whether the spiking neural network expresses a bistable or an oscillatory regime: the former exists for small values of $\kappa$, whereas the latter requires relatively large values of $\kappa$. As expected, we find that the accuracy of the mean-field model is reduced when $\kappa$ is increased and the $u \gg \kappa$ assumption of [19] is violated. The location of the fold bifurcations predicted by the mean-field theory matches the location of the fold bifurcations estimated from the spiking neural network dynamics for $1 < \kappa < 40$. However, the larger $\kappa$ becomes, the stronger is the deviation
between the bifurcation curves calculated from the mean-field model and the ones extracted from the dynamics of a spiking neural network with neuron-specific recovery variable $u_i$ [see Fig. 1(a)]. Note that the average values of $u$ do not meet the condition $u \gg \kappa$ for most values of $\kappa$ depicted in Fig. 1(a), as can be seen in the middle row of Figs. 1(d) and 1(e).

Nevertheless, we find that even for larger values of $\kappa$ the spiking neural network exhibits a bifurcation structure that is qualitatively similar to that of the mean-field model. The bistable regime is most pronounced at small values of $I$ and $\kappa$, and the oscillatory regime emerges for higher values of $I$ and $\kappa$. Violations of the small spike-frequency adaptation assumption merely lead to a shift of the bifurcation curves in parameter space; this shift increases as $\kappa$ increases. Finally, Figs. 1(d) and 1(e) demonstrate that the mean-field predictions are in better agreement with the dynamics of a spiking neuron, see [22,31,32]. This reflects the fact that the mean-field model effectively assumes that the fluctuations of the $u_i$ variable across neurons are negligible. While this assumption holds by construction in the spiking neural network with a global recovery variable $u$, it does not necessarily hold for networks with individual recovery variables $u_i$. This is particularly the case in networks with spike threshold or input current heterogeneity. Neurons with different spike thresholds will differ in their individual firing rates, which causes heterogeneity in the recovery variables $u_i$ via the dependence of $u_i$ on those firing rates, scaled by $\kappa$ [see Eq. (2)]. We conclude that spiking neural networks with neuron-specific recovery variables $u_i$ behave qualitatively similarly to spiking neural networks with a global recovery variable $u$, but that the quantitative agreement between the two becomes worse as $\kappa$ increases.

### IV. MEAN-FIELD MODELING OF DIFFERENT SPIKE WAVEFORMS

Another factor limiting the applicability of the mean-field model is the assumption that $v_p \to \infty$ and $v_0 \to -\infty$, namely that a spike is emitted as the membrane potential approaches a peak of $\infty$, and that following a spike the membrane potential resets to $-\infty$. These assumptions were necessary for the analytic derivation of the mean-field equations. However, the variety of firing patterns that the IK neuron model is able to exhibit depends on finite values of $v_p$ and $v_0$ [2,15].

In this section, we examine the mismatch between the mean-field model and spiking neural network dynamics given realistic finite values for the peak and reset potentials. To correct for this mismatch, we introduce an input rescaling factor $I^*$ that allows the mean-field model to be adapted to better match the observed dynamics of spiking neural networks with finite spike resetting parameters.

#### A. Relationship between peak/reset potential values and firing rate of the IK model neuron

We first analyze the impact of $v_p$ and $v_0$ on the dynamics of a single IK neuron. Neither parameter enters into Eq. (1); $v_p$ and $v_0$ affect the IK neuron dynamics only in the spiking regime, where spike-triggered resetting of the membrane potential takes place. It is in this regime that we examine the effect of $v_p$ and $v_0$ on the dynamics of a single IK neuron. The adiabatic approximation that $u_i$ changes infinitesimally slowly with respect to $v_i$ leads to an analytical solution to Eq. (1),

$$v_i(t) = \frac{1}{2} \left[ \sqrt{\mu_i} \tan \left( \frac{kt \sqrt{\mu_i}}{2C} \right) + \tan^{-1} \left( \frac{2(v_i(t_0) - \frac{v}{2})}{\sqrt{\mu_i}} \right) \right] + \frac{\alpha}{k},$$

(30)

where

$$\mu_i = \frac{4\beta}{k} - \left( \frac{\alpha}{k} \right)^2,$$

(31)

can be interpreted as a lumped sum of input currents to the neuron, with $\alpha = k(v_i + v_0) + g_x$ and $\beta = k v_p v_0 + g_x E - u + I$, as given by Eq. (16) and Eq. (17), respectively. For similar applications of the adiabatic approximation to neurodynamic system with multiple time scales, see [22,31,32]. We assume $\mu_i > 0$, which is equivalent to assuming that the neuron is in a spiking regime. For more detailed descriptions of the adiabatic approximation and how it can be used to absorb a spike-frequency adaptation variable into the membrane potential dynamics of a spiking neuron, see [22,31,32].

Based on Eq. (30), the spiking frequency of an IK neuron receiving a positive lumped input current $\mu > 0$ can be calculated by setting $v_i(t_0) = v_0$ and solving for the time $t$ it takes for $v_i(t)$ to reach $v_p$, yielding

$$r_i = \frac{k \sqrt{\mu_i}}{2C \gamma_i},$$

(32)

where

$$\gamma_i = \tan^{-1} \left( \frac{2v_p - \frac{v}{2}}{\sqrt{\mu_i}} \right) - \tan^{-1} \left( \frac{2v_0 - \frac{v}{2}}{\sqrt{\mu_i}} \right).$$

(33)

Equations (32) and (33) establish a functional relationship between the firing rate $r_i$ of a single IK neuron and the spike reset condition defined via $v_p$ and $v_0$.

