Exact mean-field models for spiking neural networks with adaptation

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Received: 22 March 2022 / Accepted: 15 June 2022 / Published online: 14 July 2022
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Abstract
Networks of spiking neurons with adaptation have been shown to be able to reproduce a wide range of neural activities, including the emergent population bursting and spike synchrony that underpin brain disorders and normal function. Exact mean-field models derived from spiking neural networks are extremely valuable, as such models can be used to determine how individual neurons and the network they reside within interact to produce macroscopic network behaviours. In the paper, we derive and analyze a set of exact mean-field equations for the neural network with spike frequency adaptation. Specifically, our model is a network of Izhikevich neurons, where each neuron is modeled by a two dimensional system consisting of a quadratic integrate and fire equation plus an equation which implements spike frequency adaptation. Previous work deriving a mean-field model for this type of network, relied on the assumption of sufficiently slow dynamics of the adaptation variable. However, this approximation did not succeed in establishing an exact correspondence between the macroscopic description and the realistic neural network, especially when the adaptation time constant was not large. The challenge lies in how to achieve a closed set of mean-field equations with the inclusion of the mean-field dynamics of the adaptation variable. We address this problem by using a Lorentzian ansatz combined with the moment closure approach to arrive at a mean-field system in the thermodynamic limit. The resulting macroscopic description is capable of qualitatively and quantitatively describing the collective dynamics of the neural network, including transition between states where the individual neurons exhibit asynchronous tonic firing and synchronous bursting. We extend the approach to a network of two populations of neurons and discuss the accuracy and efficacy of our mean-field approximations by examining all assumptions that are imposed during the derivation. Numerical bifurcation analysis of our mean-field models reveals bifurcations not previously observed in the models, including a novel mechanism for emergence of bursting in the network. We anticipate our results will provide a tractable and reliable tool to investigate the underlying mechanism of brain function and dysfunction from the perspective of computational neuroscience.

Keywords Neural network · Mean field · Integrate and fire · Adaptation · Bursting · Bifurcation

1 Introduction
A central topic of computational neuroscience research is to obtain computationally and/or analytically tractable models for understanding the neural dynamics that underpin brain disorders, such as epilepsy or Parkinson’s disease, or for normal functioning, such as memory or decision making. The dynamics associated with such functions or disorders result from the coordinated activity of large populations of interconnected neurons. Neural mass models, rooted in the mean field theory, aim to describe the collective activity of a neural network in terms of mean-field variables such as the population firing rate and the mean membrane potential. The development of mean-field models have been a long history spanning more than a half-century (Deco et al., 2008). In the current literature, there are two common types of model. One is based on heuristic descriptions that are designed to resemble macroscopic features of neural dynamics based on physiological observations and disregard individual behaviours of the neural network. The Wilson-Cowan model (Wilson & Cowan, 2021) is arguably the most influential
one. As an alternative, exact macroscopic descriptions have been developed through mean-field reduction of models of neural networks by using concepts from statistical physics. These models bridge the microscopic properties of individual neurons and macroscopic collective dynamics of the neural network. Thus they can account for phenomena that the heuristic models cannot, such as synchronization mechanisms that arise due to the interaction between individual neuron behaviours and network properties. The development of exact mean-field models has followed two threads, both based on the population density approach from statistical physics. This approach yields a conservation law for the population density function, which exactly represents the dynamics of the network in the limit that the number of elements becomes arbitrarily large. In one thread, this approach was applied to networks of spiking neurons (Abbott & van Vreeswijk, 1993; Treves, 1993; Nykamp & Tranchina, 2000; Omurtag et al., 2000; Apfaltrer et al., 2006; Augustin et al., 2017; Cakan & Obermayer, 2020). In the other thread, this approach was applied to networks of coupled phase models (Strogatz & Mirollo, 1991; Watanabe & Strogatz, 1993, 1994; Ott & Antonsen, 2008, 2009; Ott et al., 2011; Pikovsky & Rosenblum, 2008). Recently, these two threads have started to converge when it was shown that the OA ansatz (Ott & Antonsen, 2008, 2009; Ott et al., 2011) could be applied to networks of coupled Quadratic Integrate-and-Fire (QIF) neurons using the link between the QIF model and the theta model (Montbrió et al., 2015).

Spiking neural models involve variables closely related to biological measurements and realizations. The QIF model is a popular model for large network simulations as it only has one equation for each neuron. Further, it can be considered a canonical model as any Class I excitable system close enough to the onset of oscillations can be transformed into this form (Ermentrout & Kopell, 1986; Ermentrout, 1996; Latham et al., 2000). Following the idea of Montbrió et al. (2015), recent work has focused on developing mean-field models for QIF networks with added biophysical mechanisms or structural details to explain interesting neural activities. These additions include the incorporation of propagation delays as action potentials travel along axons or dendrites (Ratas & Pyragas, 2018; Pazó & Montbrió, 2016), gap junctions between neurons (Montbrió & Pazó, 2020) and synaptic plasticity (Gast et al., 2021; Taher et al., 2022). More information on mean-field models of such extended neural networks can be found in recent reviews (Ashwin et al., 2016; Bick et al., 2020).

While the QIF model is useful, there are many behaviours of spiking neurons it cannot reproduce. To address this, several authors have developed two-dimensional integrate-and-fire model neurons. Examples include the Izhikevich neuron (Izhikevich, 2003) and the adaptive exponential (AdEx) neuron (Brette & Gerstner, 2005). These models display spike frequency adaptation (SFA) through a recovery variable and are capable of generating a variety of spiking dynamics reported in real neurons (Izhikevich, 2004; Touboul, 2008; Gerstner et al., 2014). The SFA mechanism can improve neural coding and computation at a lower metabolic cost (Gutierrez & Denève, 2019; Fitz et al., 2020; Salaj et al., 2021) and has also been demonstrated to be important in the emergence of network bursting and synchronization (Vreeswijk & Hansel, 2001; Nesse et al., 2008; Kilpatrick & Ermentrout, 2011; Ferguson et al., 2015; Gast et al., 2020). Thus, derivation of mean-field descriptions for networks of neurons with SFA would be extremely valuable. There has been some recent work in this direction using different approaches than what we describe in this paper (Augustin et al., 2017; Cakan & Obermayer, 2020; Carlu et al., 2020; di Volo & Destexhe, 2021). The key differences lie in the neural models that can be studied, the types of approximations used in deriving the mean-field description and the type of behaviours the mean-field model can be used to study.

Since the Izhikevich neuron is the most closely linked to the QIF neuron, it is the ideal candidate to explore emergent neurodynamics of neural networks through mean-field descriptions. Further, the Izhikevich network has been widely employed to study brain function, e.g., (Izhikevich & Edelman, 2008; Dur-e-Ahmad et al., 2012) and dysfunction, e.g., (Rich et al., 2020). The Izhikevich neuron model consists of a fast subsystem based on the QIF model and a slow subsystem modelling the adaptation mechanism. Thus the Izhikevich network can be considered as a QIF network extended by SFA. In this paper, we show it is feasible to extend the Lorentzian ansatz (Montbrió et al., 2015), or the equivalent OA ansatz (Ott & Antonsen, 2008) for the phase model, to the derivation of the exact mean-field models for a network of Izhikevich neurons. The difficulty lies in how to achieve a closed set of mean-field equations with the inclusion of the mean-field expression of the adaptation variable. To do this we turn to the population density approach for spiking neurons, which was extended to two dimensional integrate-and-fire models by Nicola and Campbell (2013a, b). The quasi-steady approximation for the continuity equation in (Nicola & Campbell, 2013b) will be replaced with the Lorentzian ansatz (Montbrió et al., 2015) to drop the assumption of separation of time scales. The moment closure approach (Ly & Tranchina, 2007) will be deployed to release the dependence of the adaptation variable on the membrane potential to help close the mean-field system.

We apply our method to two examples. The first consists of a single population of identical neurons with all-to-all coupling and heterogeneous input currents. We confirm that the bifurcation structure is similar to that observed in (Nicola & Campbell, 2013b). In our model, the frequency mismatch between periodic orbits of the network and the mean-field model which occurs in (Nicola & Campbell, 2013b) has
been greatly improved. Further, we give evidence for a co-dimension two bifurcation that organizes the system behaviour. The second model is a network consisting of two populations of neurons. Both populations are all-to-all coupled internally and externally. One population has strong SFA while the other has weak SFA. We explore the bifurcations that occur as the relative proportions of the two neurons is varied and show that multiple co-dimension two bifurcations occur. One interesting consequence is that the onset of bursting in the network can change from a subcritical Hopf bifurcation of the population firing rate to a homoclinic bifurcation or saddle node on an invariant circle bifurcation.

The paper is structured as follows. In Sect. 2, we introduce the specific neuron model and network which will be used throughout. In Sect. 3, we present a detailed derivation of the mean-field equations for a neural network with a single population. The assumptions are added on one after the other to show their role in forming the final mean-field model. In Sect. 4, we perform a bifurcation analysis of the above mean-field system and show, by comparison with numerical simulation of a finite-size neural network, that the mean-field model has excellent agreement with the network behaviour. In Sect. 5, we extend the model to a network consisting of two populations of neurons and then apply the approach to the example described above. Numerical simulations and numerical bifurcation diagrams demonstrate the validity of the developed mean-field description. Finally, in Sect. 6 we conclude the paper by discussing the influence of assumptions required in our approach on the accuracy and efficacy of the mean-field models, and what can be done to extend this approach when these assumptions cannot be met.

2 The network system

The Izhikevich model is a result of reduction of the biophysically accurate Hodgkin-Huxley type neuron model through bifurcation analysis (Izhikevich, 2007). It still retains central properties of neural activity with adaptive quadratic mechanism. The network model for a population of Izhikevich neurons is described by the following discontinuous ordinary differential equations (ODEs),

\[
\begin{align*}
v'_k &= v_k(v_k - a) - w_k + \eta_k + I_{\text{ext}} + I_{\text{syn},k} \\
&= b(v_k - \theta) \\
&= a(bv_k - w_k) \\
&\text{if } v_k \geq v_{\text{peak}}, \text{ then } v_k \leftarrow v_{\text{reset}} \\
&\text{and } w_k \leftarrow w_k + w_{\text{jump}}
\end{align*}
\]

for \( k = 1, 2, \ldots, N \). Here, \( t = d/dt \) denotes the time derivative, \( v_k(t) \) is the membrane potential of \( k \)th neuron and \( w_k \) is the recovery current, which serves as an adaptation variable. By construction, \( v_k \in (-\infty, v_{\text{peak}}] \) while \( w \) has no constraints, \( w_k \in (-\infty, \infty) \). The parameter \( \eta_k \) is the intrinsic current while \( I_{\text{ext}} \) is the external common current. We will assume that \( \eta_k \) are heterogeneous and drawn from a distribution \( \mathcal{L}(\eta) \) defined on \((-\infty, \infty)\). The term \( I_{\text{syn}} \) represents the total synaptic current due to the other neurons in the network. When the voltage reaches a cut off value \( v_{\text{peak}} \), considered to be the peak of a spike, it is reset to the value \( v_{\text{reset}} \). At the same time, the adaptation variable jumps by an amount \( w_{\text{jump}} \) which affects the after spike behavior. When \( v_{\text{peak}} = -v_{\text{reset}} \) approaches \( \infty \), the Izhikevich model (1) can be transformed into the theta model with adaptation, where the neuron fires a spike whenever \( \theta \) crosses \( \pi \) (Izhikevich, 2000).

