Correlation between neural spike trains increases with firing rate

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Populations of neurons in the retina1–3, olfactory system4, visual5 and somatosensory6 thalamus, and several cortical regions7–10 show temporal correlation between the discharge times of their action potentials (spike trains). Correlated firing has been linked to stimulus encoding8, attention9, stimulus discrimination8, and motor behaviour11. Nevertheless, the mechanisms underlying correlated spiking are poorly understood2,3,11–20, and its coding implications are still debated3,13,21,22. It is not clear, for instance, whether correlations between the discharges of two neurons are determined solely by the correlation between their afferent currents, or whether they also depend on the mean and variance of the input. We addressed this question by computing the spike train correlation coefficient of unconnected pairs of in vitro cortical neurons receiving correlated inputs. Notably, even when the input correlation remained fixed, the spike train output correlation increased with the firing rate, but was largely independent of spike train variability. With a combination of analytical techniques and numerical simulations using ‘integrate-and-fire’ neuron models we show that this relationship between output correlation and firing rate is robust to input heterogeneities. Finally, this overlooked relationship is replicated by a standard threshold-linear model, demonstrating the universality of the result. This connection between the rate and correlation of spiking activity links two fundamental features of the neural code.

How do cortical cells transform correlation between their synaptic currents into correlation between their output spike trains? We addressed this question by studying pairwise spike train correlations, a strategy that can capture the full statistical structure of a neural network2,11. Correlated fluctuating currents resembling synaptic activity21 were injected into the somata of 20 unconnected cortical neurons (see Methods). The input current to cell i (i = 1, 2) was:

\[ I_i = \mu_i + \sigma \left( \sqrt{1 - \xi^2(t)} + \sqrt{\xi^2(t)} \right) \] (1)

where \( \mu_i \) is the temporal average of the current. The next term represents gaussian fluctuations with a temporal structure consistent with that in vivo3, and was composed of two weighted factors: \( \xi(t) \), which was independent for each cell, and \( \sqrt{\xi(t)} \), which was common to all cells (Fig. 1a). The input correlation coefficient, \( \rho \) (0 ≤ \( \rho \) ≤ 1), set the relative weight of the shared fluctuations, whereas \( \sigma \) set the variance of the total input current. These parameters were adjusted so that the variability and covariability of the membrane potentials21 and spike trains3 were similar to those observed in vivo (Fig. 1b). Typical spike train cross-correlation functions had positive central peaks with short timescales (∼20 ms; Fig. 1c, left), indicative of spike-time synchrony3.

To quantify spike train correlation between two cells, we computed the correlation coefficients1,7,8,10 of the spike counts, \( n_1 \) and \( n_2 \), over a sliding window of length \( T \):

\[ \rho_T = \frac{\text{Cov}(n_1, n_2)}{\sqrt{\text{Var}(n_1)\text{Var}(n_2)}} \] (2)

where Cov is the covariance and Var the variance. The correlation coefficient \( \rho_T \) is a dimensionless quantity ranging between 0 for independent and 1 for fully correlated spike trains. For large \( T \), \( \rho_T \) saturates to a value \( \rho^* \) (see Methods).

As expected, \( \rho_T \) increased with \( \rho^* \) (Fig. 1d); however, \( \rho_T \) was always less than \( \rho \), indicating that the correlation in the input currents bounds the correlation between the output spikes. Our central and more surprising finding was that \( \rho_T \) increased with the output firing rate \( v \) when \( c \) was fixed (Fig. 1e). We delivered currents with a fixed \( v \) = 0.5 and various values of \( \mu \) and \( \sigma \), thus eliciting spike trains with a range of firing rates and inter-spike interval coefficients of variation (CV). Spike trains with similar \( v \) were paired to compute \( \rho_T \). Despite \( \rho_T \) increasing strongly with rate it did not depend on the CV (inset Fig. 1e). The same increasing trend holds for all \( T \) values examined, and occurred across several identified cell types (see Supplementary Information).