#### B. Mean-field correction for spike resetting

To obtain a revised set of mean-field Eqs. (20)–(23) that correct for the effects of finite peak and reset potentials, we used the limits $v_p \to \infty$ and $v_0 \to -\infty$. In this limit, $\gamma_i \to \pi$ and Eq. (32) simplifies to

$$r_\infty = \frac{k \sqrt{\mu_i}}{2\pi C},$$

(34)
where $\mu^*_i$ is defined as
\[
\mu^*_i = \frac{4\beta^*}{k} - \left(\frac{\alpha}{k}\right)^2 .
\] (35)

In Eq. (35), $\beta^* = kv_r v_0 + gsE - u + I^*$, and $I^*$ is an “adjusted” extrinsic input current that can be different from $I$. The differences between $r_i$ and $r_\infty$ in the absence of an adjustment to $I$ are shown for different values of $v_0$ in Figs. 2(a) and 2(b) when $I^* = I$

These differences in the output firing rates of single neurons will cause a corresponding mismatch between the firing rates predicted by the mean-field theory and those of a spiking neural network with $v_p < \infty$ and/or $v_0 \gg -\infty$. At the single cell level, the difference in firing rates between $r_i$ and $r_\infty$ for $v_p < \infty$ and $v_0 > -\infty$ can be corrected by choosing the adjusted extrinsic input as
\[
I^* = \left\{\begin{array}{ll}
\pi k^2 \frac{\mu}{4k^2} + \frac{v^2}{4k^2} + u_i - kv_r v_0 - gsE & \text{if } \mu > 0, \\
I, & \text{otherwise.} 
\end{array}\right.
\] (36)

Figure 2(c) shows the resulting relationship between $I^*$ and $I$. It reveals that $I^* \geq I$ is required to achieve $r_\infty = r_i$ when $I$ is large enough to elicit spiking, and that the magnitude of the difference grows with $I$ and with $v_0$, which shapes $I^*$ through its contribution to $\gamma$. The piecewise structure of Eq. (36) preserves a monotonic and continuous relationship between $I$ and $I^*$. Continuity follows from evaluating $\lim_{\mu \to 0} I^*$ in Eq. (36). In this limit, the term $\pi k^2 \mu$ in Eq. (36) $\to 0$ and $I^* \to I$.

To incorporate this input adjustment into the mean-field theory, we derive the mean-field equations for a network of globally coupled IK neurons where the membrane potential of the $i$th neuron evolves according to
\[
C\dot{v}_i = k(v_i - v_\gamma)(v_i - v_{ri}) - u_i + I^* + gs(E - v_i),
\] (37) instead of Eq. (1). We use a first-order approximation to $\frac{\dot{v}_i}{v_i}$, which allows us to simplify Eq. (36) by replacing $v_0$ by $v_\gamma$ and assuming that both $\mu$ and $\gamma$ are functions of $v_\gamma$ instead of $v_0$. This approximation amounts to setting the corrected input $I^*$ to all neurons to that of the average neuron of the network, disregarding any potential effects in the mean-field dynamics due to fluctuations in $I^*$ caused by fluctuations in $v_\gamma$. Under this assumption, the mean-field equations can be derived as outlined in Sec. II C, to obtain
\[
C\dot{v} = \frac{\Delta x^2 k^2}{4\pi C} (v - v_\gamma) + r[k(2v - v_\gamma) - v] + k v_\gamma u_k - u + I^* + gs(E - v),
\] (38)
\[
C\dot{v} = kv(v - v_\gamma) - \pi Cr \left(\Delta x + \frac{\pi C}{k} r\right),
\] (39)
\[
\tau_s \dot{u} = b(v - v_r) - u + \tau_s k r,
\] (40)
\[
\tau_s \dot{s} = -s + \tau_s J r,
\] (41)

with $I^*$ given by Eq. (36) with $v_0 \to v_\gamma$. Importantly, the continuous nature of $I^*$ allows for the application of methods from dynamical systems theory such as numerical
parameter continuation. As shown in Figs. 2(d)–2(f), the correction term leads to a substantially improved agreement between the mean-field theory and the spiking neural network dynamics. Note, however, that the striking agreement between mean-field theory and spiking neural network dynamics shown in Figs. 2(e) and 2(f) holds for the optimal condition of a single population of IK neurons with $b = 0$ and $\kappa = 0$ (all other parameters were chosen according to Table I). Under these conditions, any potential mismatch that might arise due to the adiabatic approximation we used to obtain Eq. (30) can be neglected.

It should also be noted that the correction term $f^*$ becomes less accurate when $v_0 \geq v_r$, that is when the reset potential after spiking is above the resting membrane potential, as is the case in some bursty spiking neurons. Under this condition, spike resetting affects not only the firing rate but also subthreshold dynamics. Since $f^*$ as given by Eq. (36) applies a correction only when $\mu > 0$, no correction is applied in these subthreshold regimes.

**C. Effects of spike resetting on the dynamics of a two-population model**

To test whether the corrected $f^*$ can also improve the agreement between mean-field theory and spiking neural network dynamics for finite values of $v_p$ and $v_0$ under less optimal conditions, we considered a network of interacting regular-spiking neurons and fast-spiking interneurons, using the model equations and parameters reported in [23]. Regular-spiking neurons are modeled as excitatory neurons with spike-frequency adaptation, whereas fast-spiking interneurons are modeled as inhibitory interneurons without spike-frequency adaptation (see [2]). We compared the dynamics of the uncorrected and corrected mean-field models to the spiking neural network dynamics of this two-population network for three different spike reset conditions: $v_p = 1000$ mV and $v_0 = -1000$ mV, $v_p = 50$ mV and $v_0 = -100$ mV, and $v_p = 40$ mV and $v_0 = -60$ mV. Again, we used numerical bifurcation analysis to identify the bifurcation structure of the mean-field model in the 2D parameter space spanned by the background current to the fast-spiking neuron population $I_f$, and the width of the spike threshold distribution across fast-spiking neurons $\Delta_f$. To identify the location of the fold and Hopf bifurcations in the two-population spiking neuron network, we used the method described in the previous section. The comparison between Figs. 3(a)–3(c) and Figs. 3(d)–3(f) reveals that the corrected mean-field model predicts synchronized oscillations in the dynamics of the spiking neural network more accurately than the uncorrected mean-field model. This agreement is shown in Figs. 3(g) and 3(h), for spiking neural networks with $v_p = 1000$ mV and $v_0 = -1000$ mV or $v_p = 50$ mV and $v_0 = -100$ mV, respectively. Furthermore, we find that the bifurcation structure of the spiking neural network with realistic spike resetting, $v_p = 50$ mV and $v_0 = -100$ mV, follows the prediction of the corrected mean-field model [see Fig. 3(b)], while the IK network with less realistic spike resetting, $v_p = 1000$ mV and $v_0 = -1000$ mV, shows a bifurcation structure closer to that of the uncorrected mean-field model [see Fig. 3(d)]. As expected, the correction becomes less necessary as the absolute values of $v_p$ and $v_0$ become unrealistically large.

