We consider a pair of identical Morris-Leca80 neurons [?], with parameters adapted from [?]31 The Morris-Lecar model is a set of two first-ordeß2 differential equations that describe the membran \(\mathre{3} \) dynamics of a spiking neuron. The depolarisation84 is modelled by an instantaneous calcium current35 and the hyperpolarisation by a slow potassium86 current and a leak current. The membrane potentia \$7 v_i and potassium activation w_i of neuron i (i, j) = 381, 2) is described by:

$$\dot{v}_i = f(v_i, w_i) - \bar{g}s_j(v_i - v_s),$$
 (1)40
 $\dot{w}_i = h(v_i, w_i).$ (2)42

Here v_s is the inhibitory reversal potential, and $\frac{43}{44}$ \bar{g} and s_j are the maximal synaptic conductance and the synaptic gating, respectively, constituting the total inhibitory conductance $\bar{g}s_j$ from neuron $\frac{1}{47}$ j to neuron i. Function $f(v_i, w_i)$ describes the 48 5 membrane currents of a single cell:

$$f(v_i, w_i) = -g_{\text{Ca}} m_{\infty}(v_i)(v_i - v_{\text{Ca}}) - g_{\text{K}} w_i(v_i - v_{\text{K}})$$

The currents include a constant current I, and three 52ionic currents: an instantaneous calcium current⁵³ a potassium current, and a leak current, with 24 9 respective reversal potentials $v_{\rm Ca}$, $v_{\rm K}$, and $v_{\rm L}$, as 55 10 well as maximum conductances $g_{\rm Ca}$, $g_{\rm K}$, and $g_{\rm L}$ 56 11 The function $h(v_i, w_i)$ models the kinetics of the 7

potassium gating variable w_i , and is given by

$$h(v_i, w_i) = \frac{w_{\infty}(v_i) - w_i}{\tau_w}$$
 (460)

The steady-state activation functions m_{∞} and 3 w_{∞} as well as the default model parameters ar 64 described in the Supplementary Material S1. 16

The dynamics of the synaptic interaction 66 17 between the neurons are governed by a synapti67 18 gating variable s_i and a depression variable d_i :

$$\dot{d}_i = \begin{cases} (1 - d_i)/\tau_a \text{ if } v_i < v_\theta, \\ -d_i/\tau_b \text{ if } v_i > v_\theta, \end{cases}$$

$$(5)$$

$$\dot{s}_i = \begin{cases} -s_i/\tau_\kappa \text{ if } v_i < v_\theta, \\ (d_i - s_i)/\tau_\gamma \text{ if } v_i > v_\theta. \end{cases}$$
 (6)

Variable d_i describes a firing rate dependent 20 depletion mechanism that governs the amount of 21 22 depression acting on the synapse. The model is 23 agnostic with respect to the exact mechanism of 24 this depletion, be it pre- or post-synaptic. When the voltage is below firing threshold, depression 25 26 variable d_i recovers with time constant τ_a , while 27 synaptic variable s_i decays with time constant τ_{κ} . Because synaptic depression occurs on a much 28 slower timescale than synaptic inhibition, we assume $\tau_d \gg \tau_{\kappa}$. When the voltage is above firing threshold, variable d_i decays to zero with τ_b , while s_i quickly approaches the respective value of d_i . Since τ_{γ} is the shortest time constant of the system with $\tau_{\gamma} \ll 1$, we can assume that whenever $v > v_{\theta}$ we can we have $s_i = d_i$. The equations for the depression model were adapted from the ?] model. These equations are a mathematically tractable simplification of the established phenomenological depression model previously described by ?].

When the cells are uncoupled $(\bar{q} = 0)$, the membrane dynamics are determined by the cubic v-nullcline $v_{\infty}(v_i)$ and the sigmoid w-nullcline $w_{\infty}(v_i)$, satisfying $\dot{v}_i = 0$ and $\dot{w}_i = 0$, respectively. The two curves intersect along the middle branch of v_{∞} , creating an unstable fixed point $p_f = (v_f, w_f)$ with a surrounding stable limit cycle of period $T = T_{act} + T_{inact}$ (??A). Here T_{act} is the amount of time the cell spends in the active state when $v > v_{\theta}$, while T_{inact} is the time it $f(v_i,w_i) = -g_{\mathrm{Ca}} m_{\infty}(v_i)(v_i - v_{\mathrm{Ca}}) - g_{\mathrm{K}} w_i (v_i - v_{\mathrm{K}} 50 - g_{\mathrm{Spends}}) \text{ in the silent state when } v < v_{\theta}. \text{ Trajectories along that limit cycle have the familiar shape of the silent state when } v < v_{\theta} = -g_{\mathrm{Ca}} m_{\infty}(v_i)(v_i - v_{\mathrm{Ca}}) - g_{\mathrm{K}} w_i (v_i - v_{\mathrm{K}} 50 - g_{\mathrm{Spends}}) \text{ in the silent state when } v < v_{\theta} = -g_{\mathrm{Ca}} m_{\infty}(v_i)(v_i - v_{\mathrm{Ca}}) - g_{\mathrm{K}} w_i (v_i - v_{\mathrm{K}} 50 - g_{\mathrm{Spends}}) \text{ in the silent state when } v < v_{\theta} = -g_{\mathrm{Ca}} m_{\infty}(v_i)(v_i - v_{\mathrm{Ca}}) - g_{\mathrm{K}} w_i (v_i - v_{\mathrm{K}} 50 - g_{\mathrm{Spends}}) \text{ in the silent state}$ action potential (??B). The trajectory of an action potential can be dissected into four phases: (1) a silent phase, (2) a jump up, (3) an active phase, and (4) a jump down [see e.g.?]. During the silent phase the trajectory evolves along the left branch $(v_i < v_\theta)$ of the cubic v-nullcline. Once the trajectory reaches the local minimum of v_{∞} , it "jumps up" to the right branch $(v_i > v_\theta)$, crossing the firing threshold v_{θ} . During the active phase the trajectory then evolves along the right branch of the cubic until it arrives at the local maximum, where it "jumps down" to the left branch commencing a new cycle.

> The two-cell network model is numerically integrated using an adaptive step-size integrator for stiff differential equations implemented with XPPAUT [?] and controlled through the Python packages SciPy [?] and PyXPP [?]. The following mathematical analysis is performed on the equations of a single cell. Unless required for clarity, we will therefore omit the subscripts i, jfrom here on.

1 **Frontiers**

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