Appendix

A Proof ot Theorem 1

A mathematically rigorous exposition of Theorem 1, based on Theorem 1 in (Izhikevich, 1999) and building on the Assumptions (i)–(v) in the main text, reads:

Theorem 1. *Consider a weakly connected neural network of the form*

$$\dot{X}_j = F_j(X_j, \lambda) + \varepsilon G_j(X_1, \dots, X_N; \lambda, \rho, \varepsilon), \quad X_j \in \mathbb{R}^m, \quad j = 1, \dots, N. \tag{A1}$$

The functions $F_j : \mathbb{R}^m \times \Lambda \to \mathbb{R}^m$ describe the individual dynamics of each neuron $j = 1, \dots, N$, where $\lambda \in \Lambda$ summarizes system (control) parameters and is an element of some Banach space Λ, typically $\Lambda = \mathbb{R}^l$ for some $l > 0$. The functions $G_j : \mathbb{R}^m \times \cdots \times \mathbb{R}^m \times \Lambda \times \mathbb{R}^n \times \mathbb{R} \to \mathbb{R}^m$ describe weak perturbations of order $\mathcal{O}(\varepsilon)$ for small $|\varepsilon| \ll 1$, which are due to recurrent coupling through the other neurons or due to external inputs $\rho = \rho_0 + \mathcal{O}(\varepsilon) \in \mathbb{R}^n$ for some $n > 0$. We assume that each (uncoupled) subsystem $\dot{X}_j = F_j(X_j, \lambda)$ undergoes a SNIC bifurcation for some $\lambda = \lambda_0$, that each function G_j has the pair-wise connected form

$$G_j(X_1, \dots, X_N; \lambda_0, \rho_0, 0) = \sum_{k=1}^{N} G_{jk}(X_j, X_k) \tag{A2}$$

and each $G_{jk}(X_j, X_k) = 0$ for X_k from some open neighborhood of the saddle-node bifurcation point. Moreover, we assume that in the uncoupled system, $\varepsilon = 0$, the phase space of each $X_j \in \mathbb{R}^m$, $j = 1, \dots, N$, has an attractive normally hyperbolic compact invariant manifold $M_j \subset \mathbb{R}^m$ that is homeomorphic to $\mathbb{S}^1$.

Then, there is $\varepsilon_0 > 0$ such that for all $\varepsilon < \varepsilon_0$ and all $\lambda = \lambda_0 + \mathcal{O}(\varepsilon^2)$, there is a piece-wise continuous transformation that maps solutions of (A1) to those of the canonical network model

$$\theta_j' = (1 - \cos\theta) + (1 + \cos\theta_j) c_j(\theta_1, \dots, \theta_N; \varepsilon) + R_j(\theta_j, \varepsilon), \quad j = 1, \dots, N, \tag{A3}$$

where $\theta_j \in \mathbb{S}^1$ are phase variables, $' = d/d\tau$ and $\tau = \varepsilon t$ is slow time. The c_j are connection functions and R_j small remainders, see Eqs. (A7) and (A8) below.

The canonical model (A3) of the weakly coupled neural network (A1) can be approximated by

$$\theta_j' = (1 - \cos\theta_j) + (1 + \cos\theta_j)\Big[\eta_j + \sum_{k=1}^{N} p_{jk}(\theta_k)\Big] \tag{A4}$$

with constants $\eta_j \in \mathbb{R}$, and the functions $p_{jk}(\theta_k)$ are given by Eq. (A15) and describe smoothed δ-pulses of strength $s_{jk} = \mathcal{O}(\|G_{jk}\|)$, i.e. s_{jk} is proportional to the amplitude of the coupling in (A1); here, $\|.\|$ is the supremum norm. The duration of the emitted pulses is short as the p_{jk} satisfy $p_{jk}(\theta_k) = 0$ if $|\theta_k - \pi| > 2\sqrt{\varepsilon}$ for all $j, k = 1, \dots, N$, see Fig. 2, which can, but need not, justify their approximation by discontinuos Dirac δ-pulses at the time of a neuron's spike.

Proof.
Step 1: Invariant manifold reduction— Each uncoupled subsystem $\dot{X}_j = F_j(X, \lambda)$ has an attractive normally hyperbolic compact invariant manifold M_j. Hence, the direct product $M = M_1 \times \cdots \times M_N$ is also normally hyperbolic. Fenichel's theorem (Theorem 4.1 in Hoppensteadt and Izhikevich, 1997) states that the network dynamics (A1) then persists to have an attractive normally hyperbolic compact invariant manifold $M_\varepsilon \subset \mathbb{R}^m \times \cdots \times \mathbb{R}^m$ for sufficiently small coupling strengths $\varepsilon \ll 1$. The persistence of normally hyperbolic manifolds further ensures that there is

an open neighborhood W of both M and M_ε as well as a continuous function $h_\varepsilon\colon W \to M$ that maps solutions of (A1) to those of the local model

$$\dot{X}_j = F_j(X_j, \lambda) + \varepsilon \tilde{G}_j(X_1, \ldots, X_N; \lambda, \rho, \varepsilon), \quad X_j \in M_j, \tag{A5}$$

see (Theorems 4.3 and 4.7 in Hoppensteadt and Izhikevich, 1997).

For the X_j are $\mathcal{O}(\varepsilon^2)$-close to a SNIC bifurcation, the invariant manifolds M_j are homeomorphic to $\mathbb{S}^1$, which allows one to rewrite (A5) as

$$\dot{x}_j = f_j(x_j, \lambda) + \varepsilon g_j(x_1, \ldots, x_N; \lambda, \rho, \varepsilon), \quad x_j \in \mathbb{S}^1, \tag{A6}$$

where the f_j correspond to F_j. Due to the pair-wise connections in the ε-perturbed system (A5), one can construct the mappings from the attractive normally hyperbolic compact invariant manifolds $M_{j,\varepsilon}$ corresponding to the ε-perturbations onto the (unperturbed) manifolds M_j such that the g_j share the pair-wise connected form of the G_j, i.e.

$$g_j(x_1, \ldots, x_n; \lambda_0, \rho_0, 0) = \sum_{k=1}^{N} g_{jk}(x_j, x_k).$$

The construction of such mappings that restrict the dynamics of (A5) onto $M = M_1 \times \cdots \times M_N$ ensures that the $g_{jk}(x_j, x_k) = 0$ for x_k from a small, open neighborhood of the saddle-node bifurcation point. A more rigorous proof of the properties of the g_j follows along the lines of the proof of, and the comment after, Theorem 4.7 as well as the lines of the theorem and the remarks in Section 5.2.1 of Hoppensteadt and Izhikevich (1997).

Without loss of generality, we now assume that the saddle-node bifurcation on the limit cycle occurs at $x_j = 0$, $j = 1, \ldots, N$, when $\lambda = 0$. Thus,

$$f_j(0,0) = 0, \quad \frac{\partial f_j(0,0)}{\partial x_j} = 0, \quad \frac{\partial^2 f_j(0,0)}{\partial x_j^2} > 0 \quad \text{for all } j = 1, \ldots, N.$$

Moreover, we introduce the parameters λ_2 and ρ_0 as

$$\lambda = \varepsilon^2 \lambda_2 + \mathcal{O}(\varepsilon^3) \quad \text{and} \quad \rho = \rho(\varepsilon) = \rho_0 + \varepsilon \rho_1 + \mathcal{O}(\varepsilon^2).$$

Finally, we assume that $g_{jk}(x_j, x_k) = 0$ for $|x_k| < \sqrt{\varepsilon}/p_k$ for some constants p_k to be defined below.