Neurons in the network are connected by synapses using the standard synaptic current model (Ermentrout & Terman, 2010)

\[
I_{\text{syn},k} = g_{\text{syn}}(e_r - v_k),
\]

where \( e_r \) is the reversal potential and \( g_{\text{syn}} \) the maximum synaptic conductance, both assumed to be the same for all neurons. The synaptic gating variable, \( s_k \), lies between 0 to 1 and represents the proportion of ion channels open in the postsynaptic neuron as the result of the firing in presynaptic neurons. For simplicity, we assume that neurons are all-to-all coupled. This assumption has been widely used in the literature (Ermentrout & Terman, 2010; Montbrió et al., 2015; Byrne et al., 2020). Specifically, it is reasonable for the application to the CA3 region of hippocampus we will consider later, since this region is highly recurrently coupled (Amaral & Witter, 1989; Andersen et al., 2006; Buzsáki, 2011). We will discuss the necessity of all the assumptions we make and possible extensions that relax some of these requirements in Sect. 6. For a network with all-to-all connectivity, \( s_k \) is homogeneous across the network as every postsynaptic neuron receives the same summed input from all the presynaptic neurons, thus \( s_k = s \). The mechanism of synaptic transmission can be formally described by a linear system of ODEs with a sum of delta pulses corresponding to the times a neuron fires a spike (Ermentrout & Terman, 2010). For example, the single exponential synapse is modeled by

\[
s' = -s / \tau_s + \sum_{j=1}^{N} \sum_{k,j<k} \delta(t - t'_j),
\]

where \( \delta(t) \) is the Dirac delta function, and \( t'_j \) represents the time of the \( j \)th spike of the \( k \)th neuron. The double exponential synapse and the alpha synapse are also frequently used in the literature. Their dynamics can be described by two-coupled first-order ODEs. For simplicity, we consider the single exponential synaptic dynamics (3) in this paper. It is easy to extend our work to other synaptic models, see (Nicola & Campbell, 2013a). We assume that the synaptic parameters \( \delta_{\text{jump}} \) and \( \tau_s \) are the same for every synapse.
Here, the network system of (1)-(3) is dimensionless, which is appropriate for mathematical and numerical exploration of the neurodynamics. However, neuroscientists are normally accustomed to the dimensional form with parameters that have physiological interpretation, e.g., (Dur-e-Ahmad et al., 2012) and (Rich et al., 2020). So we present in Appendix 7.1 the details of transformation between the dimensional and dimensionless models.

3 Mean-field reduction

The network model described in the previous section is too complicated to perform tractable analysis especially when the number of neurons is large. In this section, we will develop a low-dimensional mean-field model to approximate the behaviour of the full network described by (1)-(3) within the thermodynamic limit, i.e., when $N \to \infty$.

The mean-field approximation is essentially a technique that borrows concepts and methods from statistical physics, e.g., the population density approach (Ly & Tranchina, 2007), the continuity equation (or the Fokker-Planck equation when the system is subject to noise) (Deco et al., 2008; Bick et al., 2020). We will show how to describe some vital macroscopic variables such as the population firing rate and how to derive the reduced macroscopic dynamics, cast as ODEs, through step-by-step assumptions. Our approach comes from combination of the ideas of (Nicola & Campbell, 2013a; Ly & Tranchina, 2007; Montbrió et al., 2015).

3.1 General mean-field description

We define the population density function $\rho(t,v,w,\eta)$ as the density of neurons at a point $(v,w)$ in phase space and parameter $\eta$ at time $t$. In the limit $N \to \infty$, the principle of conservation mass leads to the following evolution equation for the density function, that is, the continuity equation,

$$\frac{\partial}{\partial t} \rho(t,v,w,\eta) + \nabla \cdot \mathcal{J}(t,v,w,s,\eta) = 0,$$  

where the probability flux is defined as

$$\mathcal{J}(t,v,w,s,\eta) = \left( J_s(t,v,w,s,\eta) \right) = \left( G^v(v,w,s,\eta) \right) \rho(t,v,w,\eta)$$  

and

$$G^v(\cdot) = v(v - a) - w + \eta + I_{\text{ext}} + g_{\text{syn}}(e_r - v)$$  

$$G^w(\cdot) = a(bv - w)$$  

Note that $J_s(t,v,w,s,\eta)$ is $s$ dependent. The flux is intuitively the mass flow rate along a specific direction in phase space. A boundary condition for the flux, consistent with the resetting rule in (1), is imposed,

$$J_s(v_{\text{peak}},w) = J_s(v_{\text{reset}},w + w_{\text{jump}}).$$  

We assume the flux to be vanishing on the boundary $\partial w$ (Ly & Tranchina, 2007), i.e., in the limit $w \to \pm \infty$.

Next, we describe several macroscopic observables in terms of mean-field description, which are extremely useful in understanding neural activities underlying brain function. The population firing rate is the flux through the threshold $v_{\text{peak}}$ over the entire range of $w$ in phase space and $\eta$ in parameter space, defining

$$r(t) = \lim_{v_{\text{peak}} \to \infty} \int_{\partial w} \mathcal{J}(t,v,w,s,\eta) dw d\eta$$

$$= \int_{\partial w} \int_{\partial w} \mathcal{J}(t,v_{\text{peak}},w,s,\eta) dw d\eta$$  

The mean membrane potential is defined as

$$\langle v(t) \rangle = \int_{\partial w} \int_{\partial w} v \rho(t,v,w,\eta) dv dw d\eta$$  

where $\langle \cdot \rangle$ represent the average over the population. Additionally, we define the mean adaptation current over the population as

$$\langle w(t) \rangle = \int_{\partial w} \int_{\partial w} w \rho(t,v,w,\eta) dv dw d\eta$$  

Then, we approximate its derivative with respect to $t$, yielding

$$\langle w(t) \rangle' = \int_{\partial w} \int_{\partial w} w \frac{\partial}{\partial t} \rho(t,v,w,\eta) dv dw d\eta$$

$$\approx \langle G^w(v,w) \rangle$$

$$+ \int_{\partial w} \int_{\partial w} \eta w_{\text{jump}} \mathcal{J}(t,v_{\text{peak}},w,s,\eta) dw d\eta$$

To obtain this expression, we assume the mean adaptation with the parameter $\eta$ is sufficiently greater than the after-spike jump size, i.e., $\langle w|\eta \rangle \gg w_{\text{jump}}$, and we also assume the flux to be vanishing on the boundary $\partial w$ (Nicola & Campbell, 2013a). See Appendix 7.2.1 for more details. Further, considering the linearity of $G^w(\cdot)$ function with respect to $v$ and $w$, see Eq. (6), and the description of the population firing rate in terms of flux (8), we finally...
derive the following ODE describing the evolution of the mean adaptation variable,
\[
\langle w \rangle' = G_w^w \langle \langle v \rangle, \langle w \rangle \rangle + w_{\text{jump}} r(t) = a(b(v) - \langle w \rangle) + w_{\text{jump}} r(t).
\] (12)

By considering the relationship between the flux and the description of the population firing rate in terms of number of spikes fired by neurons (Nicola & Campbell, 2013a), we can also rewrite the synaptic dynamics (3) in terms of the firing rate as
\[
s' = -\frac{s}{\tau_s} + s_{\text{jump}} \int d\eta \int dv \mathcal{J}(t, v_{\text{peak}}, w, s, \eta)dvd\eta = -\frac{s}{\tau_s} + s_{\text{jump}} r(t). \] (13)

The two Eqs. (12) and (13) are an integral part of the final mean-field model for the network of Izhikevich neurons. They depend on two macroscopic variables: the mean membrane potential \(\langle v(t) \rangle\) and the population firing rate \(r(t)\). In the following, we will derive the dynamical system for these two variables.

### 3.2 Density function in conditional form

In this section, we take advantage of the population density approach and the moment closure assumption to reduce the dependence between the macroscopic variables. We begin by writing out the population density function in the conditional form
\[
\rho(t, v, w, \eta) = \rho^v(t, w|v, \eta)\rho^w(t, v|\eta)\mathcal{L}(\eta), \] (14)
where \(\mathcal{L}(\eta)\) is defined as the probability that a randomly chosen neuron has an intrinsic parameter \(\eta\). Then, the population firing rate in the general expression (8) can be described by the conditional probability \(\rho^v(t, v|\eta)\) as
\[
r(t) = \lim_{v \to v_{\text{peak}}} \int d\eta \mathcal{L}(\eta)\rho^v(t, v|\eta) \cdot G^v(v, \langle w|v, \eta \rangle, s, \eta)d\eta. \] (15)

Appendix 7.2.2 gives the detailed derivation. Next, we assume
\[
\langle w|v, \eta \rangle = \langle w|\eta \rangle, \] (16)
which corresponds to a first order moment closure assumption (Nicola & Campbell, 2013b). Then, we have
\[
r(t) = \lim_{v \to v_{\text{peak}}} \int d\eta \mathcal{L}(\eta)\rho^v(t, v|\eta) \cdot G^v(v, \langle w|\eta \rangle, s, \eta)d\eta. \] (17)

Similarly, the mean membrane potential is rewritten as
\[
\langle v(t) \rangle = \int dv \mathcal{L}(\eta) \int dv v \rho^v(t, v|\eta)dvd\eta, \] (18)
where we use the normalization condition on the marginal density of \(w\). Appendix 7.2.3 gives the details. Furthermore, we integrate the general continuity Eq. (4) with respect to \(w\) and use the conditional form (14), yielding
\[
\frac{\partial}{\partial t} \rho^v(t, v|\eta) = -\frac{\partial}{\partial v} \left[ G^v(v, \langle w|v, \eta \rangle, s, \eta)\rho^v(t, v|\eta) \right]. \] (19)

To obtain this expression, we used the normalization condition on the marginal density of \(w\) and the fact that the flux vanishes on the boundary \(\partial w\). Finally, by the moment closure assumption (16), we have the resulting modified continuity equation,
\[
\frac{\partial}{\partial t} \rho^v(t, v|\eta) = -\frac{\partial}{\partial v} \left[ G^v(v, \langle w|\eta \rangle, s, \eta)\rho^v(t, v|\eta) \right]. \] (20)

This modified continuity equation together with Eq. (13), an equation analogous to (12) for \(\langle w|\eta \rangle\) and Eqs. (17-18) form a closed system for the evolution of \(\rho^v(t, v|\eta)\), \(\langle w|\eta \rangle\) and \(s\). Consideration of the steady state of the solution to this system yields
\[
\bar{\rho}^v(v|\eta) \propto \frac{1}{G^v(v, \langle w|\eta \rangle, \bar{s}, \eta)} \frac{1}{v(v - \alpha) - \langle w|\eta \rangle + \eta + I_{\text{ext}} + \gamma_{\text{syn}}(\epsilon_r - v)}, \] (21)
where \(\langle w|\eta \rangle\) and \(\bar{s}\) are the steady state values of \(\langle w|\eta \rangle\) and \(s\), respectively.

