Synchrony-induced modes of oscillation of a neural field model

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We investigate the modes of oscillation of heterogeneous ring-networks of quadratic integrate-and-fire (QIF) neurons with non-local, space-dependent coupling. Perturbations of the equilibrium state with a particular wave number produce transient standing waves with a specific frequency, analogous to those in a tense string. In the neuronal network, the equilibrium corresponds to a spatially homogeneous, asynchronous state. Perturbations of this state excite the network’s oscillatory modes, which reflect the interplay of episodes of synchronous spiking with the excitatory-inhibitory spatial interactions. In the thermodynamic limit, an exact low-dimensional neural field model (QIF-NFM) describing the macroscopic dynamics of the network is derived. This allows us to obtain formulas for the Turing eigenvalues of the spatially-homogeneous state, and hence to obtain its stability boundary. We find that the frequency of each Turing mode depends on the corresponding Fourier coefficient of the synaptic pattern of connectivity. The decay rate instead, is identical for all oscillation modes as a consequence of the heterogeneity-induced desynchronization of the neurons. Finally, we numerically compute the spectrum of spatially-inhomogeneous solutions branching from the Turing bifurcation, showing that similar oscillatory modes operate in neural bump states, and are maintained away from onset.

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I. INTRODUCTION

Since the pioneering work of Wilson-Cowan [1], Amari [2, 3], and Nunez [4], continuum descriptions of neuronal activity have become a powerful modeling tool in neuroscience [5–10]. Given that the number of neurons in a small region of cortex is very large, these descriptions consider neurons to be distributed along a continuous spatial variable, and the macroscopic state of the network to be described by a single, space-dependent, firing rate variable. The resulting neural field model (NFM) generally has the form of a continuous first order integro-differential equation, greatly facilitating the computational and mathematical analysis of the dynamics of large neuronal networks.

NFMs do not generally represent proper mathematical reductions of the mean activity of a network of spiking neurons. Nevertheless, NFMs have proven to be remarkably accurate in qualitatively capturing the main types of dynamical states seen in networks of large numbers of asynchronous spiking neurons. For example it is well known that, in local networks of spiking neurons, differences between excitatory and inhibitory neurons can lead to oscillations [11–13]. The generation of these oscillations does not depend on the spatial character of the network, and hence can be observed in non-spatially dependent firing rate models [12]. When the pattern of synaptic connectivity depends on the distance between neurons, NFMs show that these differences between excitation and inhibition can lead to the emergence of oscillations and waves [3, 14]. Similar patterns can also be found in NFMs with spatially dependent delays —modeling the effect of the finite velocity propagation of action potentials [1, 15]— as a great deal of theoretical work indicates, see e.g. [16–23].

In some cases the spatio-temporal dynamics of NFMs has been directly compared to that observed in analogous networks of spiking neurons [24–26]. In this work it was found that non-space-dependent delays predict the existence of many of the spatio-temporal patterns observed in asynchronous networks of spiking neurons with non-local, space-dependent interactions. The success of NFMs in describing these patterns depends crucially on the spatial activity being highly asynchronous. In fact, it is well known that neural field descriptions fail to describe states characterized by a high degree of spike synchronization, see e.g [27].

Here we report a spatio-temporal dynamical feature of heterogeneous networks of spiking neurons with non-local interactions that, to the best of our knowledge, have been so far unexplored. We show that ring networks of spiking neurons display a number of discrete modes of oscillation, resembling those of a tense string. These modes are exclusively due to transient episodes of synchronous spiking and not due to the different time scales between excitation and inhibition, nor to the presence of any propagation or synaptic delay.

Traditional NFMs do not describe these synchrony-induced oscillations. Therefore, to investigate and characterize them, we apply a recent method to derive the firing rate equations of a globally coupled heterogeneous population of quadratic integrate and fire (QIF) neurons [28]. This method, based on the so-called Ott-Antonsen theory [29–31], leads to an exact macroscopic description of the network in terms of two macroscopic variables: the mean firing rate and the mean membrane potential. The resulting mean-field model exactly describes any state of the system, including synchronous states. Here we extend the local firing rate model in [28], to include non-local, instantaneous interactions. The resulting neural field model for heterogeneous QIF neurons (QIF-NFM) clearly displays the synchrony-induced oscill-
The location of neurons is parameterized by the angular variable \( \phi \) and the variables \( \phi_j \) and \( \phi_k \) of the excitatory and inhibitory neurons in the network under investigation. The model consists of spike synchronization modes observed in simulations of spiking neurons. We then thoroughly investigate the QIF-NFM by means of both a linear and non-linear stability analysis of the spatially homogeneous state. The analysis reveals the presence of an infinite number of oscillation modes, linked to the Fourier components of the spatial pattern of synaptic connections. The analysis also shows that all modes decay to the unpatterned state with the same rate, which depends on the degree of heterogeneity in the network. Finally, we investigate the spectrum of the spatially inhomogeneous solutions of the QIF-NFM and find similar oscillatory modes also linked to transient episodes of spike synchronization.

**II. SYNCHRONY-INDUCED MODES OF OSCILLATION IN NETWORKS OF QUADRATIC INTEGRATE AND FIRE (QIF) NEURONS**

Figure 1(a) shows a schematic representation of the spiking neuron network under investigation. The model consists of \( N \) excitatory (Red) and \( N \) inhibitory (Blue) neurons evenly arranged on a ring. Any neuron in the network interacts with all the other neurons via the distance-dependent coupling function \( J_{jk}^{c,i} = J_{jk}^{c,i}(|\phi_j - \phi_k|) \), where indices \( e, i \) denote excitatory and inhibitory connections, respectively. The synaptic projections of the \( j \)-th excitatory and inhibitory neurons (located at \( \phi_j \)) to other two nearby neurons are also schematically represented in Figure (1.a).