Step 2: Reduction of the canoncical model— Equation (A3) can be obtained from Eq. (A6) as in (Theorem 8.11 in Hoppensteadt and Izhikevich, 1997). The connection functions obey

$$c_j(\theta_1, \ldots, \theta_N; \varepsilon) = \begin{cases} \eta_j, & \text{if no neuron is firing a spike,} \\ & \text{i.e. } \forall k \in \{1, \ldots, N\}: \quad |\theta_k - \pi| > 2\sqrt{\varepsilon}, \\ \mathcal{O}(1/\varepsilon), & \text{if at least one neuron is firing a spike,} \\ & \text{i.e. } \exists k \in \{1, \ldots, N\}: \quad |\theta_k - \pi| \leq 2\sqrt{\varepsilon}. \end{cases} \tag{A7}$$

The first case follows from the local canonical model for multiple saddle-node bifurcations in weakly connected neural networks (cf. Theorem 5.4 in Hoppensteadt and Izhikevich, 1997) when noticing that $\partial_{x_k} g_j(0, \ldots, 0; 0, \rho_0, 0) = 0$ because $g_{j,k}(x_j, x_k)$ vanishes in an open neighborhood of $|x_k| = 0$; the constants η_j are defined below. The second case follows analogously to the proof of (Theorem 8.8 in Hoppensteadt and Izhikevich, 1997) when replacing $\sqrt[4]{\varepsilon}$ by $\sqrt{\varepsilon}$, see also below. Moreover, it follows from the VCON Lemma 8.2 with $\delta = \sqrt{\varepsilon}$ in (Hoppensteadt and Izhikevich, 1997) that the remainders satisfy

$$R_j(\theta_j, \varepsilon) = \begin{cases} \mathcal{O}(\sqrt{\varepsilon}), & \text{if no neuron is firing a spike,} \\ & \text{i.e. } \forall k \in \{1, \ldots, N\}: \quad |\theta_k - \pi| > 2\sqrt{\varepsilon}, \\ \mathcal{O}(\sqrt{\varepsilon} \log \varepsilon), & \text{if at least one neuron is firing a spike,} \\ & \text{i.e. } \exists k \in \{1, \ldots, N\}: \quad |\theta_k - \pi| \leq 2\sqrt{\varepsilon}. \end{cases} \tag{A8}$$

Step 3: Approximation of the canoncical model— We now follow the proof of (Proposition 8.12 in Hoppensteadt and Izhikevich, 1997), expand (A6) as a Taylor series and consider the first parts,

$$\dot{x}_j = p_j x_j^2 + \varepsilon^2 p_j \eta_j + \varepsilon \sum_{k=1}^{N} g_{jk}(x_j, x_k), \tag{A9}$$

where

$$p_j = \frac{1}{2}\frac{\partial^2 f_j(0,0)}{\partial x_j^2} \quad \text{and} \quad \eta_j = \big[D_\lambda f_j(0,0)\cdot\lambda_2 + D_\rho g_j \cdot \rho_1 + \partial_\varepsilon g_j\big]/p_j;$$

here, $D_\lambda f_j$ denotes the component-wise partial derivatives of f_j with respect to the (possibly multidimensional) bifurcation parameter $\lambda \in \Lambda$. Likewise, $D_\rho g_j$ denotes the component-wise partial derivatives of g_j with respect to an external input ρ and $\partial_\varepsilon g_j$ is the partial derivative of g_j with respect to ε, both terms are evaluated at $(x_1,\dots,x_N;\lambda,\rho,\varepsilon) = (0,\dots,0;\lambda_0,\rho_0,0)$. If neuron j does not fire, i.e. $|x_j| < \sqrt{\varepsilon}/p_j$, we can use the change of variables

$$x_j = \frac{\varepsilon}{p_j}\tan(\theta_j/2), \tag{A10}$$

and transform (A9) to

$$\dot{\theta}_j = \varepsilon\big[(1-\cos\theta_j) + (1+\cos\theta_j)\eta_j\big] + (1+\cos\theta_j)p_j\sum_{k=1}^{N} g_{jk}(x_j,x_k)\ . \tag{A11}$$

If the other neurons do not fire, either, then $|x_j| < \sqrt{\varepsilon}/p_j$ for all $j = 1,\dots,N$, the last sum vanishes and neuron j evolves on the slow time scale $\tau = \varepsilon t$ according to

$$\theta_j'(\tau) = (1-\cos\theta_j) + (1+\cos\theta_j)\eta_j\ . \tag{A12}$$

We now determine the effect of an incoming spike. Say, neuron $k \neq j$ starts to fire a spike at time $t = 0$, that is, $x_k(0) = \sqrt{\varepsilon}/p_k$. Neglecting the terms of order ε in (A11) yields

$$\dot{\theta}_j = (1+\cos\theta_j)p_j g_{jk}\big(x_j(t), x_k(t)\big)\ , \tag{A13}$$

which we must integrate from initial condition $\theta_j(0) = \theta_j^{\text{old}}$ until the end of the spike at time t^* when $x_k(t^*) = -\sqrt{\varepsilon}/p_k$. In other words, the incoming pulse $p_{jk}(t)$ through the spiking of neuron k is evolving on the slow time scale as

$$p_{jk}(\tau) = \frac{p_j}{\varepsilon} g_{jk}\left(\frac{\varepsilon}{p_j}\tan\left(\frac{\theta_j(\tau/\varepsilon)}{2}\right), \frac{\varepsilon}{p_k}\tan\left(\frac{\theta_k(\tau/\varepsilon)}{2}\right)\right) \tag{A14}$$

for $\tau \in (0, t^*/\varepsilon)$; note that $p_{jk}(\tau) = \mathcal{O}(1/\varepsilon)$ as already indicated by Eq. (A3) with the connection functions c_j given by (A7). As we assumed that neuron j does not spike, we can approximate its phase at first order to be constant $\theta_j(t) \approx \theta_j^{\text{old}}$, so that the pulse function (A14) only depends on the phase of neuron k,

$$p_{jk}(\theta_k) := \frac{p_j}{\varepsilon} g_{jk}\left(\varepsilon\tan\left(\theta_j^{\text{old}}/2\right)/p_j, \varepsilon\tan\left(\theta_k/2\right)/p_k\right). \tag{A15}$$

Together, this yields the slow dynamics of the weakly pulse-coupled canonical network (A4). Moreover, as $g_{jk}(x_j,x_k) \neq 0$ only if $|x_k| > \sqrt{\varepsilon}/p_k$, one can use Eq. (A10) and Taylor expand $1/\tan(x)$ about $x = \pi/2$ to find

$$|x_k| > \sqrt{\varepsilon}/p_k \quad \Longleftrightarrow \quad \sqrt{\varepsilon} > \frac{1}{|\tan(\theta_k/2)|} \approx \frac{|\theta_k - \pi|}{2} \quad \text{if} \quad |\theta_k - \pi| \ll 1,$$

such that $p_{jk}(\theta_k) \neq 0$ only if $|\theta_k - \pi| < 2\sqrt{\varepsilon}$ and it is zero otherwise.

Step 4: Pulse-interpretation as smoothed δ-pulses— Since $p_{jk}(\theta_k)$ as defined by Eq. (A15) is a smooth function of θ_k and nonzero only in a small neighborhood of π, see Fig. 2, one can naturally identify $p_{jk}(\theta_k)$ as a smoothed δ-pulse.