### 3.3 Lorentzian ansatz

In this section, we will further simplify the expressions of the macroscopic variables \(r(t)\) and \(\langle v(t) \rangle\), and derive the mean-field approximation for the Izhikevich network by employing the Lorentzian ansatz (Montbrió et al., 2015). To begin, we assume that the conditional probability \(\rho^v(t, v|\eta)\) satisfies a time dependent version of Eq. (21) and hence can be written in the form of Lorentzian distribution as follows,
\[
\rho^v(t, v|\eta) = \frac{1}{\pi} \frac{x(t, \eta)}{(v - y(t, \eta))^2 + x^2(t, \eta)}, \] (22)
where \(x(t, \eta)\) and \(y(t, \eta)\) are two time-dependent parameters defining half-width at half-maximum and location of the center, respectively. Moreover, \(y(t, \eta)\) is defined via the Cauchy principal value as
\[
y(t, \eta) = P.V. \int dv v \rho^v(t, v|\eta)dv = \langle v(t, \eta) \rangle, \] (23)
the reason being that the Lorentz distribution only has a mean in principal value sense. So the mean membrane potential is related to \( y(t, \eta) \) via

\[
\langle v(t) \rangle = \int_{\eta} y(t, \eta) \mathcal{L}(\eta) d\eta. \tag{24}
\]

Under the condition

\[
\nu_{\text{peak}} = -\nu_{\text{reset}} \to +\infty \tag{25}
\]

corresponding to \( \theta = \pi \) in the theta model, the population firing rate defined as Eq. (17) is also related to the Lorentzian coefficient through the intermediate expression,

\[
r(t, \eta) = \lim_{\nu_{\text{peak}} \to -\infty} \frac{\rho'(t, \nu|\eta)G'(v, \langle v|\eta, s, \eta)\rangle}{\nu_{\text{peak}} - y(t, \eta) + x^2(t, \eta)} \\
= \lim_{\nu_{\text{peak}} \to -\infty} \frac{1}{\nu_{\text{peak}} - y(t, \eta) + x^2(t, \eta)} \left[ \nu_{\text{peak}} - y(t, \eta) + x^2(t, \eta) \right] \\
+ I_{\text{ext}} + g_{\text{syn}} Y e_r - v_{\text{peak}} \right] \\
= \frac{1}{\pi} \chi(t, \eta). \tag{26}
\]

The total firing rate is then

\[
r(t) = \int_{\eta} \nu_{\text{peak}} r(t, \eta) \mathcal{L}(\eta) d\eta \\
= \int_{\eta} \frac{1}{\pi} \chi(t, \eta) \mathcal{L}(\eta) d\eta. \tag{27}
\]

For the continuity Eq. (20), we substitute the Lorentzian ansatz (22) and equate the resulting equation in the powers of \( v \), yielding

\[
x'(t, \eta) = 2xy - (\alpha + g_{\text{syn}})x, \\
y'(t, \eta) = y(y - \alpha) - x^2 - \langle w|\eta \rangle + \eta \\
+ I_{\text{ext}} + g_{\text{syn}} Y e_r - y, \tag{28}
\]

where the first equation is from the coefficient of \( v^2 \) equal to zero; the second from the coefficient of \( v \) equal to zero.

Both of them lead to disappearance of the leftover terms. See Appendix 7.2.4 for more details. By defining a complex variable

\[
z(t, \eta) = x(t, \eta) + iy(t, \eta) \\
= \pi r(t, \eta) + i\langle v(t, \eta) \rangle, \tag{29}
\]

we write Eq. (28) in complex form as

\[
\frac{\partial}{\partial t} z(t, \eta) = i \left[ -z^2(t, \eta) + iz(t, \eta)(\alpha + g_{\text{syn}}) \right. \\
- \langle w|\eta \rangle + \eta + I_{\text{ext}} + g_{\text{syn}} Y e_r \right], \tag{30}
\]

Till now, we have obtained the mean-field approximations for the dynamics of the two macroscopic variables \( r(t, \eta) \) and \( \langle v(t, \eta) \rangle \) via Eqs. (24), (27), (29) and (30). However, both of them depend on the heterogeneous current \( \eta \) and \( \langle w|\eta \rangle \) and hence on the distribution \( \mathcal{L}(\eta) \).

\subsection*{3.4 Heterogeneity with Lorentzian distribution}

Further derivation of the mean-field description in terms of the macroscopic observables \( r \) and \( \langle v \rangle \) depends on the distribution of the heterogeneous parameter, \( \eta \). Specifically, we choose \( \eta \) to have a Lorentzian distribution with center \( \bar{\eta} \) and half-width at half-maximum \( \Delta_\eta \), i.e.,

\[
\mathcal{L}(\eta) = \frac{\Delta_\eta}{\pi (\eta - \bar{\eta})^2 + \Delta_\eta^2}. \tag{31}
\]

Then, we apply the residue theorem to compute the integrals in (24) and (27) for \( \eta \in (-\infty, \infty) \) and obtain

\[
r(t) = \frac{1}{\pi} \chi(t, \bar{\eta} - i\Delta_\eta), \\
\langle v(t) \rangle = \chi(t, \bar{\eta} - i\Delta_\eta). \tag{32}
\]

One can see the derivation in Appendix 7.2.5. Further, considering \( \pi r(t) + i\langle v(t) \rangle = z(t, \bar{\eta} - i\Delta_\eta) \), evaluating the complex Eq. (30) at \( \eta = \bar{\eta} - i\Delta_\eta \) and taking into account the formula that

\[
\langle w \rangle = \int_{\eta} \langle w|\eta \rangle \mathcal{L}(\eta) d\eta \tag{33}
\]

yields the mean-field system of equations given by

\[
r' = \Delta_\eta/\pi + 2r(v) - (\alpha + g_{\text{syn}})r, \\
\langle v \rangle' = \langle v \rangle^2 - \alpha \langle v \rangle - \langle w \rangle + \bar{\eta} + I_{\text{ext}} \\
+ g_{\text{syn}} Y e_r - (\langle v \rangle - \pi^2 r^2). \tag{34}
\]

Note that the distribution \( \mathcal{L}(\eta) \) can be arbitrary. Particularly, if \( \mathcal{L}(\eta) \) has \( n \) poles in the lower half \( \eta \)-plane, one can readily obtain \( n \) sets of complex-valued mean-field ODEs analogous to Eq. (30) by evaluating the integrals (24) and (27) (Ott & Antonsen, 2008). Lorentzian distribution is a mere mathematical convenience since it has only one pole as required.

Recalling that we have already obtained the dynamical system for the mean adaptation current (12) and the synapse (13), we finally have the reduction of the network of Izhikevich neurons (1)-(3) to the following the mean-field system of ODEs,
\[ r' = \frac{\Delta \eta}{\pi} + 2r(v) - (\alpha + g_{syn})s \tau \]
\[ \langle v \rangle' = \langle v \rangle^2 - \alpha \langle v \rangle - \langle w \rangle + \bar{\eta} + I_{ext} \]
\[ + g_{syn}(r_{v} - \langle v \rangle)^{2} - \pi^{2}r^{2} \]
\[ \langle w \rangle' = a(b \langle v \rangle - \langle w \rangle) + w_{\text{jump}}r \]
\[ s' = -s/\tau_{s} + s_{\text{jump}}r. \]

### 4 Numerical analysis

We now numerically examine the dynamics of the mean-field model and demonstrate its validity in terms of reproducing the macroscopic dynamics of the network of Izhikevich neurons.

We consider an all-to-all coupled network with synapses governed by the single exponential model. The parameter values used in all simulations can be found in Table 1, unless otherwise specified in a figure. The corresponding dimensional form of the network model and parameter values are given in Appendix 7.1. Most of these values are taken from (Nicola & Campbell, 2013a, b) which were originally fit by Dur-e-Ahmad et al. (2012) to hippocampal CA3 pyramidal neuron data from (Hemond et al., 2008). The exceptions are \( v_{\text{peak}} \) and \( v_{\text{reset}} \), which are, respectively, set to large positive and negative numbers. This is to approximate \( v_{\text{peak}} \to +\infty \) and \( v_{\text{reset}} \to -\infty \), which is required for the QIF model to be well-approximated by the theta model (Izhikevich, 2007; Montbrió et al., 2015). Numerical simulation was done by using the Euler’s method in MATLAB, with time step \( dt = 10^{-3} \) and numerical continuation by using the software XPPAUT (Ermentrout, 2002). All initial conditions are chosen at the origin.

We begin with the bifurcation analysis of the mean-field model (34). Figure 1(a) and its blow-up (b) show how the population firing rate \( r \) qualitatively changes as the mean intrinsic current \( \bar{\eta} \) is varied. Figure 1(c) shows the same diagram as (a) in terms of the mean adaptation, \( \langle w(t) \rangle \), and includes a bifurcation diagram for full network (1-3) (star symbols). The mean-field model diagrams show two subcritical Andronov-Hopf bifurcations (HPs) at \( \bar{\eta} = \bar{\eta}_{HP} \approx 0.19 \) and 0.075, respectively. Unstable limit cycles emerge from these bifurcations and collide with the stable limit cycles in a saddle-node bifurcation of limit cycles (SNLC) for some \( \bar{\eta} = \bar{\eta}_{\text{SNLC}} > \bar{\eta}_{HP} \) (right branches) or \( < \bar{\eta}_{HP} \) (left branches). The system displays two small ranges of bistability between the Hopf and SNLC bifurcations. The stable periodic orbits (POs) (green dots) correspond to solutions where the individual neurons in the network exhibit synchronous bursting and stable equilibrium points (EPs) (red lines) correspond to solutions where individual neurons exhibit asynchronous tonic firing. This is clearly reflected in the time series of macroscopic variables \( r(t) \), \( \langle v(t) \rangle \) and \( \langle w(t) \rangle \) in Fig. 1(d) and (e). The mean-field Eq. (34) very accurately predict the behaviour of the full network, including the damped oscillations in (d) and the frequency of stable oscillations in (e).

Prior work has shown that bursting in a network of Izhikevich neurons is due to a balance between the inputs (intrinsic and external applied currents and synaptic inputs), which cause the neurons to spike, and the slow adaptation current, which can terminate spiking (Dur-e-Ahmad et al., 2012; Nicola & Campbell, 2013a, b). For a given level of adaptation there must be a sufficient input, but not too much, to initiate bursting. Hence the bursting in Fig. 1(a) occurs when the mean intrinsic current \( \bar{\eta} \) is not too small and not too large. Note in Fig. 1(e) that even when the population is bursting, a small subset of neurons in the population do not burst but remain tonically firing. This is due to the distribution of the heterogeneous input current. A small number of neurons will receive a large enough input current that the adaptation is not strong enough to cause these neurons to burst.

In addition, Fig. 1(c) demonstrates a great correspondence between a bifurcation diagram generated from numerical simulation of the full network (star symbols) with that obtained from the mean-field model. Here, it is worth paying more attention to the POs (green curves). This figure shows an excellent amplitude match for a wide range of the bifurcation parameter. Note in Fig. 1(c) that we compare behaviours of the variable \( \langle w \rangle \) instead of \( r \) for clarity, as \( r \) exhibits fluctuations due to the spiking of individual neurons (see Fig. 1(e)). Employing a larger number of neurons in the network model achieves a narrower spread around the value of the mean-field model, at the expense of increased computational time.

Table 1: Dimensionless parameters for the network of Izhikevich neurons

| Parameter | Value | Parameter | Value |
|-----------|-------|-----------|-------|
| \( \alpha \) | 0.6215 | \( \tau_{s} \) | 2.6 |
| \( g_{syn} \) | 1.2308 | \( e_{r} \) | 1 |
| \( a \) | 0.0077 | \( b \) | -0.0062 |
| \( s_{\text{jump}} \) | 1.2308 | \( w_{\text{jump}} \) | 0.0189 |
| \( v_{\text{peak}} \) | 200 | \( v_{\text{reset}} \) | -200 |

These parameters apply unless otherwise indicated.
chosen for the network model and the mean-field model, but the network exhibits the PO while the mean-field model exhibits the EP. Thus, it would appear that the bifurcation point leading to the PO in the mean-field model is different than that in the network model. In Fig. 2(c) the parameter \( \bar{\eta} \) is slightly less than the second bifurcation point, in the region where the mean-field model has only a stable PO. In this case, both the mean-field and network models exhibit a PO, but there is a frequency mismatch. Better agreement in examples can be achieved by slightly tuning the bifurcation parameter of the mean-field model towards higher values. See Fig. 2(b), (d). The same trend can be seen in \( \bar{\eta} \) bifurcation diagram between the mean-field model (34) and the full network. d & e: Comparison of the temporal behaviour of the network and the corresponding mean-field model when \( \bar{\eta} = 0.25 \) (s1 in (a) and (c)) and \( \bar{\eta} = 0.12 \) (s2 in (a) and (e)). First row of panels are the raster plots of 300 randomly selected neurons from the \( N = 10^4 \) in the population. Other rows of panels show, respectively, the population firing rate \( r(t) \), mean membrane potential \( \langle v(t) \rangle \) and mean adaptation variable \( \langle w(t) \rangle \) obtained from simulations of the full network (blue) and the mean-field model (red). Parameters: \( \Delta_\eta = 0.02 \), \( I_{ext} = 0 \), others are given in Table 1.