The ring architecture of the network allows one to express the excitatory and inhibitory connectivity patterns in Fourier series as

\[
J^{c,i}(\phi) = J_0^{c,i} + 2 \sum_{K=1}^{\infty} J_K^{c,i} \cos(K\phi). \quad (1)
\]

Figure (1,b) shows a particular synaptic connectivity pattern in which excitatory neurons form strong, short-range connections, whereas inhibitory projections are weaker and wider. The state of the excitatory \( (e) \) and inhibitory \( (i) \) neurons is determined by the membrane potentials \( \{v_j^{e(i)}\}_{j=1,...,N} \), which are modeled using the Quadratic Integrate and Fire (QIF) model [32, 33]

\[
\frac{dv^{e,i}}{dt} = (v_j^{e,i})^2 + I_j^{e,i} \quad (\text{+ resetting rule}), \quad (2)
\]

where \( \tau \) is the cell’s membrane time constant and, \( v_r \) and \( v_p \) correspond to the reset and peak potentials of the QIF neurons, respectively — in numerical simulations we consider \( \tau = 20 \text{ ms} \). The QIF neuron has two possible dynamical regimes depending on the input current \( I_j^{e,i} \). If \( I_j^{e,i} < 0 \), the neuron is in the excitable regime, while for \( I_j^{e,i} > 0 \) the neuron is in the oscillatory regime. In the excitable regime, an initial condition \( v_j^{e,i}(0) < \sqrt{-I_j^{e,i}} \), asymptotically approaches the resting potential \( -\sqrt{-I_j^{e,i}} \). On the other hand, initial conditions above the excitability threshold, \( v_j^{e,i}(0) > \sqrt{-I_j^{e,i}} \), make the membrane potential to grow without bound. Specifically, if \( v_j^{e,i}(0) \gg \sqrt{-I_j^{e,i}} \), the membrane potential reaches infinity approximately after a time \( \tau/v_j^{e,i} \). In practice, to avoid this divergence, we consider the following resetting rule: When the neuron’s membrane potential \( v_j^{e,i} \) reaches a certain peak value \( v_p \gg 1 \), the neuron is reset to the new value \( v_r = -v_p \) after a refractory period \( 2\tau/v_r \). On the other hand, if \( I_j^{e,i} > 0 \), the neuron is in the oscillatory regime and needs to be reset periodically. If \( v_p \gg 1 \), the frequency of the oscillatory neurons is approximately \( f_j = \sqrt{I_j^{e,i}/(\tau \pi)} \). Finally, the current \( I_j^{e,i} \) is defined as

\[
I_j^{e,i} = \eta_j^{e,i} + \tau S_j^{e}(t) + \tau S_j^{i}(t) + P_j^{e,i}(t). \quad (3)
\]

Here, \( \eta_j^{e,i} \) is a constant external current, which varies from neuron to neuron (note that the voltages \( v_j \) and currents \( I_j \) are dimensionless). The terms \( P_j^{e,i}(t) \) are time-varying common inputs, and \( S_j^{e,i}(t) \) are the mean excitatory (positive) and inhibitory (negative) synaptic activities representing all the weighted inputs received by neuron \( j \) due to spiking activity in the network:

\[
S_j^{e,i}(t) = \pm \sum_{k=1}^{N} \frac{J_{jk}^{e,i}}{2\pi N} \sum_{l \neq j, l < t} \frac{1}{\tau_e} \int_{t-\tau_e}^{t} dt' \delta^{e,i}(t' - t_k), \quad (4)
\]
neural field model for a network of QIF neurons (QIF-NFM) [34]. The detailed derivation is performed in Appendix A, and closely follows that of [28]. The reduction in dimensionality is achieved considering that the currents $\eta^{e,i}$ —which, after performing the thermodynamic limit become continuous random variables—are distributed according to a Lorentzian distribution of half-width $\Delta$ and centered at $\bar{\eta}$.

$$g(\eta^{e,i}) = \frac{1}{\pi} \frac{1}{(\eta^{e,i} - \bar{\eta})^2 + \Delta^2}. \quad (5)$$

The QIF-NFM is

$$\tau \frac{dR^{e,i}}{dt} = \frac{\Delta}{\pi \tau} + 2R^{e,i}V^{e,i}, \quad (6a)$$
$$\tau \frac{dV^{e,i}}{dt} = (V^{e,i})^2 + \bar{\eta} - (\pi \tau R^{e,i})^2 + \tau S(\phi) + P^{e,i}(\phi, t). \quad (6b)$$

and exactly describes the time evolution of the mean firing rate $R^{e,i}(\phi)$, and the population’s mean membrane potential $V^{e,i}(\phi)$ of the excitatory and inhibitory populations at any location $\phi$ of the ring —to facilitate the notation we have avoided explicitly writing the dependence of these variables on $\phi$. In the limit of instantaneous synapses, $\tau_s \to 0$ in Eqs. (4), the excitatory and inhibitory contributions of the mean field $S(\phi) = S^e(\phi) + S^i(\phi)$ reduce to $S^{e,i}(\phi) = \pm \frac{1}{2\pi} \int_{-\pi}^{\pi} J^{e,i}(\phi - \phi')R^{e,i}(\phi')d\phi'$.

A. Effective QIF-NFM

The analysis of the QIF-NFM Eq. (6) is greatly simplified considering that excitatory and inhibitory neurons have identical single cell properties. This scenario is schematically represented in Figure (1;c,d). In this case, the solutions of Eqs. (6) satisfy $R^e(\phi, t) = R^i(\phi, t) \equiv R(\phi, t)$ and $V^e(\phi, t) = V^i(\phi, t) \equiv V(\phi, t)$. These solutions exist if $P^e(\phi, t) = P^i(\phi, t) = P(\phi, t)$, and coincide with the solutions of the effective QIF-NFM

$$\tau \frac{dR}{dt} = \frac{\Delta}{\pi \tau} + 2RV, \quad (7a)$$
$$\tau \frac{dV}{dt} = V^2 + \bar{\eta} - (\pi \tau R)^2 + \tau S(\phi) + P(\phi, t). \quad (7b)$$

In this case, the mean field reduces to

$$S(\phi) = \frac{1}{2\pi} \int_{-\pi}^{\pi} \left[ J_0 + 2 \sum_{k=1}^{\infty} J_K \cos(K(\phi' - \phi)) \right] R(\phi')d\phi', \quad (8)$$

with the new Fourier coefficients $J_K$, which are related to those in Eq. (1) as $J_K = J_{-K} - J_K$, with $K = 0, 1, \ldots$, see Fig.1(d). Note that, in Figs. (2;a,b), we perturbed the spatially homogeneous state (SHS) of the system Eqs. (2,3) using a current pulse to all the excitatory neurons. The resulting dynamics is only captured by the full system Eqs. (6) and not by the effective neural field Eqs. (7). However we next show that the existence of the spatial oscillatory modes observed in Fig.2 is exclusively linked to the dynamics in the reduced manifold defined by Eqs. (7, 8).