Another, mathematically elegant way to see this was proposed by Izhikevich and Hoppensteadt, who computed the phase shift corresponding to an incoming spike of neuron k by solving Eq. (A13) through separation of variables, see (Proposition 8.12 in Hoppensteadt and Izhikevich, 1997): The new phase θ_j^{new} after the end of the spike can be approximated as

$$\theta_j^{\text{new}} = 2\arctan\left(\tan\frac{\theta_j^{\text{old}}}{2} + s_{jk}\right) - \theta_j^{\text{old}} \tag{A16}$$

with

$$s_{jk} = \frac{1}{2}\frac{\partial^2 f_j(0,0)}{\partial x_j^2}\int_{\mathbb{S}^1}\frac{g_{jk}(x_j^{\text{old}}, x_k)}{f_k(x_k,0)}dx_k \ ; \tag{A17}$$

the s_{jk} are then proportional to $\|g_{jk}\|$ and thus correspond to the amplitude of the coupling functions G_{jk}. This leads to the approximate network model

$$\theta_j' = (1-\cos\theta) + (1+\cos\theta_j)\eta_j + \sum_{k=1}^{N} w_{jk}(\theta_j)\delta(\theta_k - \pi) \tag{A18}$$

where δ is the Dirac δ-function—justified by the short duration of an incoming spike, lasting $4\sqrt{\varepsilon}$ units of slow time—and each function w_{jk} has the form

$$w_{jk}(\theta_j) = 2\arctan\left(\tan\frac{\theta_j}{2} + s_{jk}\right) - \theta_j \tag{A19}$$

For small $|s_{jk}|$, the function w_{jk} can be Taylor expanded as $w_{jk}(\theta_j) = (1+\cos\theta_j)s_{jk} + \mathcal{O}(|s_{jk}|^2)$, which yields the Dirac δ pulse-coupled model

$$\theta_j' = (1-\cos\theta_j) + (1+\cos\theta_j)\Big[\eta_j + \sum_{k=1}^{N} s_{jk}\delta(\theta_k - \pi)\Big] \tag{A20}$$

as an approximation of Eq. (A18) neglecting higher-order coupling terms.

The canonical model (A18) with the additional remainder $\mathcal{O}(\sqrt{\varepsilon}\log\varepsilon)$ as in (A8) was first presented as Theorem 1 in (Izhikevich, 1999), yet without a proof. As the remainder smoothes the δ-pulse, this motivates the notion that the pulses $p_{jk}(\theta_k)$ are indeed smoothed Dirac δ-pulses. □

Remark 4. The assumption of attractive normally hyperbolic compact invariant manifolds M_j of the uncoupled subsystems X_j is typically difficult to see, or to guarantee, in high-dimensional systems, but it is crucial to a rigorous derivation of the canonical weakly pulse-coupled network (A4) of θ-neurons.

Remark 5. In order to identify the parameters of the reduced system (A4) from high-dimensional dynamics (A1) of weakly coupled Class 1 excitable systems close to SNIC bifurcations, a bottleneck is the construction of the mapping from the ε-perturbation M_ε onto the normally hyperbolic compact invariant manifold M.

Specifically, Remarks 4 and 5 mean that even for a concrete example of weakly coupled, Class 1 excitable, conductance-based Hodgkin-Huxley-like neurons, it can be laborious—if at all possible—to reduce the system to a network of pulse-coupled θ-neurons. First, one has to

prove that the dynamics of each uncoupled Hodgkin-Huxley-like neuron evolves on an attractive normally hyperbolic compact invariant manifold that is homeomorphic to $\mathbb{S}^1$. Second, if these normally hyperbolic manifolds exists, it is another challenge to construct the mapping from the ε-perturbed original dynamics to the dynamics on these manifolds, which at the same time ought to respect the pair-wise coupling structure as in (A2). Such a reduction goes beyond the scope of the manuscript. The current manuscript rather analyzes general principles of how the shape of putatively reduced pulse-functions $p_{jk}(\theta_k)$ affect the synchronization properties and collective dynamics of pulse-coupled θ-neurons.

B Conductance-based Wang-Buzsáki neuron model

The Wang-Buzsáki model is a conductance-based Hodgkin-Huxley-like neuron model first presented in (Wang and Buzsáki, 1996), according to which an interneuron is described by a single compartment and obeys the current balance equation

$$C\frac{dv}{dt} = -g_{Na}m_\infty^3(v)h(v-E_{Na}) - g_K n^4(v-E_K) - g_L(v-E_L) + I_0 + I_{\text{app}}, \tag{B1a}$$

where $C = 1\ \mu\text{F/cm}^2$ is the capacitance, $g_L = 0.1\text{mS/cm}^2$ the leak conductance, $E_L = -65\text{mV}$, and the passive time constant is $\tau = C/g_l = 10\text{ms}$. The spike-generating Na^+ and K^+ voltage-dependent ion currents are of Hodgkin-Huxley type with activation and inactivation variables that are governed by the equations:

$$\dot{h} = \phi\big[\alpha_h(v)(1-h) - \beta_h(v)h\big], \tag{B1b}$$
$$\dot{n} = \phi\big[\alpha_n(v)(1-n) - \beta_n(v)n\big], \tag{B1c}$$

with the following functions

$$m_\infty(v) = \alpha_m(v)/\big[\alpha_m(v)+\beta_m(v)\big], \tag{B1d}$$
$$\alpha_m(v) = 0.1(v+35)/\{1-0.1\exp[-(v+35)]\}, \tag{B1e}$$
$$\beta_m(v) = 4\exp[-(v+60)/18], \tag{B1f}$$
$$\alpha_h(v) = 0.07\exp[-(v+58)/20], \tag{B1g}$$
$$\beta_h(v) = 1/\{1+\exp[-0.1(v+28)]\}, \tag{B1h}$$
$$\alpha_n(v) = 0.01(v+34)/\{1-\exp[-0.1(v+34)]\}, \tag{B1i}$$
$$\beta_n(v) = 0.125\exp[-(v+44)/80]. \tag{B1j}$$

The standard parameters are the following (Wang and Buzsáki, 1996):

$$\begin{gathered} C = 1.0\ \mu\text{F/cm}^2,\ (g_{Na}, g_K, g_L) = (35, 9, 0.1)\text{mS/cm}^2, \\ (E_{Na}, E_K, E_L) = (55, -90, -65)\text{mV},\ \phi = 5. \end{gathered} \tag{B2}$$

For a direct current $I_0 + I_{\text{app}} = I_{\text{SNIC}} \approx 0.1601\ \mu\text{A/cm}^2$, the Wang-Buzsáki neuron undergoes a SNIC bifurcation and starts firing with arbitrarily low firing rate for applied currents larger than I_{SNIC}. In line with (Devalle et al., 2017), I set $I_0 = I_{\text{SNIC}}$ and $I_{\text{app}} = 0.5\ \mu\text{A/cm}^2$ to obtain the black voltage trace with a narrow action potential in Fig. 3.

By updating some of the parameters, especially slowing down the spike generating currents, I obtained the red voltage trace with a broader action potential in Fig. 3 with the updated values:

$$g_L \mapsto 0.1g_L,\ g_{Na} \mapsto 0.28g_{Na},\ g_K \mapsto 0.38g_K,\ \phi \mapsto \phi/3,\ I_{\text{app}} \mapsto 1.35I_{\text{app}}. \tag{B3}$$