The correlation–rate relationship (Fig. 1e) might be expected owing to an increased probability of spikes occurring close in time at high rates. The coefficient \( \rho_T \) corrects for this spurious rate-dependence of correlation, as illustrated by the following example of two cells receiving inputs constructed from three independent Poisson trains \( x_1 \), \( x_2 \) and \( x_3 \) with rates \( (1 - c) \nu \), \( (1 - c) \nu \) and \( c \nu \), respectively (Fig. 1f). For a simple model that converts every input spike into an output spike, the output trains \( x_1 = x_2 + x_3 \) and \( x_2 = x_3 + x_1 \) have rate \( \nu \) and a correlation that depends on the rate of the common train \( x \) (Fig. 1f, red spikes). The correlation measured as the average product \( <n_1 n_2> / \nu^2 \) (Fig. 1f) yields \( \rho_T \) through (1) the baseline chance correlation \( \nu \), which gives a non-zero correlation even in the case \( c = 0 \); (2) the scaling of the first term by \( \nu \), which gives the absolute number of synchronous spikes. The coefficient \( \rho_T \) (equation (2)) corrects for these \( \nu \)-dependencies through baseline subtraction and appropriate normalization:

\[ \rho_T = \frac{<n_1> - <n_1>^2}{\sqrt{<n_1>^2} - <n_1>^2 - <(n_1)^2> - <n_1>^2}}. \]

Specifically, noting \( <n_1> = \nu T \) and \( <n_1^2> = \nu^2 T + (\nu T)^2 \) yields \( \rho_T = \nu \sqrt{T / \nu T} = \nu \), thus showing how \( \rho_T \) does not have a built-in dependence on \( \nu \). Therefore the correlation–rate relationship exhibited by the data (Fig. 1e) is a priori unexpected, and instead must follow from an input–output property of spiking neurons.

To analyse further the correlation–rate relationship we replaced the in vitro neurons with a pair of leaky ‘integrate-and-fire’ (LIF) model neurons23 (see Methods). We fixed \( \mu \) and \( \sigma \), and performed simulations of the LIF neuron pair to compute \( \rho_T \). The coefficient \( c \) ranged from 0 to 0.3, thus yielding \( \rho_T \) values that are comparable to

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obtained via simulations (Fig. 2a, b, dashed lines).

Figure 1 | Relationship between output spike correlation and rate in in vitro cells. a, Twenty neurons from cortical slices (n = 11) were stimulated with gaussian currents (filtered with a time constant of 5 ms), with both common and independent components. b, Fluctuating current injection (bottom) often evoked nearly synchronous spikes (top arrowheads). Red and black traces are membrane potentials from cells 1 and 2, respectively. c, Typical cross- (left) and auto-correlation (right) functions from a pair of cells firing at low (blue), medium (red) and high (green) rate. d, A typical ρT versus c for a pair of cells. The correlation ρT in a population (n = 123 pairwise comparisons) increases with √<i>n</i>/CV and does not co-vary with √<i>CV/V</i> (inset, linear regression, r = 0.13); c was fixed at 0.5. Squares represent mean and s.d. of the population, and coloured circles the examples shown in c. f, A simple cell model that generates an output spike for every input spike. The correlation ρT is fixed at c, and independent of v. Throughout we used T = 40 ms.

As with the experiments, we varied μ or σ while keeping c fixed to obtain different values of v. A plot of ρ versus v when μ (or σ) was increased while keeping σ (or μ) fixed (inset of Fig. 2c) shows that ρ rapidly increased in the physiological range of 0–30 spikes s<sup>−1</sup> and asymptoted to c at high rates (Fig. 2c). Notably, the correlation–rate curves obtained by increasing μ or σ were nearly identical, even though voltage traces, correlation functions and CVs were very different (Fig. 2c). To show that these results did not depend on specific μ and σ values, we used equation (3) to sweep a large region of the μ–σ parameter space. The resultant ρ–v values were confined to a narrow band around the stereotyped curve (Fig. 2c, shaded areas). This allowed us to rewrite equation (3) in an approximate form in which the susceptibility, S, depends only on the output rate and not on the input variables μ and σ:

ρ = S(v)c

The empirically measured ρ also increased with v for all T examined; however, the approximation that S depends only on v (equation 4) becomes progressively less accurate as T decreases (see Supplementary Information).

To determine whether the correlation–rate relationship emerges in a less controlled and more biologically relevant setting, we performed simulations of a two-layer network where neurons from layer 1 were randomly connected to neurons in layer 2 (Fig. 3a). Layer 1 consisted...
A plot of $\rho_{ij}$ versus the geometric mean of the rates $\sqrt{v_i v_j}$ shows a positive correlation (Fig. 3c). However, the trend is not nearly as tight as the one obtained in the controlled setting (compare Fig. 3c with Fig. 2c), primarily owing to the heterogeneity in $v_{ij}$ (Fig. 3b, bottom panel). After normalization by the measured $c_{ij}$ values (obtainable experimentally using intracellular recordings), we obtained $S_{ij} = \rho_{ij}/c_{ij}$ and a tight clustering of points along the same stereotyped curve emerged (Fig. 3d). If we group the pairs according to how different their rates are, we find that spike trains with similar rates (Fig. 3d, black points) showed slightly larger $S_{ij}$ and a tighter correlation–rate relationship than pairs with more different rates (Fig. 3d, grey points). The same qualitative results were obtained in networks where synapses had slow kinetics (NMDA (N-methyl-D-aspartate) and GABA$_B$), or where the total conductance was highly non-gaussian (see Supplementary Information).