III. NEURAL FIELD MODEL FOR QUADRATIC INTEGRATE AND FIRE NEURONS (QIF-NFM)

In the following, we aim to investigate the nature and origin of the spatio-temporal patterns shown in Figure 2. To analyze them, we derive the NFM corresponding to the thermodynamic ($N \to \infty$) and continuum limits of the network of QIF neurons Eqs. (2,3). In addition we also take the limit $\nu_k \to \infty$, so that the QIF model (2) is equivalent to the so-called theta-neuron model [32, 33]. This leads to an exact
so that Eq. (9) has countably infinite set of eigenvalues associated to the stability of the fixed point of the SHS, the mean field Eq. (8) becomes spatially independent, for the variable \( \Phi(\phi) = \Re(\lambda_{K}^\pm) \). Substituting Eq. (9) with \( J_0 = 0 \) into Eq. (10), it is straightforward to find the boundary

\[
J_K^o = \sqrt{2\pi \sqrt{\eta^2 + \Delta^2}} \times \eta \pm (2\pi^2 R_\ast) \times \frac{\Delta}{\pi \tau^2 R_\ast} - 1, \quad (K = 0, 1, 2 \ldots)
\]

(10)

This equation is the main result of this work, and explains the synchronization patterns shown in Fig. 2. Note that the eigenvalues Eq. (10) may be real or complex, indicating non-oscillatory or oscillatory dynamics of the evolution of perturbations of wavenumber \( K \), respectively. In particular, perturbations of any given spatial mode \( K \) are oscillatory if the condition \( J_K < 2\pi^2 R_\ast \) is fulfilled. Notably, all complex eigenvalues have the same decay rate to the SHS, since \( \Re(\lambda_{K}^\pm) = -\Delta/(\pi \tau^2 R_\ast) \) for all of them. Specifically, the decay rate is proportional to the degree of quenched heterogeneity \( \Delta \). This reflects the fact that the decay in the oscillations is in fact a desynchronization mechanism due to the distribution of inputs that the cells receive.

Substituting Eq. (9) with \( J_0 = 0 \) into Eq. (10), it is straightforward to find the boundary

\[
J_K^o = \sqrt{2\pi \sqrt{\eta^2 + \Delta^2}} \times \eta \pm (2\pi^2 R_\ast) \times \frac{\Delta}{\pi \tau^2 R_\ast} - 1, \quad (K = 0, 1, 2 \ldots)
\]

(11)

separating the parameter space into regions where standing waves of wavenumber-K are, or are not observed. This boundary is depicted with a dotted line in the phase diagram Fig. (3), together with a schematic representation of the location of the eigenvalues \( \lambda_{K}^\pm \) in the complex plane (red crosses, see also Fig.(5,a)).

A given oscillatory mode \( K \) has an associated frequency \( \nu_K = 1/(2\pi) |\Im(\lambda_{K}^\pm)| \), which differs from one another depending on the corresponding Fourier coefficients \( J_K \) of the patterns of synaptic connectivity Eq. (1). Therefore, spatial perturbations of wavenumber \( K \), produce standing waves of neural activity of frequency \( \nu_K \). Locally excitatory coupling \( J_K > 0 \) slows down these oscillations and eventually suppresses them, whereas locally inhibitory coefficients \( J_K < 0 \) are able to generate arbitrarily fast oscillations (in particular, note that all modes with \( J_K = 0 \) are oscillatory with frequency \( \nu = R_\ast \), which coincides with the mean firing rate of the uncoupled neurons).

Indeed, in Fig. (2.d), a perturbation of wavenumber \( K = 3 \) produced standing waves, since \( J_0 \) was negative. The frequency of these oscillations was fast compared to that of Fig. (2.c), where the exited mode was the first one \( K = 1 \), and given that the \( J_1 \) was positive. However, note that in both cases the decay to the SHS is similar, as predicted by the eigenvalues Eq. (10). This indicates that the desynchronization process occurs faster when the diversity \( \Delta \) of neurons is increased, and this process doesn’t depend on the oscillation mode being excited. Finally, in panels (b,c) of Fig. 3 we show numerical simulations of the QIF-NFM Eq. (7) using the same parameters as those of Fig. 2 (e,d), and the agreement is good.

C. Turing bifurcation and nonlinear stability of the SHS

As \( J_K \) is increased, the frequency \( \nu_K \) of a given oscillatory mode decreases and eventually it ceases to oscillate. Further
The results, presented in Figure (4,c) confirm that the unstable state meets a stable BS —solid Blue line— at a fold bifurcation.

Moreover, we computed numerically a bifurcation diagram of the NFM, using the spectral method developed in Reference [36] and available with Reference [37]. The results, presented in Figure (4,c) confirm that the unstable BS bifurcates subcritically for the SHS. The unstable BS then meets a stable BS —solid Blue line— at a fold bifurcation.

D. Synchrony-induced transient oscillations in Bump states

To investigate whether the synchrony-induced oscillatory modes are also present in the stationary BS, we computed their spectrum. The gray points in Fig. (5,a) show the spectrum of the unstable Bump near the subcritical Turing Bifurcation of wavelength $K = 1$. Additionally, the red crosses in Fig. (5,a) are the eigenvalues of the SHS state Eq. (10). The profile of the unstable bump is only very weakly modulated, see Fig. (5,c), and hence the spectrum of the BS is very close to that of the SHS, given by the eigenvalues $\lambda_{K}$. All these eigenvalues are complex, except two real eigenvalues which correspond to the $K = 1$ mode. One of these eigenvalues is negative and the other is very close to zero and positive, indicating that the SHS is unstable.

Additionally, it is important to note that in Fig. 5 we have taken $J_K = 0$ for all $K$ except for $K = 1, 2, 3$, and hence there is an infinite number of eigenvalues ($\lambda_0$ and $\lambda_{4,5,...}$) that are all complex and identical. In Fig. (5,a) the eigenvalues of the unstable BS seem to form a continuous band precisely around these infinitely degenerated eigenvalues and their complex conjugates. These continuous bands grow in size as one moves away from the Turing bifurcation, as it can be seen in the spectrum of the stable bump depicted in Fig. (5,b) —here red crosses also correspond to the eigenvalues of the SHS state Eq. (10). These results show that all the complex eigenvalues linked to the oscillatory modes of the SHS remain complex, suggesting that, in general, similar synchronization-induced oscillations may be present in stationary, spatially inhomogeneous neural patterns.

Finally, to illustrate this, in Fig. (5,e) we performed a numerical simulation of the QIF-NFM Eqs. (7), and perturbed the BS shown in Fig.(5,d) with a spatially inhomogeneous perturbation corresponding to the mode ($K = 6$). The perturbation decays to the BS showing a pattern that resembles that of Figs. (2). However here, the regions of the ring with the maximum values of $R_s$ —around $\phi = 0$, in panels (d,e)— oscillate at high frequencies and these oscillations slow down as $\phi \to \pm \pi$. The spectrum of the stable BS Fig. (5,b) also indicates that the decay of the fast oscillations (located at the central part of the bump, $\phi = 0$) is slow compared to that of the slow oscillations.