Fig. 1 One-parameter bifurcation diagram and time evolution. a: \( r - \bar{\eta} \) bifurcation diagram of the mean-field model (34). The red (black) lines correspond to stable (unstable) EPs and green (blue) dots correspond to stable (unstable) POs. b: Blow-up of (a) near \( \bar{\eta} = 0.19 \). Bistability is induced by a subcritical Hopf bifurcation (HB) and saddle-node bifurcations of limit cycles (SNLC). Similar qualitative changes occur at \( \bar{\eta} \approx 0.075 \). c: Comparison of the \( \langle w \rangle - \bar{\eta} \) bifurcation diagram between the mean-field model (34) and the full network (1-3). Star symbols: results obtained from numerical simulations of the full network. d & e: Comparison of the temporal behaviour of the network and the corresponding mean-field model when \( \bar{\eta} = 0.25 \) (s1 in (a) and (c)) and \( \bar{\eta} = 0.12 \) (s2 in (a) and (e)). First row of panels are the raster plots of 300 randomly selected neurons from the \( N = 10^4 \) in the population. Other rows of panels show, respectively, the population firing rate \( r(t) \), mean membrane potential \( \langle v(t) \rangle \) and mean adaptation variable \( \langle w(t) \rangle \) obtained from simulations of the full network (blue) and the mean-field model (red). Parameters: \( \Delta_\eta = 0.02 \), \( I_{ext} = 0 \), others are given in Table 1.
Furthermore, we can determine the Andronov-Hopf bifurcation manifolds in the $(\bar{\eta}, \Delta \eta)$ parameter space for the mean-field model as shown in Fig. 3(a) and (b). These curves are associated with the transition between synchronous bursting (inside the curves) and asynchronous tonic firing (outside the curves). Thus we will call the region inside the Andronov-Hopf bifurcation manifold the bursting region. We see from the network raster plots and time series of $r(t)$ (Fig. 3(c)) that the rhythmic regime disappears if the external drive $I_{\text{ext}}$ is sufficiently strong. This is a well-known effect observed in many models of adaptation induced bursting (Dur-e-Ahmad et al., 2012; Nicola & Campbell, 2013a, b). It is also consistent with the experimental data of Tateno et al. (1998) on a hippocampal CA3 slice preparation, who showed that injection of sufficient depolarizing current into pyramidal cells can cause them to transition from bursting to tonic firing. Additionally, Fig. 3(d) shows a Hopf-Hopf bifurcation resulting from intersection of two Andronov-Hopf bifurcations in the $(\bar{\eta}, w_{\text{jump}})$ parameter space. The two curves look like straight lines in such narrow scales. Secondary bifurcations can emanate from this co-dimension two bifurcation point, leading to, for example, quasiperiodic behaviour (Guckenheimer & Holmes, 1983). We leave further investigation of this bifurcation for future work.

5 Extension to two-coupled populations

A large-scale neural network can also be regarded as several coupled groups by considering different properties of cells in the network. For example, neurons can be grouped into excitatory and inhibitory populations based on the type of synapses they form, e.g., (Wilson & Cowan, 1972; Dumont & Gutkin, 2019); or into strongly and weakly adapting populations based on the amount of spike frequency adaptation they exhibit, e.g., (Nicola & Campbell, 2013a). In this section we consider a network of excitatory neurons consisting of two populations: strongly adapting neurons (population $p$) and weakly adapting neurons (population $q$). The network is all-to-all connected with single exponential synapses. This model is motivated by the experimental data of (Hemond et al., 2008) on firing properties of CA3 pyramidal neurons and the modelling studies of (Dur-e-Ahmad et al., 2012; Nicola & Campbell, 2013a, b). Each neuron in the network is characterized by the Izhikevich model given by:

\[
\begin{align*}
    v'_{m,k} &= v_{m,k}(v_{m,k} - a_{m}) - w_{m,k} + \eta_{m,k} + f_{m}^{\text{ext}} + f_{m}^{\text{syn}}, \\
    w'_{m,k} &= a_{m}(b_{m}v_{m,k} - w_{m,k}),
\end{align*}
\]

if $v_{m,k} \geq v_{\text{peak}}$, then $v_{m,k} \leftarrow v_{\text{reset}}^{m}$

and $w_{m,k} \leftarrow w_{m,k} + w_{\text{jump}}^{m}$,

where $m = p, q$ represents the two populations with $N_p$ and $N_q$ cells, respectively. The subscript $\{m,k\}$ denotes the $k$th neuron in population $m$. The subscript with only $\{m\}$ represents the corresponding parameter is homogeneous within the population $m$, but heterogeneous across the two populations. The total synaptic current $f_{m}^{\text{syn}}$ depends on the cell type. We require two maximal synaptic conductances,
where $\kappa = \frac{N_p}{N_p + N_q}$ is the proportion of strongly adapting neurons in the network and $s_p$ (respectively, $s_q$) represents the proportion of open synapses due to neurons in the strongly (respectively, weakly) adapting population. These gating variables are governed by the single exponential synapse model,

$$s_m' = \frac{S_m}{\tau_s} + \frac{s_m}{N_m} \sum_{i=1}^{N_p} \sum_{t_m < t} \delta(t - t_{m_i}).$$

(37)
where \( m = p, q \). Thus, we can apply the Lorentzian ansatz and the method in the previous sections by considering the two populations to be described by their own distinct density functions,

\[
\rho_m(t, v_m, w_m, \eta_m) = \frac{\Delta_m}{\pi (\eta_m - \bar{\eta}_m)^2 + (\Delta_m)^2}, \quad m = p, q.
\]

Finally, assuming the Lorentzian distribution of the heterogeneous currents for both populations,

\[
\mathcal{L}(\eta_m) = \frac{1}{\pi (\eta_m - \bar{\eta}_m)^2 + (\Delta_m)^2}, \quad m = p, q, \tag{38}
\]

we obtain the mean-field system consisting of a set of eight differential equations. Among them, three differential equations describe the mean-field quantities for each population,

\[
\begin{align*}
  \dot{r}_p' &= \Delta_p / \pi + 2r_p \langle v_p \rangle - r_p [G_{p}\alpha_p + \gamma_p] \\
  \langle v_p \rangle &= \langle v_p \rangle - \alpha_p \langle v_p \rangle - \langle w_p \rangle + \bar{\eta}_p \\
  \langle w_p \rangle &= \alpha_p [D_p \langle v_p \rangle - \langle w_p \rangle] + w_p^{jump} r_p
\end{align*}
\]

for the population \( p \) with strong adaptation and

\[
\begin{align*}
  \dot{r}_q' &= \Delta_q / \pi + 2r_q \langle v_q \rangle - r_q [G_{q}\alpha_q + \gamma_q] \\
  \langle v_q \rangle &= \langle v_q \rangle - \alpha_q \langle v_q \rangle - \langle w_q \rangle + \bar{\eta}_q \\
  \langle w_q \rangle &= \alpha_q [D_q \langle v_q \rangle - \langle w_q \rangle] + w_q^{jump} r_q
\end{align*}
\]

for the population \( q \) with weak adaptation. These two subsystems are coupled through synaptic currents as given in Eq. (37), with the synaptic dynamics governed by

\[
\begin{align*}
  s_p' &= -s_p / r_p + s_p^{jump} r_p \\
  s_q' &= -s_q / r_q + s_q^{jump} r_q.
\end{align*}
\]

A detailed derivation for the two-population mean field model can be found in Appendix 7.3.

In the following we analyze the dynamics of the mean-field system and examine how well it reproduces the macroscopic activities of the two-population network of Izhikevich neurons. The parameter values can be found in Table 2. Most of these values are taken from (Nicola & Campbell, 2013a, b), which were originally fit by Dur-e-Ahmad et al. (2012) to hippocampal CA3 pyramidal neuron data from (Hemond et al., 2008). The only parameters that differ between the two populations are those that govern the adaptation levels, i.e., the after-spike jump sizes \( w_m^{jump} \) and time constants \( \alpha_m \), where \( m = p, q \).

Figure 4 compares the behaviour of the mean-field model to simulations of the full network. Figure 4(c)-(h) show that the dynamics of the two-population network are very well described by the reduced mean-field description in the asynchronous tonic firing (EPs in Fig. 4(c), (e) and (g)) and synchronous bursting (POs in Fig. 4(d), (f) and (h)) regimes. Note in the bursting regime that a relatively larger fraction of neurons in the weakly adapting population is tonically firing (see (f)). This makes sense since the bursting is due to the balance of inputs and adaptation. In the weakly adapting population, a larger fraction of neurons receive sufficient input to prevent them from bursting. In addition to the strong correspondence found at these two typical parameter settings, Fig. 4(a) and (b) shows there is a great correspondence between a bifurcation diagram from numerical simulation of the two-population network (star symbols) with that obtained from the mean-field model in terms of \( \langle v \rangle_p \) and \( \langle w \rangle_q \), respectively. In spite of the excellent amplitude agreement at each parameter point shown, we should point out that the mean-field dynamics shows some quantitative differences from those of the network model, especially at the points very close to the transition between bursting and firing. The effect is very similar to what is seen for the single population network in Fig. 2. Improvement can be seen by slightly adjusting the bifurcation parameter of the mean-field model towards higher values, although it is not as effective as that shown in the single-population network.

Additionally, the mean-field model for the network of two coupled Izhikevich populations involves more complicated bifurcations compared with that of the single-population network of strongly adapting Izhikevich neurons studied in the previous section. Bifurcation analysis in Fig. 5 reveals that when the proportion of strong adapting neurons is \( \kappa = 0.8 \), the sequence of bifurcation is largely the same as that when there is a single population of strongly adapting neurons (compare Fig. 5(a) with Fig. 1(a)). With this value of \( \kappa \), as the mean intrinsic current is increased, stable periodic behaviour in the mean-field system (39)-(41) is initiated by a saddle-node bifurcation of limit cycles (SNLC) connected to a subcritical Andronov-Hopf bifurcation (HP) at \( \tilde{\eta}_p = \tilde{\eta}_q \approx 0.054 \) and terminated by the same sequence in

### Table 2: Dimensionless parameters for the two coupled Izhikevich network

| Parameter | Value | Parameter | Value |
|-----------|-------|-----------|-------|
| \( w_p^{jump} \) | 0.0189 | \( w_q^{jump} \) | 0.0095 |
| \( \gamma_p \) | 0.0077 | \( \gamma_q \) | 0.077 |
| \( g_{p}^{syn} \) | 1.2308 | \( g_{q}^{syn} \) | 1.2308 |
| \( g_{p}^{exi} \) | 1.2308 | \( g_{q}^{exi} \) | 1.2308 |
| \( I^{ext}_p \) | 0 | \( I^{ext}_q \) | 0 |

All parameters that are not shown here are the same for both populations and can be found in Table 1. These parameters apply unless otherwise indicated.