Figures 3(b), 3(c), and 3(h) further reveal that the correction term becomes less accurate as the absolute values of $v_p$ and $v_0$ decrease. The bifurcation structure predicted by the corrected mean-field model accurately captures the dynamics of the corresponding spiking neural network for $v_p = 50$ mV and $v_0 = -100$ mV, but the mean-field predictions for $v_p = 40$ mV and $v_0 = -60$ mV overestimate the areas of the oscillatory and bistable regions in the 2D parameter space spanned by $\Delta_f$ and $I_f$. This discrepancy arises because we have assumed that the firing thresholds $v_0$ of the neurons in the spiking neural network followed a heavy-tailed distribution. In the regime where the absolute values of $v_p$ and $v_0$ are small, the effective range of values for $v_0$ becomes narrower, as values such that $v_p > v_0$ or $v_0 < v_0$ are excluded. In the following section, we investigate the general issue of defining a probability distribution $p(v_0)$ on a restricted domain, and its implications for the mean-field model.

We conclude that inaccuracies between spiking network and mean-field theory arising from finite spike resetting conditions can be accounted for by introducing a corrected input term in the mean-field model. This correction term provides a substantially improved fit of the spiking network dynamics for $v_0 < v_r$ and $v_p$ large enough that the cumulative probability $\int_{v_0=p}^{v_0=\infty} p(v_0) dv_0$ is sufficiently small.

**V. MEAN-FIELD EFFECTS OF TRUNCATED DISTRIBUTIONS**

Here we consider the effects of assuming that the spiking thresholds $v_0$ in the spiking neural network follow a Lorentzian distribution across neurons, with probability density function given by Eq. (19). This assumption allows for a particularly strong reduction in the dimensionality of the mean-field equations, but it introduces a heavy-tailed distribution defined over the entire domain $[-\infty, \infty]$. The spiking threshold $v_0$ is a membrane potential, a continuous variable that can, in principle, span the unbounded domain $[-\infty, \infty]$, but the symmetry of the term $(v_i - v_r)/(v_j - v_0)$ in Eq. (1) indicates that $v_0$ effectively becomes $v_r$ and vice versa if $v_r > v_0$. So $v_0$ should be bounded from below: $v_0 > v_r > v_0$. It is evident from Eq. (1) that $v_0$ is also bounded from above, as $v_i$ could never diverge to produce a spike if $v_0 > v_p$.

Therefore in practice $v_r < v_0 < v_p$, and a truncated Lorentzian distribution must be used at the level of the spiking neural network, such that $v_r < v_0 < v_p$, $\forall i \in \{1, 2, \ldots, N\}$. We use a truncated Lorentzian that retains the symmetry of the original Lorentzian

$$p^*(v_0) = \begin{cases} g_\phi(p(v_0)) & \text{if } v_0 - \phi < v_0 < v_0 + \phi, \\ 0 & \text{otherwise,} \end{cases}$$

(42)

where $\phi$ is the truncation threshold, $g_\phi$ is a normalization constant that enforces $\int_{-\infty}^{\infty} p^*(v_0) dv_0 = 1$, and $p(v_0)$ is given by Eq. (19). We used such a truncated Lorentzian with $\phi = v_r$ for all spiking neural network results reported above. To sample from Eq. (42), we randomly sampled all spike thresholds $v_{0,i}$ using the Lorentzian density function $p(v_0)$ and resampled those neurons $i$ for which $v_{0,i}$ fell outside the truncation range. Since the mean-field derivation requires the use of
the full Lorentzian distribution, the mean-field model based on a full Lorentzian distribution over the spiking thresholds can approximate only the spiking model based on a truncated Lorentzian distribution over this model parameter.

We now examine how this approximation affects the agreement between mean-field theory and spiking neural network dynamics. To this end, we compared the average firing rate dynamics of the mean-field model to that of a spiking population of coupled regular-spiking neurons for different truncation conditions. We systematically varied either the width of the spike threshold distribution \( \Delta_s \) or the truncation threshold \( \phi \), and calculated the firing rate difference \( D_f = r - \langle r_n \rangle \). Mean-field and spiking neural network dynamics were obtained from the numerical integration of Eqs. (38)–(41) and Eqs. (1)–(3), respectively, using the parameters from Table II.