IV. CONCLUSIONS

We have reported the existence of a class of oscillatory modes in spatially distributed networks of heterogeneous spiking neurons. These modes of oscillation reflect the transient episodes of spike synchronization among the neurons and are not captured by traditional NMFs. To investigate them, we derived a novel NFM for QIF neurons, Eqs. (6) and (7), which allows us to find the eigenvalues determining the linear stability of the spatially homogeneous state. This analysis reveals two important features: (i) The frequency of each oscillation mode only depends on the corresponding Fourier coefficient of the synaptic pattern of connectivity; (ii) The decay rate is exactly the same for all modes, and is due to a
FIG. 5. (color online) Spectrum (a,b) and firing rate profiles (d,c) of an unstable (a,c) and stable (b,d) Bump States of the QIF-NFM Eqs. (7). In panel (c) the eigenvalues Eq. (10) are superimposed with red crosses. Panel (e) shows a numerical simulation of the BS of panel (d). At \( t = 0.05 \) s, a perturbation of wavenumber \( K = 6 \) is applied. Parameters are \( J_1 = 0, J_2 = 10, J_2 = 7.5, J_2 = -2.5, J_K = 0 \) for \( K > 3, \Delta = 1, \tau = 20 \) ms. Panels (a,c): \( \bar{\eta} = 2.2120 \); Panels (b,d,e): \( \bar{\eta} = 2.1828 \).

These results (not shown). In this case the desynchronization reflects an underlying phase diffusion proportional to the noise strength. Finally we investigated the existence and stability of bump states, which bifurcate from the spatially homogeneous states via Turing bifurcations. The spectrum of such bump states has a continuous part off the real axis, indicating that similar synchronization-induced oscillatory modes may also be supported by our model. Also, recent work has been done to extend the local firing rate equations derived in [28] to include synaptic kinetics [39–42] or fixed delays [43]. These studies all show that time delays due to synaptic processing generally lead to the emergence of self-sustained oscillations due to collective synchronization. Extending the QIF-NFM (6) to account for the synaptic time delays caused by synaptic processing may lead to spatio-temporal phenomena not previously observed in traditional NFM.

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**APPENDIX A: DERIVATION OF THE QIF NEURAL FIELD MODEL (QIF-NFM)**

Our derivation closely follows that of [28], but it needs to be extended to include the spatial dimension. Similar extensions from a single population of phase oscillators to a one-dimensional, spatially distributed network with non-local coupling have been done in [46–53].

Considering the thermodynamic limit \( N \to \infty \), we can drop the indexes in Eqs. (2, 3) and define the density function \( \rho^{\varepsilon,i}(\eta^{\varepsilon,i}, t, \phi) \) such that \( \rho^{\varepsilon,i}(\eta^{\varepsilon,i}, t, \phi) dv^{\varepsilon,i} d\eta^{\varepsilon,i} d\phi \) describes the fraction of neurons located between \( \phi \) and \( \phi + d\phi \), with membrane potentials between \( v^{\varepsilon,i} \) and \( v^{\varepsilon,i} + dv^{\varepsilon,i} \), and parameters between \( \eta^{\varepsilon,i} \) and \( \eta^{\varepsilon,i} + d\eta^{\varepsilon,i} \) at time \( t \). Accordingly, parameter \( \eta^{\varepsilon,i} \) becomes now a continuous random variable with probability density function \( g(\eta^{\varepsilon,i}) \). For the sake of simplicity we assume identical distributions for both excitatory and inhibitory populations \( g(\eta^{\varepsilon,i}) = g(\eta) \). The total voltage density at location \( \phi \) and time \( t \) is given by

\[
\int_{-\infty}^{\infty} \rho^{\varepsilon,i}(\eta^{\varepsilon,i}, t, \phi) g(\eta) d\eta.
\]

Conservation of the number of neurons at each \( \phi \) value is described by the continuity equation

\[
\partial_t \rho^{\varepsilon,i} = -\partial_x \left[ \left( (x^{\varepsilon,i})^2 + \eta S(\phi, t) + P^{\varepsilon,i}(\phi, t) \right) \rho^{\varepsilon,i} \right],
\]

where we have explicitly included the velocity given by equations (2) and (3) and \( S(\phi, t) = S^e(\phi, t) + S^i(\phi, t) \) represents the total synaptic activity. Next we invoke the Ott-Antonsen theory [29], by means of the Lorentzian Ansatz (LA) [28]

\[
\rho^{\varepsilon,i}(\eta^{\varepsilon,i}, t, \phi) = \frac{1}{\pi} \frac{x^{\varepsilon,i}(\phi, \eta, t)}{[x^{\varepsilon,i}(\phi, \eta, t) + y^{\varepsilon,i}(\phi, \eta, t)]^2 + x^{\varepsilon,i}(\phi, \eta, t)^2},
\]

which solves the continuity equation. The width \( x^{\varepsilon,i}(\phi, \eta, t) \) of the LA is related to the firing rate \( R^{\varepsilon,i} \) of the neural populations. Indeed, for each \( \eta \) value at time \( t \), \( R^{\varepsilon,i}(\phi, \eta, t) \) can be evaluated noting that neurons fire at a rate given by the probability flux at infinity: \( R^{\varepsilon,i}(\phi, \eta, t) = \rho^{\varepsilon,i}(v^{\varepsilon,i} \to \infty | \eta, t, \phi) x^{\varepsilon,i}(v^{\varepsilon,i} \to \infty | \eta, t, \phi) \). The limit \( v^{\varepsilon,i} \to \infty \) on the right hand side of this equation can be evaluated within the LA, and gives: \( x^{\varepsilon,i}(\phi, \eta, t) = \pi \tau R^{\varepsilon,i}(\phi, \eta, t) \). The total firing rate at a particular location \( \phi \) of the ring is then

\[
R^{\varepsilon,i}(\phi, t) = \frac{1}{\tau \pi} \int_{-\infty}^{\infty} x^{\varepsilon,i}(\phi, \eta, t) g(\eta) d\eta. \tag{A2}
\]

Additionally, the quantity \( y^{\varepsilon,i}(\eta, t) \) is, for each value of \( \eta \), the mean of the membrane potential \( y^{\varepsilon,i}(\phi, \eta, t) = \text{P.V.} \int_{-\infty}^{\infty} x^{\varepsilon,i}(\phi, \eta, t) d\eta^{\varepsilon,i} \). Therefore, this variable is related to the mean membrane potential of the neuronal population at \( \phi \) by

\[
V^{\varepsilon,i}(\phi, t) = \int_{-\infty}^{\infty} y^{\varepsilon,i}(\phi, \eta) g(\eta) d\eta. \tag{A3}
\]