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Note: The table layout is not properly rendered in this text. It is a standard LaTeX table format used for tables in academic papers and may require LaTeX or similar software to properly display.
Further, one can see from Fig. 5(c) that the stable period doubling is the case when $\tilde{\eta}_p = \tilde{\eta}_q = 0.08$ (see Appendix 7.2.1). Shown in green (population $p$) and blue (population $q$) are the population firing rates and the mean adaptation variables ($q$ & $h$) obtained from the full network. Shown in red are the corresponding variables of the mean-field models. It is also shown the raster plots of 300 randomly selected neurons from each population. Parameters: $\Delta^q = \Delta^q = 0.02$, others are given in Table 2.

reverse at the Hopf point $\tilde{\eta}_p = \tilde{\eta}_q \approx 0.135$. The system displays bistability in the narrow regions between the SNLC and HP. However, complex bifurcations occur when changing the proportion of strongly adapting neurons. Figure 5(b) shows bifurcation curves plotted in the $(\tilde{\eta}, \kappa)$ parameter plane. Saddle-node bifurcation curves (red) meet and form cusp points or tangentially intersect the curves of Andronov-Hopf bifurcation (blue) and produce a zero-Hopf bifurcation. When $\kappa = 0.8$, the system has two Hopf points, as shown in Fig. 5(a). When the proportion is reduced to $\kappa = 0.5$, the system undergoes two saddle-node bifurcations and one Andronov-Hopf bifurcation. These behaviours are also shown in the one-parameter bifurcation diagram Fig. 5(c). Further, one can see from Fig. 5(c) that the stable period behaviour is now initiated by what appears to be a saddle-node on an invariant circle bifurcation or a homoclinic bifurcation (at $\tilde{\eta}_p = \tilde{\eta}_q \approx 0.036$) and terminated by a supercritical Andronov-Hopf bifurcation at $\tilde{\eta}_p = \tilde{\eta}_q \approx 0.06$. Thus the stable periodic will have a very low frequency, which will rapidly increase as the $\tilde{\eta}$ increases. Finally, Fig. 5(d) shows a two-parameter bifurcation diagram in the $\tilde{\eta}, \Delta^\alpha$ parameter plane with $\kappa$ fixed at 0.5. Complicated bifurcation structures, including Bogdanov-Takens bifurcation, may occur for nearby parameter values. We leave further investigation of this complex bifurcation structure to future work.

Since periodic solutions in the mean-field model correspond to synchronous bursting in the full network, we can use these results to predict the effects of parameters on network behaviour. Comparing the dashed lines in Fig. 5(b) (which correspond to Fig. 5(a), (c)) shows that decreasing the proportion of strongly-adapting neurons, $\kappa$, makes bursting less likely in the sense that the range of values of the mean current $\tilde{\eta}$ for which bursting occurs is decreased. Figure 5(d) shows that decreasing the heterogeneity of the currents, $\Delta^\alpha$, has a similar effect. Further, a new transition to bursting is possible. In the two-parameter diagrams Fig. 5(b) and (d), the bursting region is now bounded in part by the Andronov-Hopf manifold (blue curve) and in part by the right-most saddle-node bifurcation (red curve). The frequency of the stable bursting behaviour near the saddle-node bifurcation, is expected to be strongly dependent on the mean intrinsic current, $\tilde{\eta}$, with quite low frequency possible near the transition point. The new transition occurs when the proportion of strongly-adapting neurons, $\kappa$, and the heterogeneity of the currents, $\Delta^\alpha$, are both small enough.

Figure 6 further illustrates the impact of the proportion of strongly adapting neurons in the network. The boundary of the bursting region is shifted into the high mean intrinsic current region for a higher proportion (shown in Fig. 6(a)) and the rhythmic regime appears as $\kappa$ is increased (shown in Fig. 6(b)). This indicates that stable bursting behavior is more likely in a network with a higher proportion of strongly adapting neurons. Figure 6(c) and (d) shows a comparison between the full network dynamics and the reduced mean-field system. The reduced description captures the essential shape of the firing activity of the full network. A little discrepancy happens to the approximation of the mean adaption variable $\langle w \rangle$. This may be due to the failure of the assumption $\langle w|\eta \rangle \gg w_{jump}$ during the derivation of dynamics of $\langle w \rangle$ (see Appendix 7.2.1).

### 6 Discussion

We have derived and validated a mean-field model for a network of heterogeneous Izhikevich neurons which display spike frequency adaptation through a recovery variable. We have further demonstrated that it is straightforward to apply our approach to multiple populations where the forces of adaptation, inhibition, or excitation interact. The mean-field models have exhibited qualitative and quantitative agreement with the full network. Using bifurcation analysis, we have identified and characterized regimes of collective bursting that emerge given appropriate levels of adaptation, external stimulus and proportion of strongly adapting neurons. The parameter values used in the numerical examples are a nondimensionalization of those fit to actual neuronal data collected in the literature. Bifurcation analysis for the mean-field system can be used to make predictions about the biological networks being studied, e.g. the emergence of synchronous bursting in the CA3 region of the hippocampus in this paper. Our model, which is an extension of those of (Dur-e-Ahmad et al., 2012) and (Nicola & Campbell, 2013a, b), is motivated by several observations. In their studies of isolated pyramidal cells in the CA3 region of hippocampus, Hemond et al. (2008) observed that there were few intrinsically bursting neurons (about 13%) with the rest exhibiting regular spiking with either strong spike frequency adaptation (37%) or weak spike frequency adaptation (46%). While it is...
well-known that spike frequency adaptation of individual neurons can give rise to bursting when the neurons are coupled (Ermentrout et al., 2001; Vreeswijk & Hansel, 2001), it is less clear that this will be the case when a significant proportion of the neurons are weakly adapting. The mean-field model (39)-(41) we derived allows us to compute bifurcation manifolds and types for the network with different proportions of strongly and weakly adapting neurons and determine the impact of various parameters on the transition between the behavior of asynchronous tonic firing and synchronous bursting in the actual neural network. In particular we found that the larger proportion of weakly-adapting neurons (small $\kappa$) makes bursting less likely and the decreasing heterogeneity of the currents (small $\Delta^\eta$) has a similar effect. While the former conclusion is not surprising, the latter is more interesting. Given the percentage of weakly vs strongly adapting neurons in the data of (Hemond et al., 2008), our results indicate that adaptation induced network bursting would only be possible if there is sufficient, but not too much, heterogeneity in the intrinsic applied current. Since the intrinsic current determines the firing rates of the model neurons, this shows that heterogeneity of firing rates may be an important factor in the generation of bursting behaviour. We also note that increasing the proportion of weakly-adapting neurons changed the bifurcation involved in the transition to bursting behaviour. With sufficient weakly-adapting neurons (50%) this transition was a homoclinic bifurcation or saddle-node on an invariant circle. For the network this translates to the potential increased variability.

Fig. 5 Bifurcation diagram of the mean-field model (39)-(41). a: The population firing rate $r_p$ with respect to the mean intrinsic current $\bar{\eta}_p = \bar{\eta}_q = \bar{\eta}$ when the proportion of strong adapting neurons $\kappa = 0.8$. b: Bifurcation curves in the ($\bar{\eta}$, $\kappa$)-parameter plane. Shown in red are the saddle-node bifurcations. Shown in blue are the Hopf bifurcations. c: $r_p$ with respect to $\bar{\eta}_p = \bar{\eta}_q = \bar{\eta}$ when $\kappa = 0.5$. A supercritical Hopf bifurcation occurs at $\bar{\eta} \approx 0.06$ and two saddle node bifurcations at $\bar{\eta} \approx 0.028$, 0.036, respectively. d: Bifurcation curves plotted in the ($\bar{\eta}$, $\Delta \eta$)-parameter plane when $\kappa = 0.5$. Shown in red are the saddle-node bifurcations. Shown in blue is the Hopf bifurcation. Parameters: $\Delta^\eta_p = \Delta^\eta_q = 0.02$, $N_p + N_q = 10^4$, others are shown in Table 2.
of the network bursting frequency including the possibility of quite low bursting frequency.

To assess the validity of our mean-field approximation, we examine all the assumptions that are imposed during the derivation. They are listed in order of appearance as follows.

1. All-to-all connectivity within the population and between different populations.
   This assumption is reasonable for the application to CA3 region of hippocampus we considered, since this region is highly recurrently coupled (Amaral & Witter, 1989; Andersen et al., 2006; Buzsaki, 2011). The approach developed in the paper can be generalized to the sparse network by using the formalism in (Ferguson et al., 2015; Di Volo & Torcini, 2018; Bi et al., 2021; Lin et al., 2020).

2. $N \rightarrow \infty$, the thermodynamic limit.
   In theory, the mean-field model is an exact description for the network of neurons in the thermodynamic limit. In the finite-size numerical experiments, the spread of

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**Fig. 6** Bifurcation diagram and time evolution comparison. **a**: Hopf bifurcation manifolds for the mean-field model (39)-(41) with different values of proportion of strongly adapting neurons $\kappa$. **b**: $r_p - \kappa$ bifurcation diagram. The red (black) lines correspond to the stable (unstable) EPs, and green dots correspond to stable limit cycles. **c & d**: Time evolution comparison between the network of two populations (35)-(37) and the corresponding mean-field model (39)-(41) when $\kappa = 0.6$ and 0.8, respectively. Shown in green (population $p$) and blue (population $q$) are the population firing rates and mean adaptation variables obtained from the full network. Shown in red are the corresponding variables in the mean-field models. The parameter values are shown in Table 2, including $N_p + N_q = 10^4$, $\bar{\eta}_p = \bar{\eta}_q = 0.08$ and $\Delta^{(p)} = \Delta^{(q)} = 0.04$ corresponding to $s_3$ in (a).
the network variables around the mean narrows as the number of neurons increases, and thus gets closer to the dynamics of the mean-field model. One can see Fig. 4 for a comparison, where $N_p = 8000$ for the strongly adapting neurons and $N_q = 2000$ for the weakly adapting neurons. The same trend can be seen in (Ciszak et al., 2021, Fig. 6).

3. $\langle w|\eta \rangle \gg w_{\text{jump}}$ the mean adaptation variable with the parameter $\eta$ is sufficiently greater than the homogeneous after-spike jump value.

This assumption is required when deriving the differential equation of $\langle w \rangle$. The small discrepancy between the mean-field model and the population of strongly adapting neurons (see time series of $\langle w \rangle$ of population $p$ in Figs. 4(g), (h), and 6(c) (d)) may result from a partial failure of the requirement. However, the mean-field description still captures the essential shape and frequency of the firing activity of the network. The accuracy could be improved by inclusion of high-order terms in the Taylor expansion (see Eq. (45) in Appendix 7.2.1). This will give rise to an extra term in the final mean-field equation for $\langle w \rangle$.

4. $\langle w|v, \eta \rangle = \langle w|\eta \rangle$, first-order moment closure assumption, also called the adiabatic approximation.

This assumption entails fast dynamics of the membrane potential. We could employ a high-order moment closure approximation, although we need to assess the cost of the added effort in terms of the improvement of the accuracy of the resulting mean-field equation (Ly & Tranchina, 2007).

5. $\rho^\eta(t, v|\eta) = \frac{1}{\pi} \frac{x(v, \eta)}{[v^2 - (v(\eta) - x)^2]^{1/2}}$, the Lorentzian ansatz on the conditional density function.

Derivation of the differential equations of the mean-field variables $r(t)$ and $\langle v(t) \rangle$ started with writing out the population density function in the conditional form $\rho(t, v, w, \eta) = \rho^\eta(t, v|\eta)\rho^w(t, w|\eta)\mathcal{L}(\eta)$ as shown in Eq. (14). When we rewrite it as $\rho(t, v, w, \eta) = \rho^\eta(t, \eta|v, w)\rho^w(t, v, w)$ and assume $\rho^w(t, v, w)$ has the Lorentzian shape, we can use the method developed in the paper to obtain a different mean-field system. Nicola and Campbell (2013b) shows how changing the expansion of the population density function can drastically change the resulting mean-field model. The expansion we used here corresponds to that used to develop “Mean-field system III” in (Nicola & Campbell, 2013b).