C Derivation of Eq. (15) from a QIF model with biophysical parameters

In a network of globally coupled, quadratic integrate-and-fire (QIF) neurons $j = 1, \dots N$, the membrane potential v_j follows the subthreshold dynamics according to (Latham et al., 2000; Izhikevich, 2007):

$$C\frac{dv_j}{dt} = g_L\frac{(v - v_{\text{rest}})(v - v_{\text{thres}})}{v_{\text{thres}} - v_{\text{rest}}} + \tilde{I}_j + \tilde{I}_{\text{syn}} + g(\tilde{I}_{\text{gap}} - v_j) \tag{C1}$$

with membrane time constant $\tau_m = C/g_L$ as the product of capacitance C and membrane resistance $1/g_L$, and resting (v_{rest}) and threshold (v_{thres}) voltages. The terms $\tilde{I}_j$, $\tilde{I}_{\text{syn}}$ and $\tilde{I}_{\text{gap}}$ denote individual external inputs as well as common recurrent synaptic input due to all-to-all chemical and electrical coupling, respectively. Neuron j elicits a spike once it reaches the apex potential v_p and is subsequently reset to v_r. Introducing $\tau = \tau_m(v_{\text{thres}} - v_{\text{rest}})$ and $\tilde{g} = g(v_{\text{thres}} - v_{\text{rest}})/g_L$, one has

$$\begin{aligned}\tau\dot{v}_j &= v_j^2 - (v_{\text{rest}} + v_{\text{thres}} + \tilde{g})v_j + v_{\text{rest}}v_{\text{thres}} + (\tilde{I}_j + \tilde{I}_{\text{syn}} + g\tilde{I}_{\text{syn}})(v_{\text{thres}} - v_{\text{rest}})/g_L \\ &=: v_j^2 - \alpha v_j + I_j(t)\end{aligned} \tag{C2}$$

where the input current $I_j(t)$ comprises quenched and noisy individual inputs as well as common recurrent synaptic inputs due to all-to-all connectivity:

$$I_j(t) = I_0 + \gamma\big[c\eta_j + (1-c)\xi_j(t)\big] + I_{\text{syn}}(t) \tag{C3}$$

with some common input I_0, independent normalized Cauchy white noise $\xi_j \neq \xi_k$ of strength $(1-c)\gamma \geq 0$ and Cauchy-Lorentz distributed time-independent inputs η_j of strength $c\gamma \geq 0$, where $c \in [0,1]$. The effect of quenched heterogeneity and Cauchy noise is identical on the collective dynamics (Pietras et al., 2023; Clusella and Montbrió, 2022), that is, the parameter $c \in [0,1]$ becomes redundant on the macroscopic level. In other words, the parameter $\gamma > 0$ captures the combined effect of both noise and heterogeneity. Note, however, that there are important differences on the individual neuron level for different choices of c (Clusella and Montbrió, 2022). Furthermore, for constant α one shifts $v_j \mapsto v_j - \alpha/2$ and updates $I_0 \mapsto I_0 - \alpha^2/4$ to obtain the QIF dynamics (15) of the main text,

$$\tau_m\dot{v}_j = v_j^2 + I_0 + I_{\text{syn}}(t) + \gamma\big[c\eta_j + (1-c)\xi_j(t)\big] \tag{C4}$$

with a slight abuse of notation of the time constant τ_m for consistency. As the quadratic term in (C4) causes the voltage to diverge in finite time, one can consider apex and reset values at infinity, that is, $v_p = -v_r = \infty$. In this case, the QIF neuron is equivalent to the θ-model (Ermentrout, 1996). With the transformation $v_j = \tan(\theta_j/2)$, neuron j spikes when its phase θ_j crosses through $\theta = \pi$ with positive speed. The phase dynamics corresponding to Eq. (C4) read (in Stratonovich interpretation of the white noise)

$$\tau_m\dot{\theta}_j = 1 - \cos\theta_j + (1+\cos\theta_j)\big[I_0 + I_{\text{syn}} + \gamma\big(c\eta_j + (1-c)\xi_j(t)\big)\big]\,. \tag{C5}$$

While the QIF dynamics (C4) represents a discontinuous dynamical system that may encompass real conceptual and mathematical difficulties due to the instantaneous reset whenever a neurons crosses the threshold at infinity (Cessac and Viéville, 2008; Kevrekidis et al., 2017), it can sometimes be advantageous to consider the θ-model dynamics (C5), which circumvents the fire-and-reset discontinuity and can thus be treated within the theory of continuous dynamical systems.

D Firing rate and voltage equations

The goal is to provide an exact low-dimensional description of the collective dynamics of the QIF neurons (15). In the thermodynamic limit, i.e. for infinitely many neurons $N \to \infty$, the collective state is described by the probability density, either $\mathcal{W}(v,t)$ in voltage space, or $\mathcal{P}(\theta,t)$ in the θ-phase description. The change from one density to the other can be achieved via $\mathcal{W}(v,t)dv = \mathcal{P}(\theta,t)d\theta$. While the evolution of densities is governed by infinite-dimensional partial differential equations, here the aim is to distill the dynamics of two characteristic macroscopic observables of the network: the mean firing rate R (the fraction of neurons spiking per infinitesimal time interval divided by that interval) and the mean voltage $V = \langle v_j \rangle$. Conveniently, these two properties can be expressed using the Fourier expansion of $\mathcal{P}(\theta,t) = (2\pi)^{-1}\{1 + \sum_{n\geq 1} Z_n(t)e^{-in\theta} + c.c.\}$. The $Z_n(t)$ are the Kuramoto-Daido order parameters, whose dynamics can be obtained from (C5) as (Pietras et al., 2023)

$$\tau_m \dot{Z}_n = n\left[i\omega Z_n + hZ_{n-1} - h^* Z_{n+1} - \gamma(Z_n + \tfrac{1}{2}Z_{n-1} + \tfrac{1}{2}Z_{n+1})\right] , \tag{D1}$$

where $\omega = 1 + I_0 + I_{\text{syn}}$ and $h = \frac{i}{2}(I_0 + I_{\text{syn}} - 1)$.

The firing rate is the flux of the probability density through the threshold π and the mean voltage is the population average of the membrane potentials $v_j = \tan(\theta_j/2) = \sin\theta_j/(1+\cos\theta_j)$. More compactly, one can find their expressions in terms of the order parameters

$$R(t) = \frac{2}{\tau_m}\mathcal{P}(\pi,t) = \frac{1}{\pi\tau_m}\left\{1 + \sum_{n=1}^{\infty}(-1)^n\left[Z_n(t) + Z_n^*(t)\right]\right\} , \tag{D2a}$$

$$V(t) = \lim_{\epsilon\to 0}\left\langle \frac{\sin\theta}{1+\cos\theta+\epsilon}\right\rangle = i\sum_{n=1}^{\infty}(-1)^n\left[Z_n(t) - Z_n^*(t)\right] . \tag{D2b}$$

Capitalizing on the similarity between R and V, Eqs. (D2) can be combined (Montbrió et al., 2015) to obtain (Pietras et al., 2023)

$$\pi\tau_m R - iV = 1 + 2\sum_{n=1}^{\infty}(-1)^n Z_n = \Phi + \lambda\frac{\mathcal{M}(-\sigma)}{\sigma}, \tag{D3}$$

where $\mathcal{M}(k)$ is a constant function that depends on the distribution $\mathcal{W}(v,0)$ of the QIF neurons' initial voltages $v_j(0)$, and the dynamics of the three complex variables $\Phi, \lambda, \sigma \in \mathbb{C}$ are governed by the ODEs

$$\tau_m\dot{\Phi} = i\Phi^2 - iI_0 - iI_{\text{syn}}(t) + \gamma, \quad \tau_m\dot{\lambda} = 2i\Phi\lambda, \quad \tau_m\dot{\sigma} = i\lambda . \tag{D4}$$

The firing rate and voltage (RV) dynamics of the QIF network follow from Eq. (D3) and are hence six-dimensional. For non-vanishing Cauchy white noise and/or for heterogeneous input currents following a Cauchy-Lorentz distribution of finite width, i.e. for $\gamma > 0$, we have proven that $\lambda \to 0$ for $t \to \infty$ and, hence, the collective dynamics becomes two-dimensional (Pietras et al., 2023). On the so-called Lorentzian manifold (Montbrió et al., 2015), $\{\lambda = 0\}$, which is closely related to the Ott-Antonsen manifold (Ott and Antonsen, 2008), the collective dynamics is solely governed by $\Phi = \pi\tau_m R - iV$, yielding the RV dynamics (Montbrió et al., 2015; Clusella and Montbrió, 2022)

$$\tau_m\dot{R} = \frac{\gamma}{\pi} + 2RV , \tag{D5a}$$

$$\tau_m\dot{V} = V^2 - (\pi\tau_m R)^2 + I_{\text{syn}} + I_0 , \tag{D5b}$$

which also describes all possible attractors of the full dynamics (D3 & D4). Correspondingly, on the Ott-Antonsen manifold the collective dynamics is uniquely described by $Q = Z_1$, which is

the Kuramoto order parameter, and all higher modes are powers of Q: $Z_n = Q^n$, see (Luke et al., 2013; So et al., 2014; Laing, 2014, 2015).