Substituting the LA (A1) into the continuity equation, we find that, for each value of \( \eta \), the variables \( x^{\varepsilon,i}(\phi) \) and \( y^{\varepsilon,i}(\phi) \) must obey two coupled equations which can be written in complex
form as
\[
\tau \partial_t w^{e,i}(\phi, \eta, t) = i \left[ \eta + \tau S(\phi, t) - (w^{e,i})^2 (\phi, \eta, t) + P^{e,i}(\phi, t) \right],
\] (A4)

where \(w^{e,i}(\phi, \eta, t) \equiv x^{e,i}(\phi, \eta, t) + iy^{e,i}(\phi, \eta, t)\). If \(\eta\) are distributed according to a Lorentzian distribution Eq. (5), the integrals in (A2) and (A3) can then be evaluated closing the integral contour in the complex \(\eta\)-plane, and using the Cauchy residue theorem. Then the firing rate and mean membrane potential depend only on the value of \(\eta\) at the pole of \(g(\eta)\) in the lower half \(\eta\)-plane: \(\pi \tau R^{e,i}(\phi, t) + iV^{e,i}(\phi, t) = w^{e,i}(\phi, \eta - i\Delta, t)\), and as a result, (A4) must be evaluated only at \(\eta = \bar{\eta} - i\Delta\) to obtain the neural field equations (Eq. (6))

\[
\tau \frac{\partial R^{e,i}}{\partial t} = \frac{\Delta}{\pi \tau} + 2R^{e,i}V^{e,i},
\]
\[
\tau \frac{\partial V^{e,i}}{\partial t} = \left( V^{e,i} \right)^2 + \bar{\eta} - (\pi \tau R^{e,i})^2 + \tau S(\phi, t) + P^{e,i}(\phi, t),
\]

where again \(S(\phi, t) = S^c(\phi, t) + S^l(\phi, t)\), and considering the limit of infinitely fast synapses, \(\tau_s \to 0\) in Eq. (4), the mean field becomes

\[
S^{e,i}(\phi, t) = \frac{1}{2\pi} \int_{-\pi}^{\pi} J^{e,i}(|\phi' - \phi|) R^{e,i}(\phi', t) d\phi',
\] (A5)

\[
s(\phi, \tilde{t}) = \frac{1}{\pi} \int_{-\pi}^{\pi} \left[ \frac{j_0}{2} + \sum_{K=1}^{\infty} j_K \cos(K(\phi' - \phi)) \right] r^{e,i}(\phi', \tilde{t}) d\phi'.
\] (A6)

**Effective NFM model**

Considering \(\tilde{P}^{e,i}(\phi, \tilde{t}) = \tilde{P}(\phi, \tilde{t})\) in Eqs.(A8), the system

\[
\dot{r} = \frac{1}{\pi} + 2vr^i,
\] (A10a)
\[
\dot{v} = v^2 + \bar{\eta} - \pi^2 r^2 + s(\phi, \tilde{t}) + \tilde{P}(\phi, \tilde{t}),
\] (A10b)

with the mean field

\[
s(\phi, t) = \frac{1}{\pi} \int_{-\pi}^{\pi} \left[ j_0 \frac{\pi}{2} + \sum_{K=1}^{\infty} j_K \cos(K(\phi' - \phi)) \right] r^{e,i}(\phi', t) d\phi'.
\] (A7)

\[
\bar{j}_K = j^r_K - j^i_K,
\]

has identical symmetric solutions as the original Eqs.(A8), i.e.

\[
r^e(t) = r^i(t) = r(t), \quad v^e(t) = v^i(t) = v(t).
\]

Equations (6) with the mean field (A5) exactly describe the macroscopic dynamics of the population of QIF neurons in terms of the local firing rates \(R^{c,i}(\phi)\) and mean membrane potentials \(V^{c,i}(\phi)\). These equations can be non-dimensionalized by rescaling variables and time as (note the difference between \(v^{c,i}_j\), the membrane potential of a single neuron \(j\), and the mean membrane potential \(v^{c,i}\)):

\[
R^{c,i} = \frac{\sqrt{\Delta}}{\tau} v^{c,i}, \quad V^{c,i} = \sqrt{\Delta} v^{c,i}, \quad t = \frac{\tau}{\sqrt{\Delta}},
\] (A8)

and parameters as:

\[
J^{c,i}_K = \sqrt{\Delta} j^{c,i}_K, \quad \bar{\eta} = \Delta \bar{\eta}, \quad P^{c,i}(\phi, t) = \Delta \tilde{P}^{c,i}(\phi, \tilde{t}).
\] (A9)

The resulting dimensionless NFM is then

\[
\dot{r}^{c,i} = \frac{1}{\pi} + 2vr^{c,i},
\] (A8a)
\[
\dot{v}^{c,i} = (v^{c,i})^2 + \bar{\eta} - \pi^2 (v^{c,i})^2 + s(\phi, \tilde{t}) + \tilde{P}^{c,i}(\phi, \tilde{t}),
\] (A8b)

where the over-dot represents derivation with respect the non-dimensional time \(\tilde{t}\), and the mean field is

\[
s(\phi, \tilde{t}) = \frac{1}{\pi} \int_{-\pi}^{\pi} \left[ j_0 \frac{\pi}{2} + \sum_{K=1}^{\infty} j_K \cos(K(\phi' - \phi)) \right] r^{c,i}(\phi', \tilde{t}) d\phi'.
\] (A9)

**APPENDIX B: LINEAR STABILITY ANALYSIS OF THE SPATIALLY HOMOGENEOUS STATE**

**Linear stability of effective QIF-NFM Eq. (7)**

The homogeneous steady state is given by the solution of Eq. (9) when \(R_\star(\phi) = R_\star\). This is equivalent to \(S_\star(\phi) = S_\star = J_0 R_\star\) that in dimensionless form is

\[
\pi^2 r_\star^4 - J_0 r_\star^3 - \bar{\eta}r_\star^2 - \frac{1}{4\pi^2} = 0,
\] (B1)

This equation is greatly simplified assuming \(J_0 = 0\), and gives

\[
r_\star = \frac{1}{\pi \sqrt{2}} \sqrt{\bar{\eta} + \sqrt{\bar{\eta}^2 + 4}}.
\] (B2)

The stability of homogeneous steady state solutions can be analyzed studying the evolution of the small (even) perturbations \((\epsilon \ll 1)\) of the SHS

\[
r(\phi, t) = r_\star + \epsilon \sum_{K=0}^{\infty} a_K(t) \cos(K\phi),
\] (B3a)
\[
v(\phi, t) = v_\star + \epsilon \sum_{K=0}^{\infty} b_K(t) \cos(K\phi).
\] (B3b)
In terms of the dimensional variables and parameters (A6, A7), the eigenvalues (B9) are \( \lambda_k t = \mu_k t \), and thus \( \lambda_k = \sqrt{\Delta} \mu_k / \tau \). In dimensional form, Eq. (B9) is

\[
\mu_{K \pm} = -\frac{1}{\pi r_s} \pm 2 \pi r_s \sqrt{\frac{J_k}{2 \pi^2 R_s}} - 1, \tag{B11}
\]

and the eigenvalues give Eq. (10) in the main text.