6. $v_{\text{peak}} = -v_{\text{reset}} \to \infty$, limit of the resetting rule when neurons fire.

Parameter values used in the paper are based on actual neuronal data except the resetting values. This choice is essential for the validity of the Lorentzian ansatz Eq. (22) (Montbrió et al., 2015), the derivation of Eq. (26) and facilitates analysis by linking the QIF model to the theta neuron model through the change of variable $v = \tan(\theta/2)$ (Izhikevich, 2007). Although not precisely biologically realistic, the theta model and its variants have been used in the literature to explore phenomena, such as chaotic dynamics in large, sparse-balanced networks (Monteforte & Wolf, 2010), rhythm generation (Kilpatrick & Ermentrout, 2011), wave propagation in the cortex (Ozan & Ermentrout, 2001; Ermentrout et al., 2002; Byrne et al., 2020) and models for EEG (Byrne et al., 2022). For the neuron model that exhibits a saddle-node on an invariant circle (SNIC) bifurcation, it is possible to reduce it to the theta model with adaptation, e.g. (Ermentrout & Kopell, 1986; Ermentrout, 1996; Kilpatrick & Ermentrout, 2011). For the system not near a SNIC, but near some other bifurcation satisfying fairly general and biophysically plausible conditions, one can still obtain the theta model with adaptation (Izhikevich, 2000). When dealing with a biological network based on experimental data, we should treat the assumption carefully, as changing $v_{\text{peak}}$ and $v_{\text{reset}}$ can affect firing rates and estimation of the mean membrane potential. In the numerical experiments, we choose $v_{\text{peak}} = -v_{\text{reset}} = 200$ as that in (Dumont et al., 2017), which is far away from the normal range of the membrane potential $v$. We have found that different spiking thresholds lead to some bias in averaging $v$ over the full network, even if they meet the assumption requirement. Montbrió et al. (2015) attempted to address this drawback by adding a refractory period to the network model, that is, the time for neurons taken from $v_{\text{peak}}$ to infinity and minus infinity to $v_{\text{reset}}$. This effectively makes the firing rate of the asynchronous tonic firing (EPs) and the mean membrane potential of the network match those of the theta model and hence the mean-field model. However, values of $v_{\text{peak}}$ and $v_{\text{reset}}$ were still far away from the normal range of $v$. Moreover, because lack of adaptation, the model in (Montbrió et al., 2015) cannot exhibit synchronous bursting (POs). In addition, to avoid numerical delicacies near the spiking threshold, Pyragas and Pyragas (2021) transformed the QIF form to the form of the theta neuron when performing the numerical simulation.

7. $\mathcal{L}(\eta) = \frac{1}{\pi} \frac{\Delta_x}{(\eta - \tilde{\eta})^2 + \Delta^2}$, distribution of the heterogeneous current.

Many parameters can be the sources of heterogeneity in a network. Here we chose the intrinsic current $\eta$, but our approach could be applied to other choices, such as the synaptic conductance $g_{\text{syn}}$. In addition, the choice of the Lorentzian function is just a mathematical convenience. It can sharply reduce the complexity of the resulting mean-field model when evaluating the integrals in (24) and (27). Unfortunately, the Lorentzian distribution is physically implausible since both its
expected value and its variance are undefined. Other distributions including Gaussian have been discussed in the literature. Particularly, if a distribution \( \mathcal{L}(\eta) \) has \( n \) complex-conjugate pole pairs, one can readily obtain the mean-field model consisting of \( n \) complex-value ODEs in the form (30) (Ott & Antonsen, 2008). Further, Ott and Antonsen (2009) and Montbrió et al. (2015) both pointed out that mean-field systems obtained using the Lorentzian and Gaussian distributions had qualitatively identical bifurcation structures. However, Klinshov et al. (2021) claimed that the quantitative difference did matter in terms of both transient and asymptotic dynamics. They also showed how to compute the integrals in (24) and (27) with the help of the rational approximation and residue theory when \( \mathcal{L}(\eta) \) is a Gaussian distribution. Certainly, the resulting mean-field model has higher dimensions.

For the numerical experiments, Montbrió et al. (2015) generated a set of \( N \) input currents that accurately reproduced a Lorentzian distribution and used the same set of input currents for all simulations with \( N \) neurons, given by

\[
\eta_k = \bar{\eta} + \Delta_\eta \tan\left(\frac{\pi}{2} \frac{2k - N - 1}{N + 1}\right). \tag{42}
\]

In this paper, we take a different approach, except Fig. 2. We generate the distribution by using the technique of inverse transform sampling. Specifically, for the \( k \)th neuron, we have

\[
\eta_k = \bar{\eta} + \Delta_\eta \tan\left(\pi (r_k - 0.5)\right). \tag{43}
\]

where value of the cumulative distribution function \( r_k \) is randomly sampled from the uniform distribution on \((0, 1)\). This is a basic method for pseudo-random number sampling from any probability distribution. The advantage is that the distribution of the heterogeneous currents is more realistic. The drawback is the number of neurons \( N \) involved in the simulation must be large enough to exhibit a good approximation of the Lorentzian distribution. For example, \( N = 10^4 \) in our simulations. Additionally, numerical results obtained in every simulation are a little different since the current distributed to each neuron is different each time. Specifically, for our mean-field approximations for the network of Izhikevich neurons (both single and two populations), the deterministic rule (42) gives a narrower spread of the variable around the value of the mean-field model for a given \( N \) than the random algorithm (43). However, the random algorithm is still capable of achieving a good match of the amplitude and (usually) frequency of POs between the full network and the mean-field model.

See details in Fig. 7. As another example, Ciszak et al. (2021) showed that similar accuracy of the mean-field results was observed by employing a network of \( N = 10^4 \) nodes with the deterministic generation rule and one of \( N = 10^6 \) nodes with the random algorithm.

1. \( \eta \in (-\infty, \infty) \), range of the heterogeneous current.

This assumption is adopted in evaluation of the integrals (24) and (27) using the residue theorem. For the neural network to be realistic in spite of this requirement, the distribution range of the heterogeneous parameter should be much wider than its half-width at half-maximum to achieve Eq. (32) (see Eq. (50) in Appendix 7.2.5).

In sum, all the above eight requirements are not truly indispensable for applicability of the developed mean-field models. Some choices are a mere mathematical convenience, and insights gained from the macroscopic description are more generally applicable.

To our knowledge, the mean-field models we derived in this paper represent the first exact macroscopic description mainly in terms of the population firing rate for a spiking neural network with adaptation, which does not rely on assumptions of explicit separation of time-scales, weak coupling or averaging.

Nicola and Campbell (2013a, b) proposed a set of mean-field models for the homogeneous and heterogeneous Izhikevich network. A quasi-steady approximation was used by assuming the time scale of adaptation sufficiently large. The resulting mean-field model was a system of switching ordinary differential equations and the bifurcation theory of non-smooth systems had to be involved to perform further dynamical analysis. We used similar parameter values and thus our work can be directly compared. The shape of the bursting region and dependence of bursting on various parameter is consistent, which is satisfying since the bursting mechanism in the underlying network model is the same. Interestingly, we have found a novel mechanism for emergence of synchronous bursting in the neural network of two-coupled populations. Bursting was initiated by what appeared to be a saddle-node on an invariant circle bifurcation or a homoclinic bifurcation (see Fig. 5(c)). Moreover, our results greatly improve on those of (Nicola & Campbell, 2013a, b) in the sense of representation of the frequency of bursting. Gast et al. (2020) developed a smooth mean-field system for the QIF network with adaptation. The SFA mechanism acted additively to the dynamics of the membrane potential, just like the Izhikevich neuron. However, its adaptation variable was specifically expressed as a convolution of the membrane potential with an integral kernel. This treatment facilitates finding the closed set of mean-field equations, but lacks generality. Additionally, the adaptation dynamics
in (Gast et al., 2020) was also assumed to be slow enough that the variable was regarded as constant, finally leading to the same derivation process as that in (Montbrió et al., 2015). Recently, Bandyopadhyay et al. (2022) have derived a mean-field model for the network of the Hodgkin-Huxley neurons including the effect of ion-exchange between intracellular and extracellular environment. They use quasi-steady state assumptions and numerical fitting to approximate the Hodgkin-Huxley model by a piecewise-defined QIF model and then apply the Montbrió approach (Montbrió et al., 2015). However, the incorporation of the ion-exchange dynamics is ad hoc. By comparison, we provide explicit and solid mathematical foundations underlying the derivation process, that show how to incorporate such additional variables into a mean-field model.

In addition to the work built upon the Montbrió approach (Montbrió et al., 2015) dedicated to the network of QIF-type neurons with adaptation, there are a few other approaches to derive the mean-field models from populations of other models of adapting neurons. Overall, the adiabatic approximation (first or higher order statistic moments) is commonly employed to deal with the adaptation variables. The differences lie at how to develop the dynamics of the population firing rate. For example, di Volo et al. (2019) adopted a master equation formalism (El Boustani & Destexhe, 2009) to obtain a macroscopic Markovian description of the homogeneous network of AdEx integrate-and-fire neurons and subsequently extended it to the corresponding heterogeneous network (di Volo & Destexhe, 2021). Simultaneously, Carlu et al. (2020) generalized the approach to networks of more complex neuron models, including Hodgkin-Huxley and Morris-Lecar. The main ingredient of the master equation formalism is the derivation of a stationary transfer function. Key assumptions in the derivation of the mean-field model are that the network dynamics be asynchronous and that time-scale of the adaptation be slower than the spiking dynamics of the individual neurons (di Volo et al., 2019). Therefore, the deduced mean-field model cannot address the emergence of bursting induced synchronization, but is well-suited at describing the response of networks to external stimuli (see Fig. 3 and 4 in (Carlu et al., 2020)). In addition, different from the above mean-field approximation, which takes

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**Fig. 7** Time series of $r(t)$ for the single-population network (1-3) (blue) and its mean-field model (34) (red). We also show different distributions of the heterogeneous current $\eta$ when its mean $\bar{\eta} = 0.12$. In (a), the currents are generated deterministically according to the rule (42), while (b) and (c) are two independent experiments where the currents are generated in a random way as Eq. (43). Other parameters are the same as those in Fig. 1(d).
into account the network with finite neurons, Cakan and Obermayer (2020) presented a mean-field model based on a linear-nonlinear cascade (Augustin et al., 2017) of a network of AdEx neurons in the thermodynamic limit $N \to \infty$. The approach begins with the replacement of the synaptic current by a Gaussian white noise process, then formulates the stochastic network using the Fokker-Planck equation as the continuity equation. In our approach, we consider a deterministic network and use the counterpart continuity Eq. (4). Further, in (Cakan & Obermayer, 2020), similar to the master equation formalism, precomputed stationary transfer functions are necessary to derive the cascade-based mean-field model, which indirectly links to the dynamics of the population firing rate and suffers the same incapability in describing the neurons displaying bursting behaviours. In contrast, our approach introduces a Lorentzian distribution as the solution to the continuity equation, that leads to simpler and direct firing rate equations as the final mean-field description and goes beyond the limitation to bursting.

Interaction between fast and slow processes in a network of spiking neurons can induce much richer dynamics, especially the emergence of population bursting activity and the resulting spike synchronization. Those regimes are of interest to describe both normal and pathological neural network dynamics. The mean-field models developed in this paper provide a tractable and direct firing rate equations as the final mean-field solution to the continuity equation, that leads to simpler and direct firing rate equations as the final mean-field description and goes beyond the limitation to bursting.