![Bifurcation structure and network dynamics in an excitatory-inhibitory network for different spike reset conditions.](image)

**FIG. 3.** Bifurcation structure and network dynamics in an excitatory-inhibitory network for different spike reset conditions. (a)–(c) 2D bifurcation diagrams in the plane of fast-spiking spike threshold heterogeneity \( \Delta_{fs} \) and fast-spiking neuron input \( I_{fs} \) for three different spike reset conditions. Regions of parameters space depicted in green (gray) represent synchronized-oscillatory (asynchronous-bistable) regimes as predicted by the corrected mean-field model. Solid green (gray) lines depict the Hopf (fold) curves predicted by the corrected mean-field model. Black diamonds represent cusp bifurcations, and green circles represent generalized Hopf bifurcations. Green (gray) shading marks depict the locations of Hopf (fold) bifurcations from the spiking neural network dynamics. (d)–(f) Same as (a)–(c), except that mean-field predictions follow from the uncorrected mean-field model. (g), (h) Synaptic activation variable \( s \) (dimensionless) as a function of time for regular \( rs \) (blue) and fast \( fs \) (orange) spiking neurons, for two different spike reset conditions. Solid lines represent the solutions of the spiking network, dashed lines represent the mean-field model. The input to the fast-spiking neurons was stepped from \( I_{fs} = 20 \) pA (no shading) to \( I_{fs} = 30 \) pA (light gray shading), to \( I_{fs} = 40 \) pA (dark gray shading). We chose \( I_{rs} = 50 \) pA, \( \Delta_{fs} = 0.3 \) mV, and all other parameters as specified in the Appendix.

**TABLE II.** Model parameters for IK neurons used to study the effects of truncated Lorentzian spike threshold distributions.

| Parameter | Value  | Parameter | Value  |
|-----------|--------|-----------|--------|
| \( C \)   | 100 pF | \( k \)   | 0.7 nS/mV |
| \( v_r \) | −80 mV | \( I \)   | 250 pA  |
| \( g \)   | 1 nS   | \( E \)   | 0 mV   |
| \( \tau_u \)| 33.33 ms | \( \tau_f \)| 6.0 pF  |
| \( \kappa \)| 20 pA  | \( b \)   | −2.0 nS |
| \( J \)   | 15     | \( N \)   | 10000  |
| \( v_{ir} - v_{ir} \)| 1000 mV |
The results of these calculations are shown in Fig. 4. As expected, either increasing the width of the spike threshold distribution $\Delta_v$ or reducing the truncation threshold $\phi$ leads to an increased average firing rate difference $(D_v)$ between mean-field prediction and spiking network dynamics [Figs. 4(b) and 4(c)]. In both cases the cumulative probability density in the truncated tails is increased, rendering the mean-field assumption less accurate. Interestingly, increases in $\Delta_v$ led to a decreased variance of $D_v$, whereas decreases in $\phi$ led to an increased variance of $D_v$. We argue that the power spectral densities of the population spiking activity depicted in Fig. 4(d) reflect a desynchronization in the neural dynamics caused by increased spike threshold heterogeneity. Specifically, we find that the power spectral densities become more flat as the spike threshold heterogeneity increases, reflecting a loss of synchrony-induced resonance frequencies [see Figs. 4(d) and 4(e)]. Both decreasing the width of the spike threshold distribution and decreasing the truncation threshold result in a more homogeneous spiking network and thus cause increased synchrony in its firing rate fluctuations. As can be seen from the difference between Figs. 4(d) and 4(e), the spectral properties of these synchronous network dynamics depend on the particular shape of the spike threshold distribution.

For a more detailed discussion of the relationship between population dynamics and spike threshold heterogeneity, see [23]. Importantly, we find that the magnitude of the average firing rate differences between mean-field model and spiking network is small compared to the firing rate fluctuations in the spiking network; this discrepancy is thus likely to have little effect on qualitative aspects of network dynamics. This is as seen in Figs. 3(a)–3(c), where increases in the width of the spike threshold distribution of fast-spiking neurons $\Delta_f$, (y axis) do not affect the accuracy of the mean-field predictions.

VI. CONCLUSION

The spiking activity of neurons is shaped by their underlying electrophysiological properties; different cell types typically exhibit dramatically different spiking responses to the same input. To understand the computational consequences of this diversity, we must study its effect at the level of neural population dynamics. Approaches such as mean-field modeling provide insight into the emergent dynamics of neural populations, but these models most commonly treat all neurons as identical copies of each other and omit physiological properties differentially associated with known cell types in the brain. Here we have presented a mean-field model of a network of coupled Izhikevich (IK) neurons with biophysiological state variables and parameters, an approach that allows us to predict how neural population dynamics are shaped by the distinct response properties of individual neurons.

A key advantage of IK model neurons is that the parameters and state variables of the model neurons, such as the membrane capacitance, the membrane potential, or the maximum conductance of synapses, are based on electrophysiological properties that can be measured directly [2,16]. Through the tuning of these parameters, the IK model can represent various neuron and synapse types, and thus account for different sources of neural heterogeneity in the brain [15,22]. These features render our mean-field model particularly well suited for interpreting neural recordings and developing large-scale models of multiple interacting neuron types [17]. Our work contributes to such efforts by providing a mean-field model that links single cell properties to population-level dynamics, thus helping to bridge different scales of brain organization [3,4,33,34].

Our model also introduces a novel approach to account for heterogeneity in neuron spike thresholds, a property of neuron populations that has been well characterized experimentally (e.g., [35–37]). As demonstrated in [23], the degree of variance in spiking thresholds across a neural population has strong effects on the dynamic regimes that these populations exhibit. We found these effects to determine the dynamic regimes of excitatory-inhibitory circuits that represent key elements of cortical organization [38–40]. Here we provided a detailed analysis of the simplifying assumptions required to derive the mean-field model for populations of heterogeneous spiking neurons and the biases that these simplifying assumptions introduce in the predicted mean-field dynamics.
For the derivation of the mean-field model, we built upon previous work that derived mean-field equations for spiking neural networks [12,13] in a manner that avoids often invoked asynchronous firing [41–44], an assumption that negates the possibility of collective oscillations. The significant progress provided by this alternative approach was initially limited by the use of abstract spiking neuron models not based on identifiable physiological parameters. Compared to the IK model, these models apply to only a limited range of neuron types and spiking patterns. While numerous studies have extended mean-field theory to account for mechanisms such as spike-frequency adaptation [32], synaptic plasticity [45,46], or gap junctions [47,48], all of these studies were based on spiking neuron models written in terms of dimensionless variables and parameters. As a result, these models do not provide a direct link between the model parameters and experimentally accessible quantities that characterize neural structure and function.