The experiments, simulations and theory, although showing a clear relationship between $\rho$ and $v$, do not give a mechanistic understanding of the relationship. To develop this intuition, we examined phenomenological neuron models defined by a simple function, $f$, that transforms an input random variable, $I$, with gaussian statistics $(\mu, \sigma)$ to an output random variable, $n$ (Fig. 4a). The inputs $I_1$ and $I_2$ to a pair of such neuron models were constructed in analogy to our two-cell system so that their correlation coefficient was $c$ (compare Fig. 1a to Fig. 4a). We varied the mean input, $\mu$, while keeping $\sigma$ and $c$ fixed (as in Fig. 2c, red curve), and computed the mean output $<n>$ and the output correlation coefficient $\rho$ (equation (2)). When $f$ was strictly linear in $I$ (Fig. 4c, top row), then $\rho=c$ for all values of $\mu$, making $\rho$ independent of $<n>$. However, when $f$ was threshold-linear, which captures the rectifying property of spike generation, $\rho$ increased with $<n>$ (Fig. 4b), as was observed previously (Figs 1c and 2c). The fact that the simple threshold-linear model reproduced the correlation–rate relationship indicates that the specific dynamics of the cell model are not vital to the relationship. Modifying the threshold-linear transfer, to include saturation, produced a non-monotonic relationship between $\rho$ and $<n>$ (Fig. 4c, middle row). Similarly, adding a boosting non-linearity to the threshold-linear model introduces a small concavity in the $\rho–<n>$ relationship (Fig. 4c, bottom row). Despite this diversity of behaviour across these examples, one general feature is clear: when the input distribution $P(I)$ (coloured gaussians) overlapped with the nonlinear region of $f$, then $\rho$ was significantly less than $c$.

Figure 3 | Correlation–rate relationship in a simple network. a, Two-layer network with sparse random connectivity (connection probability was 0.25) where layer 1 contained excitatory and inhibitory cells firing with Poisson statistics. Layer 2 cells were conductance-based LIF neurons with randomly chosen synaptic weights. b, Distribution of $r$ (top), CV (middle) and $\rho_{ij}$ and $c_{ij}$ (bottom) across layer 2. c, $\rho_{ij}$ versus $\sqrt{\sigma_i \sigma_j}$ for layer 2 (linear regression $r = 0.57$). d, $S_{ij} = \rho_{ij}/c_{ij}$ versus $\sqrt{\sigma_i \sigma_j}$ for each pair shown in c. Pairs were grouped according to $q_{ij} = v_i/v_j$ (with $v_i \leq v_j$): $q_{ij} > 0.5$ (black) and $q_{ij} \leq 0.5$ (grey).

Figure 4 | Nonlinearities shape the correlation–rate relationship in a phenomenological neural model. a, The transfer $n_i = f(I_i)$ for a pair of models; note the parallels with Fig. 1a. b, $f$ and the $\rho–v$ relationship for the threshold-linear model. Overlaid are the input densities $P(I)$ (coloured gaussians) for three values of $\mu$. c, Same as in b using three different $f$ functions (see text). d, The joint densities $P(I_1,I_2)$ and $P(n_1,n_2)$ for the threshold-linear model for the three values of $\mu$ in b. The threshold (white lines) partitions the input plane $(I_1,I_2)$ into four quadrants. $P(I_1,I_2)$ is transformed differently in each quadrant (middle column). In b–d we set $\sigma = 0.3$ and the results are valid for $c$ in the interval $(0,0.5)$, whereas $c = 0.5$ in panel c.
For the threshold and saturating nonlinearities this effect was especially prominent as \( \rho \) limited to zero.