As the focus of this paper lies on analyzing the steady states of the system, it suffices to consider the RV dynamics (D5) on the invariant Lorentzian manifold. Moreover, when starting on the Lorentzian manifold, i.e. when initializing the voltages $v_j(0)$ according to a Cauchy-Lorentz distribution (which corresponds to a wrapped Cauchy distribution of initial phases $\theta_j(0)$), then for all times $t \geq 0$ the voltage distribution $\mathcal{W}(v,t)$ is given by the Cauchy-Lorentz distribution (Montbrió et al., 2015; Pietras et al., 2019; Clusella and Montbrió, 2022)

$$\mathcal{W}(v,t) = \frac{\tau_m R(t)}{[v - V(t)]^2 + [\pi \tau_m R(t)]^2} \,. \tag{D6}$$

E Chemical interaction via general pulses

Chemical synaptic interactions are modeled as the postsynaptic response $s(t)$ to a presynaptic pulse $p(t)$. In the following, I briefly present possible choices of the pulse profile $p(\theta) = p(\theta(t))$. As in the main text, I assume that the pulse function is smooth and has the following properties:

1. p is localized around $\theta = \pi$ (or when the membrane potential diverges, $v \to \infty$, respectively),
2. $p(\theta) \geq 0$ is non-negative and vanishes at least once,
3. the total area is normalized $\int_0^{2\pi} p(\theta) d\theta = 2\pi$.

The pulse function is periodic and thus admits an expansion as a Fourier series

$$p(\theta) = \sum_{k=-\infty}^{\infty} c_k e^{ik\theta}, \quad c_0 = 1. \tag{E1}$$

Then, the mean presynaptic pulse activity $P = \langle p \rangle$ is represented via the order parameters as

$$P = \langle p \rangle = \int_0^{2\pi} p(\theta) \mathcal{P}(\theta,t) d\theta = 1 + \sum_{k=1}^{\infty} (c_k^* Z_k + c_k Z_k^*) \,. \tag{E2}$$

As a side note, I remark that one could have defined the mean presynaptic activity (E2) also in the QIF voltage description as $\langle p \rangle = \int_{\mathbb{R}} p(v) \mathcal{W}(v,t) dv$. In the phase description, however, one can capitalize on the fact that $p(\theta)$ and $\mathcal{P}(\theta,t)$ admit a discrete Fourier series representation, which facilitates the proposed reduction to great extent. The voltage formulation of the QIF neurons may therefore be interpreted based on the transformation $v = \tan(\theta/2)$ to the θ-model (Pietras et al., 2023), e.g., the pulse function in v can be obtained via $p(\theta) = p(2 \arctan(v(t)))$.

δ-spikes. When neurons only emit a unitary pulse at the instant when they spike, i.e. when $\theta = \pi$, then the pulse profile can be expressed as the limit of a Dirac δ-function $p_{\delta,\pi}(\theta) = 2\pi\delta(\theta - \pi)$. The Fourier coefficients are $c_k = (-1)^{|k|}$ and the synaptic activity reduces via Eq. (D2a) to the mean firing rate

$$P_{\delta,\pi} = \langle p_{\delta,\pi} \rangle = 1 + \sum_{k=1}^{\infty} (-1)^k (Z_k + Z_k^*) = \pi \tau_m R(t) \,. \tag{E3}$$

Ariaratnam-Strogatz (AS) pulses. Ariaratnam and Strogatz (2001) suggested a family of pulse profiles $p_{\mathrm{AS},n}(\theta) = a_n (1 - \cos\theta)^n$ with $a_n = \frac{2^n (n!)^2}{(2n)!}$, where $n \in \mathbb{N}$ is an integer parameter. The AS pulses are smooth, but in the limit $n \to \infty$ they converge to δ-spikes. The average synaptic activity is a finite sum of the order parameters

$$P_{\mathrm{AS},n} = \langle p_{\mathrm{AS},n} \rangle = 1 + (n!)^2 \sum_{k=1}^{n} (-1)^k \frac{Z_k + Z_k^*}{(n+k)!(n-k)!} \,. \tag{E4}$$

Rectified-Poisson (RP) pulses. Gallego et al. (2017) suggested the following pulse form

$$p_{\mathrm{RP},r}(\theta) = \frac{(1-r)(1-\cos\theta)}{1+2r\cos\theta+r^2}\ ,$$

where the "sharpness" parameter $r \in (-1,1)$ determines the width of the pulse: For $r=-1$, $p_{\mathrm{RP},r}=1$ is flat; for $r=0$, $p_{\mathrm{RP},r}(\theta)=1-\cos\theta$ coincides with the AS pulse with $n=1$; and in the limit $r\to 1$, $p_{\mathrm{RP},r}(\theta)$ becomes a δ-spike. The Fourier coefficients are $c_k = \frac{1+r}{2r}(-r)^{|k|}$ and the average activity is the infinite series

$$P_{\mathrm{RP},r} = \langle p_{\mathrm{RP},r}\rangle = 1 + \frac{1+r}{2r}\sum_{k=1}^{\infty}(-r)^k\big[Z_k + Z_k^*\big]. \tag{E5}$$

Kato-Jones pulses. The pulse shapes above are symmetric about $\theta=\pi$ with $p(0)=0$. Allowing for asymmetric pulses and loosen pulse-assumption 1, one can generalize the RP pulse in the same way as Kato and Jones (2015) generalized the wrapped Cauchy distribution (a.k.a. Poisson kernel). I assume that the Fourier coefficients c_k of the pulse function (E1) are

$$c_k = ae^{i\varphi}\big(re^{i\psi}\big)^k, \quad k=1,2,\ldots, \quad c_{-k}=c_k^*\ .$$

One recovers the RP pulse for $\varphi=0$ and $a=\frac{1+r}{2r}$. By imposing pulse-property 2 from above, I set $a=(1-r^2)/(2r)/(1-r\cos\varphi)$ and obtain the general pulse profile $p_{r,\varphi,\psi}$ with parameters r,φ and ψ

$$p_{r,\varphi,\psi}(\theta) = 1 + \frac{1-r^2}{1-r\cos\varphi}\frac{\cos(\theta-\psi-\varphi)-r\cos(\varphi)}{1-2r\cos(\theta-\psi)+r^2}\ , \tag{E6}$$

where the parameter $\varphi\in[-\pi,\pi)$ governs the asymmetry of the pulse: for $\varphi>0$, the pulse is skewed to the right of the peak, and for $\varphi<0$ to the left. The average synaptic activity is represented as an infinite series

$$P_{r,\varphi,\psi} = \langle p_{r,\varphi,\psi}\rangle = 1 + \frac{1-r^2}{2r(1-r\cos\varphi)}\sum_{k=1}^{\infty} r^k\Big[e^{-i(\varphi+k\psi)}Z_k + e^{i(\varphi+k\psi)}Z_k^*\Big]. \tag{E7}$$

F Recurrent coupling in terms of collective variables

As shown in Appendix E, it is possible to represent the relevant observables R,V and the mean fields $P=\langle p\rangle$ governing the dynamics of the neural population via the order parameters Z_k. Such representations can directly be used for numerical simulations of the ensemble of neurons in the thermodynamic limit, with a proper truncation of the infinite series. However, it is advantageous to express the mean presynaptic pulse activity P through the new complex variables Φ,λ,σ, and eventually through R and V on the Lorentzian manifold.