Substituting (B3) into the mean field (A11), we obtain a perturbed mean field around \( s_*(\phi) \)

\[
s(\phi, t) = s_*(\phi) + \epsilon \sum_{K=0}^{\infty} j_K a_K(t) \cos(K \phi). \tag{B4}
\]

Linearizing Eqs. (A10) around the fixed point \((r_*, v_*)\), gives

\[
\sum_{K=0}^{\infty} \mu_K a_K \cos(K \phi) = 2 \sum_{K=0}^{\infty} [r_*(\phi)b_K + v_*(\phi)a_K] \cos(K \phi),
\]

\[
\sum_{K=0}^{\infty} \mu_K b_K \cos(K \phi) = \sum_{K=0}^{\infty} [2v_*(\phi)b_K + (j_K - 2\pi^2 r_*(\phi))a_K] \cos(K \phi), \tag{B5}
\]

where we have used the Ansatz \( a_K(t) = a_K e^{\mu_K t} \) and \( b_K(t) = b_K e^{\mu_K t} \), where \( \mu_K \) represents the dimensionless eigenvalue of the \( K \)th mode. For SHS states, \((r_*(\phi), v_*(\phi)) = (r_*, v_*)\), the modes in Eqs. (B5) decouple and, for a given mode \( K \), we find the linear system

\[
\mu_K \begin{pmatrix} a_K \\ b_K \end{pmatrix} = L_* \begin{pmatrix} a_K \\ b_K \end{pmatrix}, \tag{B6}
\]

with:

\[
L_* = \begin{pmatrix} 2v_* \\ j_K - 2\pi^2 r_* \end{pmatrix}. \tag{B7}
\]

Equation (B6) has a general solution:

\[
\begin{pmatrix} a_K(t) \\ b_K(t) \end{pmatrix} = A_+ u_+ e^{\mu_{K+} t} + A_- u_- e^{\mu_{K-} t}, \tag{B8}
\]

where \( A_\pm \) are arbitrary constants. The eigenvalues \( \mu_{K \pm} \) are given by

\[
\mu_{K \pm} = -\frac{1}{\pi r_s} \pm 2 \pi r_s \sqrt{\frac{\sqrt{\Delta}}{2 \pi^2 R_s}} - 1, \tag{B9}
\]

with eigenvectors

\[
u_\pm = \begin{pmatrix} \pm 1 \\ \sqrt{\frac{\sqrt{\Delta}}{2 \pi^2 r_s}} \end{pmatrix}. \tag{B10}\]

Linear stability of the full QIF-NFM

For the full QIF-NFM Eq. (6), the perturbation around the SHS state has the form

\[
\begin{aligned}
\dot{r}_{\epsilon, \perp}(\phi, t) &= r_* + \epsilon \sum_{K=0}^{\infty} a_{K \epsilon}(t) \cos(K \phi), \\
\dot{v}_{\epsilon, \perp}(\phi, t) &= v_* + \epsilon \sum_{K=0}^{\infty} b_{K \epsilon}(t) \cos(K \phi).
\end{aligned} \tag{B11}
\]

In this case, the linear stability of the SHS state with respect to perturbations of the \( K \)-spatial mode is determined by the characteristic equation

\[
\lambda_K \begin{pmatrix} a_K \\ b_K \end{pmatrix} = \begin{pmatrix} 2v_* \\ j_K - 2\pi^2 r_* \end{pmatrix} \begin{pmatrix} a_K \\ b_K \end{pmatrix} = \begin{pmatrix} 2v_* \\ j_K - 2\pi^2 r_* \end{pmatrix} \begin{pmatrix} a_K \\ b_K \end{pmatrix} \tag{B12}
\]

For each \( K \) mode, the linearized system has a general solution

\[
\begin{pmatrix} a_{K \epsilon}(t) \\ b_{K \epsilon}(t) \end{pmatrix} = A_+ u_{K \epsilon} e^{\mu_{K+} t} + A_- u_{K \epsilon} e^{\mu_{K-} t} + B_+ u_{K \epsilon} e^{\mu_{K+} t} + B_- u_{K \epsilon} e^{\mu_{K-} t}, \tag{B13}\]

where \( A_\pm \) and \( B_\pm \) are arbitrary constants. The eigenvectors

\[
u_{K \pm} = \begin{pmatrix} \pm 1 \\ \sqrt{\frac{\sqrt{\Delta}}{2 \pi^2 r_s}} \end{pmatrix}. \tag{B14}\]

have eigenvalues

\[
\mu_{K \pm} = -\frac{1}{\pi r_s} \pm 2 \pi r_s \sqrt{\frac{\sqrt{\Delta}}{2 \pi^2 R_s}} - 1. \tag{B15}\]
These eigenvalues coincide with those of the reduced system (B9), and are associated with the standing waves shown in Figure 2. Additionally, the eigenvector

\[ u_{K\perp} = \begin{pmatrix} j \nu_K^- \\ j \nu_K^+ \\ j \nu_K^- \\ j \nu_K^- \end{pmatrix}, \]  

(B16)

and its complex conjugate \( \bar{u}_{K\perp} \), with associated eigenvalue

\[ \mu_{\perp} = -\frac{1}{\pi \nu \tau} + i2\pi \nu \tau. \]  

(B17)

and its complex conjugate \( \bar{\mu}_{\perp} \), correspond to modes of oscillation of the uncoupled system. Indeed, note that the eigenvalues (B17) are independent of the connectivity, and correspond to oscillatory modes which are already present in a single population of uncoupled neurons —note that eigenvalues (B15) reduce to (B17) for all the modes with \( j_K = \tilde{j}_K - j_K = 0 \).

**APPENDIX C: SMALL-AMPLITUDE EQUATION NEAR THE SPATIALLY HOMOGENEOUS STATE**

**Critical eigenvectors**

Right at the bifurcation, the only undamped mode is the critical one given by \( u_+ \) in (B16), that reduces to the critical eigenmode:

\[ u_c = \begin{pmatrix} r_+ \\ -v_+ \end{pmatrix}. \]  

(C1)

At criticality, the critical eigenmode of \( L_c \) satisfies

\[ L_c u_c = 0 \]

where \( L_{cc} \) corresponds to the operator (B7) evaluated at \( j_{K_c} \). The left critical eigenvector of the operator \( L_{cc} \) is then defined as

\[ u_c^\perp L_{cc} = 0 \]

what gives

\[ u_c^\perp = \pi \begin{pmatrix} -v_+ \\ r_+ \end{pmatrix}^T, \]  

(C2)

where the constant has been taken to normalize the eigenvectors, so that they satisfy \( u_c^\perp u_c = 1 \).