The corresponding dimensionless form of the network model is

$$\frac{dV_k}{dT} = \frac{C}{k_1} (V_k - V_T)(V_k - V_R) - W_k$$

$$+ I_{app,k} + G_{syn}(E_r - V_k)$$

$$\tau_W \frac{dW_k}{dT} = \beta (V_k - V_R) - W_k$$

$$\frac{ds}{dT} = -\frac{s}{\tau_s} + \frac{S_{\text{jump}}}{N} \sum_{k=1}^{N} \sum_{t_{kj} < t} \delta(t - t_{kj})$$

if $V_k \geq V_{\text{peak}}$, then $V_k \leftarrow V_{\text{reset}}$

and $W_k \leftarrow W_k + W_{\text{jump}}$

where $k = 1, 2, \ldots, N$. Parameters are interpreted in Table 3 and values are chosen to fit hippocampal CA3 pyramidal neuron data.

The corresponding dimensionless form of the network model is

$$\frac{dv_k}{dt} = v_k (v_k - \alpha) - w_k + I_k + g_{\text{syn}}(e_r - v_k)$$

$$\frac{dv_k}{dt} = a (bv_k - w_k)$$

$$\frac{ds}{dt} = -\frac{s}{\tau_s} + \frac{S_{\text{jump}}}{N} \sum_{k=1}^{N} \sum_{t_{kj} < t} \delta(t - t_{kj})$$

where $I_k$ corresponds to $\eta_k + I_{\text{ext}}$ in Eq. (1).

From the transformations,

$$V_k - V_R = V_k + |V_R| = |V_R| \left(1 + \frac{V_k}{|V_R|}\right)$$

$$\frac{dV_k}{dT} = \frac{C}{k_1 |V_R|} \frac{dv_k}{dt} = \frac{C}{k_1 |V_R|} \frac{dt}{dT} \frac{dv_k}{dt} = \frac{dv_k}{dt}$$

we can define

$$V_R = V_R + 40 - \frac{E_r}{k_1}$$

### 7 Appendix

#### 7.1 Dimensional Izhikevich network and its nondimensionalization

The dimensional form of the network of Izhikevich neurons is the same as that described by Nicola and Campbell (2013a) and given by

$$C \frac{dV_k}{dT} = k_1 (V_k - V_T)(V_k - V_R) - W_k$$

$$+ I_{app,k} + G_{syn}(E_r - V_k)$$

$$\tau_W \frac{dW_k}{dT} = \beta (V_k - V_R) - W_k$$

$$\frac{ds}{dT} = -\frac{s}{\tau_s} + \frac{S_{\text{jump}}}{N} \sum_{k=1}^{N} \sum_{t_{kj} < t} \delta(t - t_{kj})$$

if $V_k \geq V_{\text{peak}}$, then $V_k \leftarrow V_{\text{reset}}$

and $W_k \leftarrow W_k + W_{\text{jump}}$

where $k = 1, 2, \ldots, N$. Parameters are interpreted in Table 3 and values are chosen to fit hippocampal CA3 pyramidal neuron data.

The corresponding dimensionless form of the network model is

$$\frac{dv_k}{dt} = v_k (v_k - \alpha) - w_k + I_k + g_{\text{syn}}(e_r - v_k)$$

$$\frac{dv_k}{dt} = a (bv_k - w_k)$$

$$\frac{ds}{dt} = -\frac{s}{\tau_s} + \frac{S_{\text{jump}}}{N} \sum_{k=1}^{N} \sum_{t_{kj} < t} \delta(t - t_{kj})$$

where $I_k$ corresponds to $\eta_k + I_{\text{ext}}$ in Eq. (1).

From the transformations,

$$V_k - V_R = V_k + |V_R| = |V_R| \left(1 + \frac{V_k}{|V_R|}\right)$$

$$\frac{dV_k}{dT} = \frac{C}{k_1 |V_R|} \frac{dv_k}{dt} = \frac{C}{k_1 |V_R|} \frac{dt}{dT} \frac{dv_k}{dt} = \frac{dv_k}{dt}$$

we can define

| Parameter | Value | Description |
|-----------|-------|-------------|
| $C$       | 250pF | Membrane capacitance |
| $k_1$     | 2.5nS/mV | Scaling factor |
| $V_R$     | -65mV | Resting potential |
| $V_T$     | -24.6mV | Threshold potential |
| $G_{\text{syn}}$ | 200nS | Synaptic conductance |
| $E_r$     | 0mV | Reversal potential |
| $\beta$   | -1nS | Scaling factor |
| $\tau_W$  | 200mS | Time constant of $W$ |
| $\tau_s$  | 4mS | Time constant of $s$ |
| $S_{\text{jump}}$ | 0.8 | Coupling strength |
| $W_{\text{jump}}$ | 200pA | After-spike jump size |

$V_R = V_R + 40 - \frac{E_r}{k_1}$
Table 4  Scaling relations between the dimensionless and dimensional neural networks

| Term1 | Term2 |
|-------|-------|
| $v_i = 1 + \frac{V_i}{|V_i|}$ | $\delta k = \frac{w_k}{k_1|V_i|^2}$ |
| $s = \bar{s}$ | $T = \frac{k_1|V_i|^2}{c}$ |
| $v_{\text{peak}} = 1 + \frac{V_{\text{peak}}}{|V_i|}$ | $v_{\text{reset}} = 1 + \frac{V_{\text{reset}}}{|V_i|}$ |
| $\alpha = 1 + \frac{V_i}{|V_i|}$ | $g_{\text{syn}} = \frac{e_{\text{syn}}}{k_1|V_i|}$ |
| $a = \left(\frac{e_a k_1|V_i|}{c}\right)^{-1}$ | $b = \frac{\beta}{k_1|V_i|}$ |
| $s_{\text{jump}} = s_{\text{jump}} c \frac{k_1}{k_1|V_i|}$ | $w_{\text{jump}} = \frac{w_{\text{jump}}}{k_1|V_i|^2}$ |
| $e_v = 1 + \frac{v}{|V_i|}$ | $I = \frac{I_{\text{jump}}}{k_1|V_i|^2}$ |

Table 4 gives the whole scaling relationship between the dimensionless and dimensional systems.

7.2 Intermediate derivation steps for the mean-field equations

7.2.1 Derivation of Eq. (11)

The derivation is modified from Nicola and Campbell (2013b). The continuity Eq. (4) yields

$$\langle w \rangle' = \int d\eta \int dv \int dw \frac{\partial}{\partial t} \rho(t, v, w, \eta) dw d\eta$$

$$= - \int d\eta \int dv \int dw w \left( \frac{\partial \mathcal{F}}{\partial v} + \frac{\partial \mathcal{F}}{\partial w} \right) dw d\eta$$  \hspace{1cm} (44)

Next, we evaluate the two terms on the right hand side of (44), respectively. For the first term, we integrate by parts and change the order of integration as needed, then obtain

$$\text{Term1} = \int \int \int \int \frac{\partial}{\partial t} \mathcal{F}(t, v, w, s, \eta) dw d\eta$$

$$= \int \int \int \int \mathcal{F}(t, v, w, s, \eta) dw d\eta$$

$$= \int \int \int \int \mathcal{F}(t, v, w, s, \eta) dw d\eta$$

$$= \int \int \int \int \mathcal{F}(t, v, w, s, \eta) dw d\eta$$

$$= \int \int \int \left( \mathcal{F}(t, v_{\text{peak}}, w, s, \eta) \right) dw d\eta$$

$$= \int \int \left( \mathcal{F}(t, v_{\text{peak}}, w, s, \eta) \right) dw d\eta$$

$$= \int \int \left( \mathcal{F}(t, v_{\text{peak}}, w, s, \eta) \right) dw d\eta$$

Assuming $\langle w | \eta \rangle \gg w_{\text{jump}}$, we apply a Taylor expansion and integration by parts and have

$$\text{Term1} = \int \int \int \int \frac{\partial}{\partial t} \mathcal{F}(t, v_{\text{peak}}, w, s, \eta) dw d\eta$$

$$= \int \int \int \int \mathcal{F}(t, v_{\text{peak}}, w, s, \eta) dw d\eta$$

Similarly, for the second term of (44), we have

$$\text{Term2} = \int \int \int \int \frac{\partial}{\partial t} \mathcal{F}(t, v, w, \eta) dw d\eta$$

$$= \int \int \int \int \mathcal{F}(t, v, w, \eta) dw d\eta$$

Finally, we obtain the differential equation of the mean adaptation variable in terms of flux given by

$$\langle w \rangle' = \langle G(v, v, w) \rangle + \int \int \int w_{\text{jump}} \mathcal{F}(t, v_{\text{peak}}, w, s, \eta) dw d\eta.$$

7.2.2 Derivation of Eq. (15)

$$r(t) = \lim_{t \rightarrow v_{\text{peak}}} \int \int \int \mathcal{F}(t, v, w, s, \eta) dw d\eta$$

$$= \lim_{t \rightarrow v_{\text{peak}}} \int \int \int G^*(v, w, s, \eta) \rho(t, v, w, \eta) dw d\eta$$

$$= \lim_{t \rightarrow v_{\text{peak}}} \int \int \int G^*(v, w, s, \eta) \rho^*(t, v, w, \eta) \rho'(t, v | \eta) \mathcal{L}(\eta) dw d\eta$$

$$= \lim_{t \rightarrow v_{\text{peak}}} \int \int \int \mathcal{L}(\eta) \rho'(t, v | \eta) \cdot \int G^*(v, w, s, \eta) \rho^*(t, v | \eta) \mathcal{L}(\eta) dw d\eta$$

$$= \lim_{t \rightarrow v_{\text{peak}}} \int \int \int \mathcal{L}(\eta) \rho'(t, v | \eta) \cdot G^*(v, w, s, \eta) \mathcal{L}(\eta) dw d\eta.$$
7.2.3 Derivation of Eq. (18)

\[
\langle v(t) \rangle = \int \int \int v \rho(t, v, w, \eta) \, dv \, dw \, d\eta
\]

\[
= \int \int \int v \rho^{w}(t, w|v, \eta) \cdot \rho^{\prime}(t, v|\eta) \, dv \, dw \, d\eta
\]

\[
= \int \mathcal{L}(\eta) \int v \rho^{\prime}(t, v|\eta) \, dv \, d\eta,
\]

7.2.4 Derivation of Eq. (28)

We substitute the LA ansatz

\[
\rho^{\prime}(t, v|\eta) = \frac{1}{\pi} \frac{x(t, \eta)}{[y - y(t, \eta)]^2 + x^2(t, \eta)}.
\]

into the continuity equation in terms of the conditional density function,

\[
\frac{\partial}{\partial t} \rho^{\prime}(t, v|\eta) = - \frac{\partial}{\partial v} \left[ G^{\prime}(v, \langle \eta|w\rangle, s) \rho^{\prime}(t, v|\eta) \right].
\]

Then, the first term becomes

\[
\partial_t \rho^{\prime}(t, v|\eta) = \frac{1}{\pi} \frac{x(t, \eta)}{[(y - y(t, \eta))^2 + x^2(t, \eta)]^2},
\]

and the second term,

\[
\frac{\partial}{\partial v} \left[ G^{\prime}(\cdot) \rho^{\prime}(\cdot) \right] = (2v - a - g_s) \rho^{\prime}(t, v|\eta)
\]

\[
+ \left[ v^2(v - a) - \langle \eta|w\rangle + \eta + I_{ext} + g_{syn} \langle \eta|w\rangle - \langle \eta|w\rangle \right] \partial_v \rho^{\prime}(t, v|\eta),
\]

where we can also obtain

\[
\partial_v \rho^{\prime}(t, v|\eta) = \frac{1}{\pi} \frac{2x(v - y)}{[(v - y)^2 + x^2]^2}.
\]

Equating the resulting equation in the powers of \(v\), yielding

\[
v^2 \left[ x^2 - 2xy + (a + g_{syn})v + \left( 2v^2y - 2v^2y \right) 
+ 2x(2x^2 + y^2) + 2v(v - a - \langle \eta|w\rangle + \eta - I_{ext} - g_{syn} \langle \eta|w\rangle)ight]
+ \left[ (v^2)^2 - 2xy^2 - (a + g_{syn})(x^2 + y^2) + 2xy(\eta - \langle \eta|w\rangle + g_{syn} \langle \eta|w\rangle) \right] = 0
\]

Letting the coefficients of \(v^2\) and \(v\) be zero, we have

\[
x' = 2xy - (a + g_{syn})x,
\]

\[
y' = y(y - a) - x^2 - \langle \eta|w\rangle + \eta + I_{ext} + g_{syn} \langle \eta|w\rangle - \langle \eta|w\rangle.
\]

Both of them lead to the disappearance of the rest of terms in Eq. (46).