Here we presented a mean-field model that does provide such a link and may therefore be used to make experimentally testable predictions about the effect of the physiological properties of individual neurons on population dynamics. For example, we can determine how changes in neural resting potentials, spike waveforms, or rate of spike-frequency adaptation can be expected to change population responses. These predictions might be tested through direct experimental manipulation, or by studying how physiological properties naturally vary across cortical regions and layers, as in [49], and relating these differences to cell population dynamics across regions.

To assess the feasibility of using mean-field techniques to study the population dynamics of physiologically relatable neural models, we analyzed the validity of the mean-field model under violations of three key assumptions that are required for the closed form derivation presented here.

We first examined the assumptions regarding the strength of spike-frequency adaptation in the spiking neural network. Previous studies have demonstrated that spike-frequency adaptation has a critical impact on the emergence of synchronized states such as population bursting in networks of coupled excitatory neurons [31,32,50]; this is therefore an important element to include in mean-field population models. To derive the equation for the mean-field dynamics of the average recovery variable \(u\), we followed [19] in assuming that spike-frequency adaptation is weak in comparison to the magnitude of the recovery variable. As expected, we found that the violation of this assumption decreases the agreement between the predicted mean-field dynamics and the actual dynamics of the spiking neural network. However, our results suggest that the bifurcation structure of the spiking neural network is preserved in the mean-field model even when spike-frequency adaptation is strong. Although the input intensity at which bifurcations occur is shifted relative to that of the spiking network, our mean-field model nonetheless captured the emergence of synchronized and bistable states observed in spiking neural networks. We conclude that our mean-field model is a useful tool for analyzing population dynamics in the presence of spike-frequency adaptation.

We next examined the assumptions pertaining to the spike reset condition in the mean-field model. As discussed in [13,48], the derivation of the mean-field equations requires the assumption that IK neurons produce their spike when \(v_t = v_p \to \infty\); upon spiking, the IK neurons are reset to \(v_t = v_0 \to -\infty\). This assumption is particularly problematic when using biophysically realistic neuron models, as neural membrane potentials fall within a relatively narrow range [1]. Furthermore, setting \(v_0\) and \(v_p\) to specific, finite values is needed for IK model neurons to reproduce the spiking dynamics of different biological neuron types [15,17,51]. Our results indicate that imposing realistic spike reset conditions mostly leads to an increase in the average firing rate of the spiking network model relative to the mean-field model. We derived a rescaling of the background input to the network and showed that this adjustment is sufficient to correct for the increased firing rate introduced by realistic spike reset conditions. This rescaled input can be used in the mean-field equations, leading to a significantly improved agreement between mean-field and spiking neural network dynamics. We conclude that the mean-field model derived in this work can describe the mean-field dynamics of spiking networks with realistic spike reset conditions.

Finally, we examined our assumption that neural spike thresholds follow a Lorentzian distribution. In a biological system, values in the heavy tails of the Lorentzian will never be observed; the values of \(v_0\) cannot exceed the peak potential \(v_p\) or be lower than the resting potential \(v_r\). The spiking neural networks are based on a truncated Lorentzian distribution of spike thresholds with \(v_r < v_0 < v_p\), whereas the derivation of the mean-field model requires us to assume a full Lorentzian distribution. We found that the agreement between mean-field and spiking models depended on how strongly the distribution for \(v_0\) was truncated in the spiking neural network; however, the difference between mean-field predictions and spiking neural network dynamics was small in comparison to the finite-size fluctuations of the latter. Furthermore, we found a good agreement between mean-field predictions and spiking neural network dynamics in all models examined in this work, once inaccuracies caused by spike frequency adaptation or narrow spike reset conditions were accounted for. We conclude that the full Lorentzian approximation for the distribution of spike thresholds leads to mean-field predictions that reliably match the dynamics of spiking neural networks. This result implies that truncated Lorentzian distributions can be used to fit experimental measurements of spike thresholds in biological neural populations, and that our mean-field model can then be used to analyze their population dynamics. A similar approach has been used to analyze the impact of spike threshold heterogeneities in different interneuron populations on the dynamics of mesoscopic brain circuits [23]. There, the statistics of distinct spike threshold distributions measured in brain slices (see [35,52]) were fitted to truncated Lorentzian distributions and the impact of spike threshold heterogeneities on the phase transitions of mesoscopic brain circuits was analyzed.

In conclusion, we have derived and analyzed a mean-field model of interacting heterogeneous spiking neurons. Our detailed analysis of the mean-field model predictions provides a clear picture of the conditions under which the mean-field predictions can be expected to be a reliable representation of the dynamics of spiking networks. As our mean-field model
was built upon IK neural models, it provides a degree of flexibility and biophysical detail that allows it to be applied to neural recordings in a wide range of brain regions and systems.

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APPENDIX: TWO-POPULATION MODEL

In this Appendix we present the full set of equations and parameter values used for the two-population excitatory-inhibitory model. Each population is characterized by a system of three equations of the same form as Eqs. (1)–(3), with additional terms proportional to couplings $J_{a,b}$ that determine the strength of interaction within and between populations $a$ and $b$.