For any P expressed as an infinite series of the form (E7)—in fact, the δ-spike and the RP pulse (E5) are special cases of the Kato-Jones pulse (E6)—, one can employ the same approach as in the derivation of (D3). As I will present in Appendix G, one finds the full expression for the mean presynaptic activity $P_{r,\varphi,\psi}=P_{r,\varphi,\psi}(\Phi,\lambda,\sigma)$ with Kato-Jones pulses $p_{r,\varphi,\psi}$ given by Eq. (E6) in terms of the complex variables Φ,λ,σ:

$$\begin{aligned} P_{r,\varphi,\psi} = \mathrm{Re}\Bigg\{ &\frac{(1-r^2)(1+\Phi)e^{-i\varphi}+(r-\cos\varphi)u}{r(1-r\cos\varphi)u} + \\ &\frac{2\lambda(1-r^2)e^{-i(\varphi+\psi)}}{u\,[\lambda(1+re^{-i\psi})-\sigma u]\,(1-r\cos\varphi)}\mathcal{M}\Big(-\sigma+\frac{\lambda}{u}(1+re^{-i\psi})\Big)\Bigg\} \end{aligned} \tag{F1}$$

where I introduced the auxiliary variable $u=1-re^{-i\psi}+\Phi(1+re^{-i\psi})$. For δ-spikes, $(r,\varphi,\psi)\to(1,0,\pi)$, one finds that $u\to 1$ and $\langle p_{1,0,\pi}\rangle = \langle p_{\delta,\pi}\rangle = \mathrm{Re}\big[\Phi+\lambda\mathcal{M}(-\sigma)/\sigma\big] = \pi\tau_m R$ as expected from (D3).

On the Lorentzian manifold

Since the interest lies in asymptotic dynamical regimes, I focus on the dynamics on the Lorentzian manifold and take $\lambda \to 0$, where $\Phi = \pi\tau_m R - iV$ holds. Hence, the mean presynaptic activity simplifies as

$$
\begin{aligned}
P_{r,\varphi,\psi}(R,V) &= \mathrm{Re}[\alpha/\beta] = a/b, \quad \text{where} \\
\alpha &= (1-r^2)(1+\pi\tau_m R - iV)e^{-i\varphi} \\
&\quad + (r-\cos\varphi)\big[1 - re^{-i\psi} + (\pi\tau_m R - iV)(1+re^{-i\psi})\big] \\
\beta &= r(1-r\cos\varphi)\big[1 - re^{-i\psi} + (\pi\tau_m R - iV)(1+re^{-i\psi})\big] \\
a &= (1-2r\cos\varphi + r^2)\big[1 + (\pi\tau_m R)^2 + V^2\big] + (1-r^2)2\pi\tau_m R \\
&\quad + \big[1 - (\pi\tau_m R)^2 - V^2\big]\big[\cos(\psi+\varphi) - 2r\cos(\psi) + r^2\cos(\psi-\varphi)\big] \\
&\quad + 2V\big[\sin(\psi+\varphi) - 2r\sin(\psi) + r^2\sin(\psi-\varphi)\big] \\
b &= (1-r\cos\varphi)\big\{(1+r^2)\big[1 + (\pi\tau_m R)^2 + V^2\big] + (1-r^2)2\pi\tau_m R\big\} \\
&\quad - 2r(1-r\cos\varphi)\big\{2V\sin\psi + \big[1 - (\pi\tau_m R)^2 - V^2\big]\cos\psi\big\}
\end{aligned}
\tag{F2}
$$

and is fully expressed by the firing rate R and mean voltage V. In sum, one obtains a closed set of firing rate equations (D5) for R and V with the synaptic input I_{syn} as a function of R and V through (F2), which is the formula (22) of the main text. I will now present special cases of pulse profiles $p_{r,\varphi,\psi}$ such that (F2) simplifies further.

F.1 Symmetric pulses ($\varphi = 0$)

For pulses $p_{r,\varphi,\psi}$ that are symmetric ($\varphi = 0$) about the pulse peak at $\psi \in [0, 2\pi)$ with width $r \in (0,1]$, one has

$$
\begin{aligned}
P_{r,0,\psi}(R,V) &= a_s/b_s, \quad \text{where} \\
a_s &= (1+r)2\pi\tau_m R + (1-r)\big[1 + (\pi\tau_m R)^2 + V^2 + c_s\big] \\
b_s &= (1-r^2)2\pi\tau_m R + (1+r^2)\big[1 + (\pi\tau_m R)^2 + V^2\big] - 2rc_s \\
c_s &= 2V\sin\psi + \big[1 - (\pi\tau_m R)^2 - V^2\big]\cos\psi
\end{aligned}
\tag{F3}
$$

For symmetric pulses $p_{r,0,\pi}$ about the peak at $\psi = \pi$, one retrieves the RP pulse with width $r \in (0,1]$. Here, the pulse peak $\psi = 2\arctan(v_{\mathrm{thr}})$ coincides with the QIF threshold $v_{\mathrm{thr}} = v_p \to \infty$, and the pulse is strongest when a neuron spikes. One finds

$$
P_{\mathrm{RP},r}(R,V) = \frac{2\pi\tau_m R[1 + r + (1-r)\pi\tau_m R] + 2(1-r)V^2}{[1 + r + (1-r)\pi\tau_m R]^2 + (1-r)^2 V^2} \quad \overset{r\to 1}{\longrightarrow} \quad \pi\tau_m R. \tag{F4}
$$

For shifted RP pulses $p_{r,0,\psi}$, $\psi \neq \pi$, one can describe the mean presynaptic activity $P_{r,0,\psi}(R,V) = a_s/b_s$ in terms of the virtual threshold $v_{\mathrm{thr}} = \tan(\psi/2)$ as:

$$
\begin{aligned}
a_s &= 4\pi\tau_m R(1+v_{\mathrm{thr}}^2) + 2(1-r)\big[(\pi\tau_m R v_{\mathrm{thr}})^2 + (1 + V v_{\mathrm{thr}})^2 - \pi\tau_m R(1+v_{\mathrm{thr}}^2)\big] \\
b_s &= 4\big[(\pi\tau_m R)^2 + (V - v_{\mathrm{thr}})^2\big] + (1-r)^2(1+v_{\mathrm{thr}}^2)\big[(1-\pi\tau_m R)^2 + V^2\big] \\
&\quad - 4(1-r)\big[(V - v_{\mathrm{thr}})^2 - \pi\tau_m R(1 - \pi\tau_m R + v_{\mathrm{thr}}^2)\big]
\end{aligned}
$$

F.2 Dirac δ-pulses with peak phase $\psi \in [0, 2\pi)$

For Dirac δ-pulses in the limit $r \to 1$ and $\varphi = 0$, a unitary pulse is emitted at phase $\theta = \psi$, that is whenever a neuron's membrane potential v increases through the virtual threshold $v_{\mathrm{thr}} =$

$\tan(\psi/2)$. Then,

$$P_{1,0,\psi}(R,V) = \frac{2\pi\tau_m R}{1+(\pi\tau_m R)^2+V^2-\{2V\sin\psi+[1-(\pi\tau_m R)^2-V^2]\cos\psi\}} \overset{\psi\to\pi}{\longrightarrow} \pi\tau_m R\,, \tag{F5}$$

or alternatively, cf. Eq. (24),

$$P_{1,0,\psi}(R,V) = P_{\delta,v_{\rm thr}}(R,V) = \frac{\pi\tau_m R(1+v_{\rm thr}^2)}{(\pi\tau_m R)^2+(V-v_{\rm thr})^2} \overset{v_{\rm thr}\to\infty}{\longrightarrow} \pi\tau_m R\,.$$

As discussed in Section 5.2, this expression can be interpreted as an advanced ($\psi<\pi$) or delayed ($\psi>\pi$) pulse-coupling. It is, however, not a delay in the traditional sense $P_{\delta,v_{\rm thr}} \neq \pi\tau_m R(t-\tau_l)$ with fixed latency τ_l, but rather the latency arises as a combination of the virtual voltage threshold $v_{\rm thr} = \tan(\psi/2)$ and the membrane time constant τ_m. Still, one may consider the resulting RV dynamics (20) with shifted δ-pulse-coupling (24) as a low-dimensional approximation of the infinite-dimensional collective dynamics with real delays, $\tau_l \neq 0$.