7.2.5 Derivation of Eq. (32)

To evaluate the two integrals

\[
r(t) = \int \frac{1}{\pi} x(t, \eta) \mathcal{L}(\eta) d\eta
\]

\[
\langle v(t) \rangle = \int \langle \eta|w\rangle \mathcal{L}(\eta) d\eta,
\]

we begin by denoting the line integral around an oriented rectifiable curve \(\gamma\),

\[
\oint \int f(\eta) d\eta = \oint \int \frac{1}{\pi} x(t, \eta) \mathcal{L}(\eta) d\eta.
\]

Substituting the Lorentzian assumption

\[
\mathcal{L}(\eta) = \frac{1}{\pi} \frac{\Delta_{\eta}}{(\eta - \eta)^2 + \Delta^2_{\eta}}
\]

yields

\[
f(\eta) = \frac{x(t, \eta)}{\pi^2} \frac{\Delta_{\eta}}{(\eta - \eta)^2 + \Delta^2_{\eta}}
\]

\[
= \frac{1}{2i} \frac{x(t, \eta)}{\eta - (\eta + i\Delta_{\eta})} - \frac{1}{\eta - (\eta - i\Delta_{\eta})}.
\]

Then, the residue of \(f(\eta)\) at the point \(\eta_1 = \eta - i\Delta_{\eta}\) is

\[
\text{Res}(f, \eta_1) = -\frac{x(t, \eta + i\Delta_{\eta})}{2\pi^2 i}
\]

Let’s define the clockwise contour \(\gamma\) (shown in Fig. 8) that goes along the real line from \(-a\) to \(a\) and then clockwise along a semicircle centered at the origin from \(a\) to \(-a\).

Take \(a\) to be greater than \(\Delta_{\eta}\), so that the point \(\eta_1 = \eta - i\Delta_{\eta}\) is enclosed within the curve.

According to the residue theorem, we have

\[
\oint \int f(\eta) d\eta = 2\pi i \times \text{Res}(f, \eta_1) = \frac{1}{\pi} x(t, \eta + i\Delta_{\eta})
\]

\[
\oint \int f(\eta) d\eta = \int_{\text{straight}} f(\eta) d\eta + \int_{\text{arc}} f(\eta) d\eta
\]

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Fig. 8 The contour \( \gamma \) for the parameter \( \eta \) using to solve the integrals \( (47) \) when \( a \rightarrow +\infty \).

and thus
\[
\int_{-a}^{a} f(\eta)d\eta = \frac{1}{\pi} x(t, \tilde{\eta} - i\Delta_\eta) - \int_{arc} f(\eta)d\eta \tag{48}
\]

Next, we will show the integral \( \int_{arc} f(\eta)d\eta \) goes to zero as \( a \rightarrow +\infty \). From the estimation lemma, also known as the ML inequality, we have
\[
\left| \int_{arc} f(\eta)d\eta \right| \leq l(\text{arc}) \cdot \sup_{\text{arc}} \left| \frac{1}{\pi^2} x(t, \eta) \frac{\Delta_\eta}{(\eta - \tilde{\eta})^2 + \Delta_\eta^2} \right| \tag{49}
\]

where \( l(\text{arc}) \) is the arc length of the contour \( \gamma \) shown in Fig. 8. By observing that the arc is half the circumference of a circle with radius \( a \), we have
\[
l(\text{arc}) = \frac{1}{2}(2\pi a) = \pi a.
\]

Then, we seek the upper bound \( \sup_{\text{arc}} \left| \frac{1}{\pi^2} x(t, \eta) \frac{\Delta_\eta}{(\eta - \tilde{\eta})^2 + \Delta_\eta^2} \right| \).

By the triangle inequality we see that
\[
|\eta - \tilde{\eta}|^2 = |(\eta - \tilde{\eta})|^2
= |(\eta - \tilde{\eta})|^2 + \Delta_\eta^2 - \Delta_\eta^2
\leq |(\eta - \tilde{\eta})|^2 + \Delta_\eta^2 + \Delta_\eta^2.
\]

Thus,
\[
|(\eta - \tilde{\eta})^2 + \Delta_\eta^2| \geq |\eta - \tilde{\eta}|^2 - \Delta_\eta^2.
\]

When \( \eta \) goes to \( \pm \infty \), i.e., \( a \rightarrow +\infty \), we have
\[
\left| \frac{1}{|\eta - \tilde{\eta}|^2 + \Delta_\eta^2} \right| \leq \frac{1}{|\eta - \tilde{\eta}|^2 - \Delta_\eta^2} = \frac{1}{a^2 - \Delta_\eta^2}.
\]

Further, assuming that the half-width \( |x(\eta, t)| \) is bounded above by a constant \( C \), we obtain
\[
\left| \frac{1}{\pi^2} x(t, \eta) \frac{\Delta_\eta}{(\eta - \tilde{\eta})^2 + \Delta_\eta^2} \right| \leq \frac{C \Delta_\eta}{\pi^2} \cdot \frac{1}{a^2 - \Delta_\eta^2}.
\]

Finally, from Eqs. (48-50) we derive
\[
\int_{-\infty}^{\infty} f(\eta)d\eta = \frac{1}{\pi} x(t, \tilde{\eta} - i\Delta_\eta),
\]

that is,
\[
\rho(t) = \int_{-\infty}^{\infty} f(\eta)d\eta = \frac{1}{\pi} x(t, \tilde{\eta} - i\Delta_\eta).
\]

Similarly, we can derive
\[
\rho(t, v) = \int_{-\infty}^{\infty} y(t, \eta)g(\eta, t)d\eta = y(t, \tilde{\eta} - i\Delta_\eta).
\]

7.3 Mean-field approximation for two-coupled Izhikevich populations

Considering a network of two-coupled populations of Izhikevich neurons \( (35)-(37) \), we describe the two ensembles of neurons by their own distinct probability density function. Each density follows a continuity equation in the form of Eq. (20). For the population \( p \), we have
\[
\frac{\partial}{\partial t} \rho_p(t, v|\eta_p) = - \frac{\partial}{\partial v} \left[ G_p^\prime(v, (w|\eta_p), s, \eta_p) \rho_p(t, v|\eta_p) \right] \tag{51}
\]

where \( G_p^\prime(\cdot) \) is defined as Eq. (6) with the subscript \( p \) for each element. A boundary condition for the flux \( \mathcal{J}_p^\prime(\cdot) = G_p^\prime(\cdot) \rho_p^\prime(\cdot) \) is imposed as Eq. (7) according to the reset mechanism of the Izhikevich neuron.

Assume that the solution of the continuity Eq. (51) has the form of a Lorentzian distribution,
\[
\rho_p(t, v|\eta_p) = \frac{1}{\pi} \frac{\chi_p(t, \eta_p)}{[v - y_p(t, \eta_p)]^2 + \chi_p^2(t, \eta_p)}. \tag{52}
\]

The firing rate of the population \( r_p(t) \) is the flux through the threshold \( v_{\text{peak}}^p \). Having set the threshold at infinity and considering the Lorentzian solution (52), we obtain
\[
r_p(t) = \lim_{v_{\text{peak}}^p \rightarrow \infty} \int_{\eta_p} \mathcal{L}_p(\eta_p) \rho_p(t, v|\eta) \cdot G_p^\prime(v, (w|\eta), s, \eta_p) d\eta_p
\]
\[
= \int_{\eta_p} \frac{1}{\pi} \chi_p(t, \eta_p) \mathcal{L}_p(\eta_p) d\eta_p \tag{53}
\]
where $L_p$ defines a probability that a randomly chosen cell in the population $p$ has an intrinsic parameter $\eta_p$. In addition, the mean membrane potential has a relationship with $y_p(t, \eta_p)$ via

$$\langle v(t) \rangle_p = \int_{-\infty}^{\infty} y_p(t, \eta_p) L_p(\eta_p) d\eta_p,$$

(54)

like that in Eq. (24) but with the subscript $p$.

Assume the distribution of the heterogeneous parameter $\eta_p$ has the form of Lorentzian function,

$$L_p(\eta_p) = \frac{\Delta_p}{\pi (\eta_p - \bar{\eta}_p)^2 + \Delta_p^2}.$$

(55)

The integrals in (53) and (54) can be evaluated by using the residue theorem and Eq. (55). Then, denoting the complex variable $z_p(t, \eta_p) = x_p(t, \eta_p) + i y_p(t, \eta_p)$, we can derive an explicit relation of the firing rate and the mean membrane potential,

$$\pi r_p(t) + i \langle v(t) \rangle_p = z_p(t, \bar{\eta}_p - i \Delta_p),$$

(56)

where $z_p(t, \eta_p)$ follows the complex differential equation,

$$\frac{d}{dt} z_p = i \left[ -z_p^2 + i \eta_p (\alpha_p + GS_p) ight] - \langle w | \eta_p \rangle_p + \eta_p + r_{ext}^p + e_r^p GS_p].$$

(57)

Here, $GS_p$, given in Eq. (37), is defined as the compound synaptic conductance resulting from the coupling within and between the populations.

Applying (56) into (57) and combining the mean adaptation dynamics in Eq. (12) with the subscript $p$ will therefore lead to the mean-field model for the population $p$,

$$r'_p = \Delta_p^2/\pi + 2\pi r_p(v)_p - r_p[GS_p + \eta_p] + \langle v \rangle_p^2 - \alpha_p(\langle v \rangle_p - (w)_p + \bar{\eta}_p) + r_{ext}^p + GS_p [e_r^p - \langle v \rangle_p] - \pi z_r^2$$

$$\langle w \rangle_v = a_p (b_p(v)_p - \langle v \rangle_p) + w_{jump} r_p$$

and for the population $q$,

$$r'_q = \Delta_q^2/\pi + 2\pi r_q(v)_q - r_q[GS_q + \eta_q] + \langle v \rangle_q^2 - \alpha_q(\langle v \rangle_q - (w)_q + \bar{\eta}_q) + r_{ext}^q + GS_q [e_r^q - \langle v \rangle_q] - \pi z_r^2$$

$$\langle w \rangle_q = a_q (b_q(v)_q - \langle v \rangle_q) + w_{jump} r_q$$

(58)

(59)

These two subsystems are coupled through synaptic currents as given in Eq. (37), with the synaptic dynamics governed by

$$s'_p = -s_p/r_p + s_{jump}^p r_p$$

$$s'_q = -s_q/r_q + s_{jump}^q r_q.$$

(60)

Acknowledgements This work benefited from the support of the Natural Sciences and Engineering Research Council of Canada. We would like to thank the reviewers for their comments which helped improve this article.

Code availability statement The computer code for this study can be found on ModelDB (McDougall et al., 2017) under the accession number: 267382.

Declarations

Conflict of interest statement The authors declare they have no conflict of interest.

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