The spiking neural network dynamics of a system of all-to-all coupled excitatory regular-spiking and inhibitory fast-spiking neurons are given by the following:

$$
C_{rs} \frac{d}{dt} v_{rs}^{fs} = k_{rs}^{fs}(v_{rs}^{fs} - v_{rs}^{fs}) - u_{rs}^{fs} + I_{rs}^{fs} + J_{rs}^{gs} s_{gs}(E_{gs} - v_{rs}^{gs}) + J_{rs}^{gs} s_{gs}(E_{gs} - v_{rs}^{gs}),
$$
(A1a)

$$
\tau_{rs}^{fs} \frac{d}{dt} u_{rs}^{fs} = b_{rs}^{fs}\left(\sum_{j=1}^{N} v_{j}^{rs} - v_{rs}^{rs}\right) - u_{rs}^{fs} + \tau_{rs}^{fs} s_{rs}(v_{rs}^{rs} - v_{rs}^{fs}),
$$
(A1b)

$$
\tau_{rs}^{gs} \frac{d}{dt} s_{rs}^{gs} = -s_{rs}^{gs} + \frac{\tau_{rs}^{gs}}{N} \sum_{j=1}^{N} \delta(v_{j}^{rs} - v_{rs}^{rs}),
$$
(A1c)

$$
C_{fs} \frac{d}{dt} v_{fs}^{fs} = k_{fs}^{fs}(v_{fs}^{fs} - v_{fs}^{fs}) - u_{fs}^{fs} + I_{fs}^{fs} + J_{fs}^{gs} s_{gs}(E_{gs} - v_{fs}^{gs}) + J_{fs}^{gs} s_{gs}(E_{gs} - v_{fs}^{gs}),
$$
(A1d)

$$
\tau_{fs}^{fs} \frac{d}{dt} u_{fs}^{fs} = b_{fs}^{fs}\left(\sum_{j=1}^{N} v_{j}^{fs} - v_{fs}^{fs}\right) - u_{fs}^{fs} + \tau_{fs}^{fs} s_{fs}(v_{fs}^{fs} - v_{fs}^{fs}),
$$
(A1e)

$$
\tau_{fs}^{gs} \frac{d}{dt} s_{fs}^{gs} = -s_{fs}^{gs} + \frac{\tau_{fs}^{gs}}{N} \sum_{j=1}^{N} \delta(v_{j}^{fs} - v_{fs}^{fs}),
$$
(A1f)

where the superscripts $rs$ and $fs$ denote regular-spiking and fast-spiking populations, respectively. This model has four coupling terms $J_{a,b}$, $a, b \in \{r, f\}$, reflecting the strength of input from a given population $b$ to a given recipient population $a$. The mean-field equations for this two-population model can be derived following the procedure outlined in the main text to obtain

$$
C_{rs} \frac{d}{dt} r_{rs}^{fs} = \frac{\Delta_{rs}^{rs}(k_{rs}^{fs})^{2}}{\pi C_{rs}^{fs}}(v_{rs}^{rs} - v_{rs}^{rs}) + r_{rs}^{fs}(2v_{rs}^{rs} - v_{rs}^{rs} - \bar{v}_{rs}^{rs}) - J_{rs}^{gs} s_{gs}(E_{gs} - v_{rs}^{gs}),
$$
(A2a)

$$
C_{fs} \frac{d}{dt} r_{fs}^{fs} = k_{fs}^{fs}(v_{fs}^{fs} - v_{fs}^{fs}) + k_{fs}^{fs} v_{fs}^{fs} \bar{v}_{fs}^{rs} - \pi C_{fs}^{fs}(\Delta_{fs}^{fs} + \frac{\pi C_{fs}^{fs}}{k_{fs}^{fs}}) - u_{fs}^{fs} + I_{fs}^{fs},
$$
(A2b)

$$
\tau_{rs}^{rs} \frac{d}{dt} u_{rs}^{rs} = b_{rs}^{rs}(v_{rs}^{rs} - v_{rs}^{rs}) - u_{rs}^{rs} + \tau_{rs}^{rs} k_{rs}^{fs} v_{rs}^{fs},
$$
(A2c)

$$
\tau_{rs}^{fs} \frac{d}{dt} s_{rs}^{fs} = -s_{rs}^{fs} + \tau_{rs}^{fs} r_{rs}^{fs},
$$
(A2d)

$$
C_{fs} \frac{d}{dt} s_{fs}^{fs} = \frac{\Delta_{fs}^{fs}(k_{fs}^{fs})^{2}}{\pi C_{fs}^{fs}}(v_{fs}^{fs} - v_{fs}^{fs}) + r_{fs}^{fs}(2v_{fs}^{fs} - v_{fs}^{fs} - \bar{v}_{fs}^{rs}) - J_{fs}^{gs} s_{gs}(E_{gs} - v_{fs}^{gs}),
$$
(A2e)

$$
C_{rs} \frac{d}{dt} v_{rs}^{fs} = k_{rs}^{fs}(v_{rs}^{fs} - v_{rs}^{fs}) + k_{rs}^{fs} v_{fs}^{fs} \bar{v}_{rs}^{rs} - \pi C_{rs}^{fs}(\Delta_{rs}^{fs} + \frac{\pi C_{rs}^{fs}}{k_{rs}^{fs}}) - u_{rs}^{fs} + I_{rs}^{fs},
$$
(A2f)

$$
\tau_{fs}^{rs} \frac{d}{dt} u_{rs}^{rs} = b_{rs}^{rs}(v_{rs}^{rs} - v_{rs}^{rs}) - u_{rs}^{rs} + \tau_{fs}^{rs} k_{fs}^{fs} r_{fs}^{fs},
$$
(A2g)

$$
\tau_{fs}^{fs} \frac{d}{dt} s_{fs}^{fs} = -s_{fs}^{fs} + \tau_{fs}^{fs} r_{fs}^{fs},
$$
(A2h)