**Amplitude equation**

Except for initial transients, the amplitude of the bifurcating solution at criticality is expected to contain only the component \( u_+ \). In the following we derive a small-amplitude equation for the bump solutions using multiple-scale analysis, see e.g. [54]. First, let the solution of Eqs. (A10) be written as the perturbation expansion

\[ \begin{cases} r(\phi, \tilde{t}) \\ v(\phi, \tilde{t}) \end{cases} = \begin{cases} r_+ \\ v_+ \end{cases} + \epsilon \begin{cases} r_e(\phi, \tilde{t}, \tilde{T}) \\ v_e(\phi, \tilde{t}, \tilde{T}) \end{cases} + \epsilon^2 \begin{cases} r_{ce}(\phi, \tilde{t}, \tilde{T}) \\ v_{ce}(\phi, \tilde{t}, \tilde{T}) \end{cases} + \ldots \]  

(C3)

where \( (r_+, v_+) \) is the state SHS given by the solutions of (B1), and \( \epsilon \ll 0 \) is a small parameter, which measures the distance from the Turing bifurcation. In addition we define a long time scale \( \tilde{T} = \epsilon^2 \tilde{t} \), that is considered to be independent of \( \tilde{t} \). Accordingly, the differential operator in Eqs. (A10) may be replaced by:

\[ \partial_{\tilde{t}} \rightarrow \partial_{\tilde{t}} + \epsilon^2 \partial_{\tilde{T}}. \]

Since the asymptotic expansion is going to be performed in the vicinity of a stationary bifurcation, we set \( \partial_{\tilde{T}} = 0 \) so that the only temporal variations occur with the slow time scale \( \tilde{T} \).

Additionally, in our analysis we use the parameter \( j_1 \) as the bifurcation parameter, and we write it as

\[ j_1 = \tilde{j}_1^2 + \epsilon^2 \delta j_1, \]  

(C4)

where \( \tilde{j}_1^2 \) is the critical value of \( j_1 \) at which the Turing bifurcation occurs, given by Eq. (11), with \( K = 1 \). Accordingly, the (non-dimensionalized) connectivity footprint (1) is

\[ j(\phi) = j_c(\phi) + 2\epsilon^2 \delta j_1 \cos \phi, \]  

(C5)

with

\[ j_c(\phi) = j_0 + 2j_1^2 \cos \phi + 2 \sum_{K=2}^{\infty} j_K \cos(K \phi), \]  

(C6)

where \( j_K < j_{K_c} \) for \( K \neq 1 \). To simplify the notation, we hereafter omit to explicitly write the dependence of \( r_{e,ce}, \ldots \) and \( v_{e,ce}, \ldots \) on the variables \( \tilde{t}, \tilde{T} \) and \( \phi \). Substituting (C3) and (C5) into the mean field (A11):

\[
\begin{align*}
\frac{d}{d\tilde{t}} s(\phi) &= \frac{1}{2\pi} \int_{-\pi}^{\pi} (r_+ + \epsilon r_e + \epsilon^2 r_{ce} + \ldots) j_c(\phi - \phi')d\phi' + \epsilon^2 \frac{1}{\pi} \int_{-\pi}^{\pi} (r_+ + \epsilon r_e + \epsilon^2 r_{ce} + \ldots) \delta j_1 \cos(\phi - \phi')d\phi' \\
&\equiv \langle r_+ + \epsilon r_e + \epsilon^2 r_{ce} + \ldots \rangle_c + 2\epsilon^2 \langle r_+ + \epsilon r_e + \epsilon^2 r_{ce} + \ldots \rangle_c \\
&= r_0 + \epsilon (r_e)_c + \epsilon^2 (r_{ce})_c + \epsilon^2 (r_{ce})_c \cos \phi + \ldots
\end{align*}
\]  

(C7)

\[
\begin{align*}
\frac{d}{d\tilde{t}} v(\phi, \tilde{t}) &= -\frac{1}{\pi \nu \tau} \int_{-\pi}^{\pi} (r_+ + \epsilon r_e + \epsilon^2 r_{ce} + \ldots) \delta j_1 \cos(\phi - \phi')d\phi' \\
&= r_0 + \epsilon (r_e)_c + \epsilon^2 (r_{ce})_c + \epsilon^2 (r_{ce})_c \cos \phi + \ldots
\end{align*}
\]  

(C8)
Plugging expansions (C3) and (C5) into the NFM Eqs. (A10), we obtain
\[ \epsilon^2 \partial_t (\epsilon r_e + \epsilon^2 r_{ee} + \ldots) = \epsilon(2v_s r_e + 2r_s v_e) + \epsilon^2(2v_s r_{ee} + 2r_s v_e + 2r_s v_{ee}) + \epsilon^3(2v_s r_{ee} + 2r_s v_{ee}) + \ldots. \]
and
\[ \epsilon^2 \partial_t (\epsilon v_e + \epsilon^2 v_{ee} + \ldots) = \epsilon(v_s v_e - 2\pi^2 r_s r_e + \langle \epsilon \rangle_c) + \epsilon^2(v_s^2 - \pi^2 v_e^2 + 2v_s v_e - 2\pi^2 r_s r_{ee} + \langle r_{ee} \rangle_c) + \epsilon^3(2v_s v_{ee} - 2\pi^2 r_s r_{ee} + \langle r_{ee} \rangle_c) + \ldots. \]

These equations can be written in a more compact form as
\[ -(L_c + \epsilon^2 L_{ee}) \begin{bmatrix} r_e \\ v_e \end{bmatrix} + \epsilon^2 \begin{bmatrix} r_{ee} \\ v_{ee} \end{bmatrix} = 0, \tag{C9} \]
defining the linear and nonlinear operators
\[ L_c = \begin{pmatrix} 2v_s & 2r_s \\ 2v_s - 2\pi^2 r_s & 2v_s \end{pmatrix}, \]
\[ L_{ee} = \begin{pmatrix} -\partial_r & 0 \\ 0 & -\partial_r \end{pmatrix}, \]
\[ N_{ee} = \begin{pmatrix} 2r_s v_e \\ v_e^2 - \pi^2 v_e^2 \end{pmatrix}, \]
\[ N_{ee} = \begin{pmatrix} 2r_s v_e + 2r_s v_{ee} \\ v_e v_{ee} - 2\pi^2 r_s r_{ee} \end{pmatrix}. \]