F.3 Asymmetric pulses with $\psi = \pi$

For asymmetric pulses, $\varphi \neq 0$, of finite width $r \in (0,1)$, the pulse has no longer its peak at ψ, but it will be shifted to the right ($\varphi>0$) or to the left ($\varphi<0$). In fact, the pulse peak is no longer at $\theta=\psi$, or equivalently at $v=v_{\rm thr}$, but is shifted towards $v_{max} = \frac{(1+r)v_{\rm thr}\cos(\varphi/2)+(1-r)\sin(\varphi/2)}{(1+r)\cos(\varphi/2)-(1-r)v_{\rm thr}\sin(\varphi/2)}$. Likewise, the pulse vanishes for $v_{min} = \frac{(1+r)v_{\rm thr}\sin(\varphi/2)-(1-r)\cos(\varphi/2)}{(1+r)\sin(\varphi/2)+(1-r)v_{\rm thr}\cos(\varphi/2)}$. The corresponding peak and trough phases $\theta_{max/min}$ can be found via $\theta_{max/min} = 2\arctan(v_{max/min})$.

For the virtual threshold $v_{\rm thr}$ coinciding with the QIF threshold, $v_{\rm thr}=v_p\to\infty$ or $\psi=\pi$, one has $v_{max} = (1+r)/(1-r)/\tan(-\varphi/2)$ and $v_{min} = (1+r)\tan(\varphi/2)/(1-r)$, so that for $\varphi>0$ the pulse is strongest after the neuron has spiked (because $(1+r)/(1-r)>0$ for $r\in(0,1)$). In fact, for positively slightly skewed, narrow pulses, i.e. for $0<\varphi\ll\pi$ and $0\ll r<1$, the pulse sharply increases shortly before a neuron spikes but reaches its peak value only after the spike, and then decays moderately as the neuron returns to its resting potential, see Fig. 8(a, red and green curves). Such a behavior is characteristic for second-order synapses with distinct rise- and decay-time constants, see, e.g., (Clusella et al., 2022a). Including a peak phase ψ different from π further allows to shift the onset of the pulse and, thus, to model also a latency effect of synaptic transmission.

Setting $\psi=\pi$, one obtains on the Lorentzian manifold

$$\begin{aligned} P_{r,\varphi,\pi}(R,V) &= a_p/[b_p(1-r\cos\varphi)], \quad \text{where} \\ a_p &= b_p - \left[(1+r)^2-(1-r)^2(\pi^2\tau_m^2R^2+V^2)\right]\cos\varphi - 2(1-r^2)V\sin\varphi \\ b_p &= [(1+r)+(1-r)\pi\tau_m R]^2 + (1-r)^2V^2 \end{aligned} \tag{F6}$$

For $\psi\neq\pi$, the mean presynaptic activity $P_{r,\varphi,\psi}$ has to be determined from (F2).

G Derivation of mean-field and coupling expressions

In order to derive the expression for the recurrent coupling in terms of the collective variables Φ,λ,σ, see Appendix E, I follow the same procedure as in (Pietras et al., 2023). In brief, I use Eq. (15) of Cestnik and Pikovsky (2022b),

$$Z_n(t) = \sum_{m=0}^{n}\binom{n}{m}\beta_m(t)Q(t)^{n-m}\,,$$

and the complex-valued variables Q, y, and s that are obtained from Φ, λ, σ through the transformations

$$Q = \frac{1-\Phi}{1+\Phi}, \quad y = \frac{2\lambda}{(1+\Phi)^2}, \quad s = \sigma - \frac{\lambda}{1+\Phi}\,. \tag{G1}$$

Together with the identity $\sum_{n=0}^{\infty}\binom{n+k-1}{k-1}x^n = (1-x)^{-k}$, one finds

$$\begin{aligned}
\sum_{n=0}^{\infty}\xi^n Z_n &= \sum_{n=0}^{\infty}\xi^n\sum_{m=0}^{n}\binom{n}{m}\beta_m Q^{n-m} = \sum_{m=0}^{\infty}b_m\sum_{n=m}^{\infty}\xi^n\binom{n}{m}Q^{n-m}\\
&= \sum_{m=0}^{\infty}b_m\xi^m\sum_{n=0}^{\infty}\xi^n\binom{n+m}{m}Q^n = \sum_{m=0}^{\infty}b_m\xi^m\sum_{n=0}^{\infty}\binom{n+m}{m}(\xi Q)^n\\
&= \frac{1}{1-\xi Q} + \sum_{m=1}^{\infty}\alpha_m\,(y\xi)^m\,(1-\xi Q)^{-(m+1)}\\
&= (1-\xi Q)^{-1}\left[1+\sum_{m=1}^{\infty}\alpha_m\left(\frac{y\xi}{1-\xi Q}\right)^m\right],
\end{aligned}$$

where first one has to use $b_0 \equiv 1$ and then substitute $\beta_m(t) = \alpha_m(t)y(t)^m$. Now I use the definitions of generating functions $\mathcal{A}$ and $\mathcal{M}$ [Eqs. (19) and (21) of Cestnik and Pikovsky (2022b)]:

$$\mathcal{A}(k) = \sum_{m=1}^{\infty}\alpha_m k^m = \frac{k}{k-s}\mathcal{M}(k-s)$$

This yields with $k = y\xi/(1-\xi Q)$

$$\begin{aligned}
\sum_{n=0}^{\infty}\xi^n Z_n &= (1-\xi Q)^{-1}\left[1+\sum_{m=1}^{\infty}\alpha_m k^m\right]\\
&= (1-\xi Q)^{-1}\left[1+\frac{y\xi}{y\xi - s(1-\xi Q)}\mathcal{M}\left(-s+\frac{y\xi}{1-\xi Q}\right)\right].
\end{aligned}$$

For any complex constant $C \in \mathbb{C}$, one then obtains (recall that $Z_0 = 1$ for normalization)

$$\sum_{n=1}^{\infty}\left[C\xi^n Z_n + C^*\left(\xi^*\right)^n Z_n^*\right] =$$
$$2\mathrm{Re}\left\{\frac{C}{1-\xi Q}\left[1+\frac{y\xi}{y\xi - s(1-\xi Q)}\mathcal{M}\left(-s+\frac{y\xi}{1-\xi Q}\right)\right]\right\} - 2\mathrm{Re}(C)\,.$$

For example, one finds an expression for the firing rate R by setting $C = 1$ and $\xi = -1$ and using (D2a),

$$\pi\tau_m R = 1 + \sum_{n=1}^{\infty}\left[(-1)^n Z_n + c.c.\right] =$$
$$2\mathrm{Re}\left\{(1+Q)^{-1}\left[1+\frac{y}{y+s(1+Q)}\mathcal{M}\left(-\frac{y+s(1+Q)}{1+Q}\right)\right]\right\} - 1.$$

Inserting transformation (G1) yields expression (D3): $\pi\tau_m R = \mathrm{Re}\left\{\Phi + \lambda\mathcal{M}(-\sigma)/\sigma\right\}$.