The default parameters for the regular-spiking neurons in Table I represent a version of the regular-spiking neuron

| TABLE III. Model parameters for a fast-spiking IK neuron. |
|---------------------------------------------|-----------------|-----------------|
| Parameter | Value | Parameter | Value |
| $C$ | 20 pF | $k$ | 1.0 nS/mV |
| $v_r$ | -55 mV | $\bar{v}_r$ | -40 mV |
| $g$ | 1 nS | $E$ | -65 mV |
| $\tau_u$ | 5.0 ms | $\tau_f$ | 8.0 ms |
| $\kappa$ | 0 pA | $b$ | 0.025 nS |
| $J$ | 15 | $N$ | 10000 |
| $v_{p\rightarrow r}$ | 1000 mV | $\Delta_v$ | 1 mV |

| TABLE IV. Coupling strengths for the two-population model. |
|---------------------------------------------|-----------------|-----------------|
| Parameter | Value | Parameter | Value |
| $J_r$ | 16 | $J_f$ | 16 |
| $J_f$ | 4 | $J_{r,f}$ | 4 |
suggested in [2], modified here to exhibit reduced spike-frequency adaptation and different values of the peak and reset potentials.

The default parameters for the fast-spiking neurons in Table III represent a version of the fast-spiking neuron suggested in [2], modified here to exhibit different values of the peak and reset potentials.

The synaptic connection strengths used for networks of coupled regular- and fast-spiking neurons are reported in Table IV.

[1] P. Dayan and L. F. Abbott, *Theoretical Neuroscience: Computational and Mathematical Modeling of Neural Systems* (MIT Press, Cambridge, MA, 2001).

[2] E. M. Izhikevich, *Dynamical Systems in Neuroscience* (MIT Press, Cambridge, MA, 2007).

[3] G. Deco, V. K. Jirsa, P. A. Robinson, M. Breakspear, and K. J. Friston, The dynamic brain: From spiking neurons to neural masses and cortical fields, *PLoS Comput. Biol.* 4, e1000902 (2008).

[4] S. Coombes, Large-scale neural dynamics: Simple and complex, *NeuroImage* 52, 731 (2010).

[5] D. R. Chialvo, Emergent complex neural dynamics, *Nat. Phys.* 6, 744 (2010).

[6] H. R. Wilson and J. D. Cowan, Excitatory and inhibitory interactions in localized populations of model neurons, *Biophys. J.* 12, 1 (1972).

[7] F. H. Lopes da Silva, A. Hoeks, H. Smits, and L. H. Zetterberg, Model of brain rhythmic activity, *Kybernetik* 15, 27 (1974).

[8] B. H. Jansen and V. G. Rit, Electroencephalogram and visual evoked potential generation in a mathematical model of coupled cortical columns, *Biol. Cybern.* 73, 357 (1995).

[9] P. A. Robinson, C. J. Rennie, and J. J. Wright, Propagation and stability of waves of electrical activity in the cerebral cortex, *Phys. Rev. E* 56, 826 (1997).

[10] F. Deville, A. Roxin, and E. Montbrió, Firing rate equations require a spike synchrony mechanism to correctly describe fast oscillations in inhibitory networks, *PLoS Comput. Biol.* 13, e1005881 (2017).

[11] S. Coombes and A. Byrne, Next generation neural mass models, in *Nonlinear Dynamics in Computational Neuroscience*, edited by F. Corinato and A. Torcini, PoliTO Springer Series (Springer, Cham, 2019), pp. 1–16.

[12] T. B. Luke, E. Barreto, and P. So, Complete classification of the macroscopic behavior of a heterogeneous network of Theta neurons, *Neural Comput.* 25, 3207 (2013).

[13] E. Montbrió, D. Pazó, and A. Roxin, Macroscopic Description for Networks of Spiking Neurons, *Phys. Rev. X* 5, 021028 (2015).

[14] C. Bick, M. Goodfellow, C. R. Laing, and E. A. Martens, Understanding the dynamics of biological and neural oscillator networks through exact mean-field reductions: A review, *J. Math. Neurosci.* 10, 9 (2020).

[15] E. Izhikevich, Simple model of spiking neurons, *IEEE Trans. Neural Netw.* 14, 1569 (2003).

[16] E. Izhikevich, Which model to use for cortical spiking neurons? *IEEE Trans. Neural Netw.* 15, 1063 (2004).

[17] E. M. Izhikevich and G. M. Edelman, Large-scale model of mammalian thalamocortical systems, *Proc. Natl. Acad. Sci. USA* 105, 3593 (2008).

[18] E. Ott and T. M. Antonsen, Low dimensional behavior of large systems of globally coupled oscillators, *Chaos* 18, 037113 (2008).

[19] L. Chen and S. A. Campbell, Exact mean-field models for spiking neural networks with adaptation, *J. Comput. Neurosci.* 50, 445 (2022).

[20] S. Rich, H. Moradi Chameh, J. Lefebvre, and T. A. Valiante, Loss of neuronal heterogeneity in epileptogenic human tissue impairs network resilience to sudden changes in synchrony, *Cell Rep.* 39, 110863 (2022).

[21] W. Nicola and S. A. Campbell, Bifurcations of large networks of two-dimensional integrate and fire neurons, *J. Comput. Neurosci.* 35, 87 (2013).

[22] I. Guerreiro, M. di Volo, and B. Gutkin, Exact reduction methods for networks of neurons with complex dynamic phenotypes, arXiv:2206.10370 (2022).

[23] R. Gast, S. A. Solla, and A. Kennedy, Effects of neural heterogeneity on spiking neural network dynamics, arXiv:2206.08813 (2022).

[24] Y. A. Kuznetsov, *Elements of Applied Bifurcation Theory* (Springer Science & Business Media, New York, 2013).

[25] R. Gast, D. Rose, C. Salomon, H. E. Möller, N. Weiskopf, and T. R. Knösche, PyRates—A Python framework for rate-based neural simulations, *PLoS ONE* 14, e0225900 (2019).