Next we collect terms by order in \( \epsilon \). At first order we recover the linear problem (B6) at the Turing bifurcation:
\[ \begin{pmatrix} \partial_t & \partial_r & 2v_s \\ \partial^2 + 2\pi^2 r_s & 2v_s \end{pmatrix} \begin{bmatrix} r_e \\ v_e \end{bmatrix} = 0. \]
Recalling that \( j_1^T \) is given by Eq. (C4), we find the neutral solution:
\[ \begin{bmatrix} r_e \\ v_e \end{bmatrix} = A u_c \cos \phi, \tag{C10} \]
where \( A \) is the small amplitude with slow time dependence that we aim to determine, and \( u_c \) is the critical eigenmode given by Eq. (C1). Substituting the solution \( r_{ee} = A u_c \cos \phi \) into the nonlinear forcing terms \( N_{ee} \) we find
\[ N_{ee} = \frac{A^2}{2} \begin{pmatrix} \pi^{-1} v_e^2 - \pi^2 r_s^2 \end{pmatrix} \left[ 1 + \cos(2\phi) \right], \]
which implies that, at second order, the solution must necessarily contain homogeneous and second spatial components
\[ \begin{bmatrix} r_{ee} \\ v_{ee} \end{bmatrix} = \begin{bmatrix} r_{ee0} \\ v_{ee0} \end{bmatrix} + \begin{bmatrix} r_{ee2} \\ v_{ee2} \end{bmatrix} \cos(2\phi). \]
Equating the homogeneous, second order terms of equation (C9) we find
\[ \begin{pmatrix} 2v_s \\ 0 \end{pmatrix} \begin{bmatrix} \partial_t & \partial_r & 2v_s \\ \partial^2 + 2\pi^2 r_s & 2v_s \end{pmatrix} \begin{bmatrix} r_{ee0} \\ v_{ee0} \end{bmatrix} = \frac{A^2}{2} \begin{pmatrix} \pi^{-1} v_e^2 \\ v_e^2 - \pi^2 r_s^2 \end{pmatrix} \],
\[ N_{ee} = -A \cos \phi \begin{pmatrix} v_e (2r_{ee0} + r_{ee2}) \\ \pi^2 r_s (2r_{ee0} + r_{ee2}) + v_e (2v_{ee0} + v_{ee2}) \end{pmatrix} - A \cos(3\phi) \begin{pmatrix} v_e r_{ee2} - r_s v_{ee2} \\ \pi^2 r_s r_{ee2} + v_e v_{ee2} \end{pmatrix}. \]
Thus, the solvability condition gives
\[ u_e \left( r_e \phi \frac{\partial}{\partial r} A - \delta j_1 r_e A \right) = -A u_e \left( \frac{v_e (2r_ee + r_e) - r_e (2v_ee + v_e)}{\pi^2 r_e (2r_ee + r_e) + v_e (2v_ee + v_e)} \right), \] (C16)
Substituting the coefficients (C11, C12, C13, C14) into Eq. (C16) gives the desired amplitude equation
\[ \partial_r A = \pi r_e^2 \delta j_1 A + \tilde{a} A^3, \] (C17)
where the parameter \( \tilde{a} \) is
\[ \tilde{a} = \pi \left( 5v_e^4 + \pi^4 r_e^4 - \frac{5}{2} \right) \left( \frac{1}{j_1 - j_0} + \frac{1/2}{j_1 - j_2} \right) - v_e \left( \frac{j_0}{j_1 - j_0} + \frac{j_2/2}{j_1 - j_2} \right), \] (C18)
Equating Eq. (C18) to zero, gives the critical boundary \( j_2^c \) separating sub-critical and super-critical Turing bifurcations:
\[ j_2^c = \frac{3j_1^c - j_0}{2} + \frac{6(j_1^c - j_0)^2 \pi^2 r_e^3}{5 + 4\pi^2 r_e^3 (3j_0 - j_1^c - 10\pi^2 r_e^3 + 4\pi^6 r_e^5)} \] (C19)
In dimensional form, Eqs. (C17, C18, C19) are respectively:
\[ \tau \partial_r A = \frac{\tau^2 R_e^2}{\Delta} \delta J_1 A + a A^3, \] (C20)
\[ a = \left[ \pi \left( \frac{5\Delta^3}{16\pi^4 r_e^4 R_e^4} + \frac{\pi^4 r_e^4}{\Delta} \frac{\Delta}{2} \right) \left( \frac{1}{j_1^c - j_0} + \frac{1/2}{j_1^c - j_2} \right) + \frac{\Delta}{2\pi \tau R_e} \left( \frac{J_0}{j_1^c - j_0} + \frac{j_2/2}{j_1^c - j_2} \right) \right], \] (C21)
and
\[ J_2^c = \frac{3j_1^c - j_0}{2} + \frac{6(j_1^c - j_0)^2 \pi^2 r_e^3}{5 + 4\pi^2 r_e^3 \left( 3j_0 - j_1^c - 10\pi^2 r_e^3 + \frac{4\pi^6 r_e^5}{\Delta} \right)} \] (C22)

**APPENDIX D: NUMERICAL SIMULATIONS**

**Numerical simulation of the QIF model**

In numerical simulations we used the Euler scheme with time step \( dt = 10^{-3} \). Additionally, we considered the peak and reset values \( v_p = -v_r = 100 \). The Algorithm used to simulate the QIF neuron (2) is shown in Fig. IV.

To numerically implement the ring network of QIF neurons we divided the ring into \( n = 100 \) intervals located at \( \phi_m = 2\pi m/n - \pi, m = 1, \ldots, n \). At each interval \( \phi_m \), we considered \( N^c_m = N/(2n) = 2.5 \cdot 10^3 \) excitatory and \( N^i_m = 2.5 \cdot 10^3 \) inhibitory neurons.

Then we distributed the neurons in each interval \( \phi_m \) using a Lorentzian distribution Eq. (5). For each \( \phi_m \) we used the inverse cumulative distribution function (quantile function):
\[ \eta_i = \eta + \Delta \tan \left( \frac{\pi 2i - N_m - 1}{N_m + 1} \right), i = 1, \ldots, N_m. \] (D1)

Perturbations were implemented such that, at a certain time \( t_0 = 0.05 \) s, a spatially modulated pulse was applied with the form:
\[ P^{ \phi \tau} (\phi, t) = A \left( e^{(t - t_0)/\tau_r} - 1 \right) \cdot \cos (K \cdot \phi), \] (D2)
where the amplitude was \( A = 0.3 \), \( K \) represents the wavenumber of the perturbation and \( \tau_r = 4 \times 10^{-3} \) s is the rising time constant of the perturbation. The perturbations had a duration of \( \Delta t = 0.01 \) s.

The instantaneous firing rates in Fig. 2 are obtained binning time and counting the spikes of neurons in each interval \( \phi_m \) within a sliding time window of size \( \delta t = 0.01 \) s (in dimensionless time, \( \delta t = 0.5 \)).
**Fig. 6.** Algorithm used for the Euler integration of the QIF neuron Eq. (2).