One can further apply the calculation presented above to calculate the mean presynaptic activity $P = \langle p\rangle$ for the pulses $p_{r,\varphi,\psi}$ given by (E6). Setting $C = e^{-i\varphi}$ and $\xi = re^{-i\psi}$, one finds

that (E7) reduces to

$$P_{r,\varphi,\psi} = \frac{r-\cos\varphi}{r(1-r\cos\varphi)} + \frac{1-r^2}{r(1-r\cos\varphi)} \times \\ \times \mathrm{Re}\left\{ \frac{e^{-i\varphi}}{1-re^{-i\psi}Q} \left[1 + \frac{yre^{-i\psi}}{yre^{-i\psi} - s(1-re^{-i\psi}Q)} \mathcal{M}\left(-s + \frac{yre^{-i\psi}}{1-re^{-i\psi}Q} \right) \right] \right\}. \tag{G2}$$

With the transformation (G1), Eq. (G2) can be expressed in the variables Φ, λ, σ and one finally obtains Eq. (F1),

$$P_{r,\varphi,\psi}(\Phi,\lambda,\sigma) = \mathrm{Re}\left\{ \frac{(1-r^2)(1+\Phi)e^{-i\alpha} + (r-\cos\alpha)u}{r(1-r\cos\alpha)u} + \right. \\ \left. + \frac{2\lambda(1-r^2)e^{-i(\alpha+\psi)}}{u\left[\lambda(1+re^{-i\psi}) - \sigma u\right](1-r\cos\varphi)} \mathcal{M}\left(-\sigma + \frac{\lambda(1+re^{-i\psi})}{u} \right) \right\}$$

with the auxiliary variable $u = 1 - re^{-i\psi} + \Phi(1+re^{-i\psi})$.

H Finite resetting & nonsymmetric spikes

In (Montbrió and Pazó, 2020), Montbrió and Pazó showed that the assumption of symmetric threshold and reset values $v_p = -v_r \to \infty$ can be relaxed in the QIF-RV dynamics on the Lorentzian manifold, leading to novel low-dimensional firing rate equations with arbitrary spike asymmetry. This may become relevant in the case of the QIF model with biophysically realistic parameter values, see Appendix C. For instance, (Latham et al., 2000) considered the following parameters

$$v_r = -65mV, \quad v_t = -50mV, \quad v_p = 20mV, \quad v_r = -80mV, \tag{H1}$$

for which the resetting is nonsymmetric. The spike-asymmetry can be quantified with a real, positive parameter $a > 0$ as the ratio

$$a \equiv \frac{v_p}{-v_r}. \tag{H2}$$

When $a = 1$, the resetting rule of the QIF model is symmetric; this spike-symmetry is implicitly assumed in the transformation between the QIF and the θ-neuron models. For $a > 1$, the spike is net-positive, whereas for $a < 1$ it is net-negative, see (Montbrió and Pazó, 2020). The QIF dynamics (C1) with parameters (H1) yields a spike-asymmetry of $a \approx 3.4$.

To determine the collective dynamics with nonsymmetric spikes beyond the Lorentzian manifold and in terms of the three complex variables Φ, λ, σ, one can follow Montbrió and Pazó (2020) and consider the following derivation as an approximation of the actual network dynamics for finite apex and repolarization values $v_p = -av_r < \infty$. The firing rate R will still be computed as the flux at infinity. But for the mean voltage V, one has to compute

$$V(t) = \lim_{v_r \to -\infty} \int_{v_r}^{v_p = -av_r} v\mathcal{W}(v,t)dv = \left[\text{p.v.} \int_{-\infty}^{\infty} + \lim_{v_r \to -\infty} \int_{v_r}^{-av_r} \right] v\mathcal{W}(v,t)dv \tag{H3}$$

where I split the integral into two parts: one integral with symmetric integration limits (Cauchy principal value), and another one with the remaining integration interval (Montbrió and Pazó, 2020). I have already computed the first integral

$$V_s = \text{p.v.} \int_{-\infty}^{\infty} v\mathcal{W}(v,t)dv \tag{H4}$$

with symmetric resetting in Eq. (D2b) where I used the identities $\mathcal{W}(v,t)dv = \mathcal{P}(\theta,t)d\theta$ and $v = \tan(\theta/2)$. To compute the second integral, one finds analogously

$$\lim_{v_r\to-\infty}\int_{v_r}^{-av_r} v\mathcal{W}(v,t)dv = \lim_{v_r\to-\infty}\int_{v_r}^{-av_r}\frac{(\sin\theta)\mathcal{P}(\theta,t)}{1+\cos\theta}d\theta$$
$$= \frac{\log(a)}{\pi}\left[1+\sum_{n=1}^{\infty}(-1)^n[Z_n+Z_n^*]\right] = \tau_m\log(a)R(t)\ .$$

Hence, the mean voltage V with asymmetric spikes $a>0$ becomes

$$V(t) = V_s(t) + \tau_m\log(a)R(t)\ . \tag{H5}$$

The relation (D3) between the complex variables $\Phi,\lambda,\sigma\in\mathbb{C}$ and the firing rate R and mean voltage V then becomes

$$\Phi+\lambda\frac{\mathcal{M}(-\sigma)}{\sigma} = 1+2\sum_{n=1}^{\infty}(-1)^nZ_n = \pi\tau_mR - iV_s = \tau_m[\pi+i\log(a)]R - iV, \tag{H6}$$

with $\mathcal{M}(k)$ as defined above and the dynamics of Φ,λ,σ continue to be governed by system (D4). On the Lorentzian manifold, $\lambda\to0$, we then obtain the firing rate equations (see also Montbrió and Pazó, 2020)

$$\tau_m\dot{R} = \frac{\gamma}{\pi}+2RV_s\ , \tag{H7a}$$
$$\tau_m\dot{V}_s = V_s^2-(\pi\tau R)^2+I_{\rm syn}+I_0\ . \tag{H7b}$$

The two ordinary differential equations for the mean firing rate R and the auxiliary variable $V_s = V-\tau_m\log(a)R$ describe the dynamics of the ensemble exactly in the limit of infinite N, v_p, and $-v_r$. The dynamics (H7) for asymmetric spikes takes on an identical form as the RV dynamics (20) in the main text. For instantaneous global coupling via δ-spikes (i.e. Dirac δ-pulses emitted at the presynaptic spike times), $I_{\rm syn}$ is proportional to τ_mR and the spike asymmetry only shifts the mean membrane potential as in Eq. (H5), but has no qualitative effect on the collective dynamics. For electrical coupling via gap junctions of strength $g>0$, the asymmetric spike effectively introduces a (linear) virtual chemical coupling as $I_{\rm syn}$ now incorporates the additional term $g\tau_m\log(a)R$ and thus influences the onset of collective oscillations (Montbrió and Pazó, 2020). For instantaneous pulse-coupling via pulses $p_{r,\varphi,\psi}$ of strength J, the asymmetric spike will influence the collective dynamics in a non-trivial way as the recurrent synaptic input $I_{\rm syn}$ in Eq. (H7) depends on the mean pulse activity

$$I_{\rm syn} = JP_{r,\varphi,\psi}(R,V) = JP_{r,\varphi,\psi}(R,V_s+\tau_m\log(a)R)\ . \tag{H8}$$

The explicit voltage-dependence of the pulse-coupling thus introduces a virtual chemical coupling mediated by the firing rate R, which enters the RV dynamics (H7) in a highly nonlinear way and in interplay with V_s via Eq. (22).

It is an open question how this nonlinear virtual coupling affects the collective dynamics. Another open question concerns the simulation of QIF neurons with finite v_p and v_r interacting via smooth pulses $p_{r,\varphi,\psi}(2\arctan v)$, as these pulses technically require that the voltage variable $v\in(-\infty,\infty)$ can take on arbitrary values. It remains to be shown how the collective dynamics of such microscopic network simulations correspond to the exact macroscopic theory, Eqs. (H7) and (H8).