[26] E. J. Doedel, T. F. Fairgrieve, B. Sandstede, A. R. Champneys, Y. A. Kuznetsov, and X. Wang, AUTO-07P: Continuation and bifurcation software for ordinary differential equations, Tech. Rep. (2007), http://indy.cs.concordia.ca/auto.

[27] E. M. Izhikevich, Resonate-and-fire neurons, *Neural Netw.* 14, 883 (2001).

[28] A. Spiegler, S. J. Kiebel, F. M. Atay, and T. R. Knösche, Bifurcation analysis of neural mass models: Impact of extrinsic inputs and dendritic time constants, *NeuroImage Comput. Models Brain* 52, 1041 (2010).

[29] C. Cakan and K. Obermayer, Biophysically grounded mean-field models of neural populations under electrical stimulation, *PLoS Comput. Biol.* 16, e1007822 (2020).

[30] K. Gasior, K. Korshunov, P. Q. Trombley, and R. Bertram, Fast-slow analysis as a technique for understanding the neuronal response to current ramps, *J. Comput. Neurosci.* 50, 145 (2022).

[31] G. Gigante, M. Mattia, and P. Del Giudice, Diverse Population-Bursting Modes of Adapting Spiking Neurons, *Phys. Rev. Lett.* 98, 148101 (2007).

[32] R. Gast, H. Schmidt, and T. R. Knösche, A mean-field description of bursting dynamics in spiking neural networks with short-term adaptation, *Neural Comput.* 32, 1615 (2020).

[33] A. Engel, C. Gerloff, C. Hilgetag, and G. Nolte, Intrinsic coupling modes: Multiscale interactions in ongoing brain activity, *Neuron* 80, 867 (2013).

[34] J. Vohryzek, J. Cabral, P. Vuust, G. Deco, and M. L. Kringlebach, Understanding brain states across spacetime informed by whole-brain modelling, *Philos. Trans. R. Soc. A* 380, 20210247 (2022).

[35] Y. Wang, M. Toledo-Rodriguez, A. Gupta, C. Wu, G. Silberberg, J. Luo, and H. Markram, Anatomical, physiological
and molecular properties of Martinotti cells in the somatosensory cortex of the juvenile rat, J. Physiol. 561, 65 (2004).

[36] W. Yang, Y. Carrasquillo, B. M. Hooks, J. M. Nerbonne, and A. Burkharter, Distinct balance of excitation and inhibition in an interareal feedforward and feedback circuit of mouse visual cortex, J. Neurosci. 33, 17373 (2013).

[37] G. T. Neske, S. L. Patrick, and B. W. Connors, Contributions of diverse excitatory and inhibitory neurons to recurrent network activity in cerebral cortex, J. Neurosci. 35, 1089 (2015).

[38] T. C. Potjans and M. Diesmann, The cell-type specific cortical microcircuit: Relating structure and activity in a full-scale spiking network model, Cereb. Cortex 24, 785 (2014).

[39] T. Schwalger, M. Deger, and W. Gerstner, Towards a theory of cortical columns: From spiking neurons to interacting neural populations of finite size, PLoS Comput. Biol. 13, e1005507 (2017).

[40] Z. Jonke, R. Legenstein, S. Habenschuss, and W. Maass, Feedback inhibition shapes emergent computational properties of cortical microcircuit motifs, J. Neurosci. 378511 (2017).

[41] D. J. Amit and N. Brunel, Model of global spontaneous activity and local structured activity during delay periods in the cerebral cortex, Cereb. Cortex 7, 237 (1997).

[42] C. v. Vreeswijk and H. Sompolinsky, Chaotic balanced state in a model of cortical circuits, Neural Comput. 10, 1321 (1998).

[43] N. Brunel, Dynamics of sparsely connected networks of excitatory and inhibitory spiking neurons, J. Comput. Neurosci. 8, 183 (2000).

[44] S. El Boustani and A. Destexhe, A master equation formalism for macroscopic modeling of asynchronous irregular activity states, Neural Comput. 21, 46 (2009).

[45] H. Taher, A. Torcini, and S. Olmi, Exact neural mass model for synaptic-based working memory, PLoS Comput. Biol. 16, e1008533 (2020).

[46] R. Gast, T. R. Knösche, and H. Schmidt, Mean-field approximations of networks of spiking neurons with short-term synaptic plasticity, Phys. Rev. E 104, 044310 (2021).

[47] B. Pietras, F. Devalle, A. Roxin, A. Daffertshofer, and E. Montbrió, Exact firing rate model reveals the differential effects of chemical versus electrical synapses in spiking networks, Phys. Rev. E 100, 042412 (2019).

[48] E. Montbrió and D. Pazò, Exact Mean-Field Theory Explains the Dual Role of Electrical Synapses in Collective Synchronization, Phys. Rev. Lett. 125, 248101 (2020).

[49] R. D. Hodge, T. E. Bakken, J. A. Miller, K. A. Smith, E. R. Barkan, L. T. Graybuck, J. L. Close, B. Long, N. Johansen, O. Penn et al., Conserved cell types with divergent features in human versus mouse cortex, Nature (London) 573, 61 (2019).

[50] G. Fuhrmann, H. Markram, and M. Tsodyks, Spike frequency adaptation and neocortical rhythms, J. Neurophysiol. 88, 761 (2002).

[51] M. D. Humphries, R. Wood, and K. Gurney, Dopamine-modulated dynamic cell assemblies generated by the GABAergic striatal microcircuit, Neural Netw. Cortical Microcircuits 22, 1174 (2009).

[52] D. Lau, E. V.-S. d. Miera, D. Contreras, A. Ozaita, M. Harvey, A. Chow, J. L. Noebels, R. Paylor, J. I. Morgan, C. S. Leonard, and B. Rudy, Impaired fast-spiking, suppressed cortical inhibition, and increased susceptibility to seizures in mice lacking Kv3.2 K⁺ channel proteins, J. Neurosci. 20, 9071 (2000).