The correlation between \( I_1 \) and \( I_2 \) (or \( n_1 \) and \( n_2 \)) is graphically represented as the eccentricity of their joint probability density function, \( P(I_1,I_2) \) (or \( P(n_1,n_2) \)). The eccentricity, loosely defined as the elongation of the probability density function along the diagonal \( I_1 = I_2 \), increases with correlation. The eccentricity of \( P(I_1,I_2) \) is, by construction, uniquely determined by \( \rho \), so that for \( \rho = 0 \) the probability density function has a circular distribution, whereas as \( \rho \) approaches 1 the probability density function becomes increasingly confined to the diagonal. In contrast, the eccentricity of \( P(n_1,n_2) \) depends on both \( \rho \) and \( \mu \) (Fig. 4d, bottom row), as the following analysis using the threshold-linear model shows. The threshold, associated with \( f_i \) partitions the input plane \( (I_1,I_2) \) into four quadrants (Fig. 4d, middle column). In each quadrant, the transformation of \( P(I_1,I_2) \) into \( P(n_1,n_2) \) via \( f_i \) is different. When \( \mu \) is large, most of the mass of \( P(I_1,I_2) \) is contained in quadrant 1, so that \( P(I_1,I_2) \) is mapped approximately linearly to \( P(n_1,n_2) \). As a consequence, \( P(n_1,n_2) \) inherits much of the eccentricity of \( P(I_1,I_2) \) (Fig. 4d, bottom-left panel), implying \( \rho \approx \rho \). As \( \mu \) decreases, the mass of \( P(I_1,I_2) \) is shifted from quadrant 1 to the other three quadrants, and \( f_i \) distorts \( P(I_1,I_2) \) so that the eccentricity of \( P(n_1,n_2) \), and hence \( \rho \), decreases (Fig. 4d, middle and right panels of bottom row). Thus, as \( \mu \) is varied both \( \rho \) and \( \rho \) are expected to co-vary, yielding the correlation–rate relationship shown in Figs 1e, 2c and 4b. An analogous description can be made using the spiking LIF model (see Supplementary Information).

The above analysis shows that nonlinearities in the transfer function can cause different \( \psi \)-dependencies of \( \rho \) and allows us to explore the effect of other nonlinearities present in neurons. Phenomena such as spike refractoriness (see Fig. 6a of ref. 27), synaptic depression (see Fig. 2A of ref. 28), or dendritic sublinear summation (see Fig. 2 of ref. 29) impose a saturation on rate that would cause the \( \rho \)-\( \psi \) relationship to reach a maximum and begin to decrease (Fig. 4c, middle row). Dendritic super-linear summation (Fig. 4A of ref. 30 and Fig. 2 of ref. 29) introduces boosting effects that would yield small inflections in the \( \rho \)-\( \psi \) relationship (Fig. 4c, bottom row). These observations predict that single neuron/synaptic nonlinearities can be an important determinant of network correlation.

A relationship between firing rate and pairwise correlation has, until now, been elusive\(^{8,10,17,18}\). A consequence of the correlation–rate relationship is that, if the firing rate of a population of neurons is tuned to a certain feature (for example, orientation of a bar), the population correlation inherits the same tuning. Consistent with this prediction, \( \rho_{\psi} \) measured from neurons in primary visual cortex shows a tuning to stimulus orientation when \( T < 50 \text{ ms} \). A direct test of the correlation–rate relationship would require intracellular recordings where measurements of both the afferent current and spike train correlations, as a stimulus is varied, could be obtained\(^{24}\). Nevertheless, analysis of extracellular multi-unit recordings from macaque V1 (A. Kohn and M. Smith, personal communication) and electroseptory receptors (J. Middleton, J. Benda, A. Longtin and L. Maler, personal communication) shows a correlation–rate relationship similar to the one obtained in our network (Fig. 3c), supporting the generality of our result. The correlation–rate relationship prompts a re-examination of rate- and correlation-based coding hypotheses\(^{8,9,11–13,16,21,22}\), because these measures are mechanistically intertwined.

**METHODS SUMMARY**

Whole-cell somatic recordings were made from layer 5 neurons in slices (300 \( \mu \text{m} \)) from auditory and somatosensory cortices of young mice (postnatal day P14 to P24). Surgical, slicing and recording techniques were done as described previously\(^{16}\) and followed guidelines established by the NYU Animal Welfare Committee. Computer-generated currents (equation (1); duration \( L = 1 \text{ s} \)) were delivered to 1–4 neurons every 1–5 s for a total of \( N \) = 100 trials. The terms \( \xi_i(t) \) and \( \xi_j(t) \) were gaussian noises low-pass-filtered with a time constant of 5 ms. The spike count from the \( i \)th cell in the 4th trial, \( n_i^4(t) \), was the number of spikes that occurred in the temporal window \((t + T, T)\). Time was discretized with \( \Delta t = 1.2 \text{ ms} \). The spike count shift-corrected covariance was:

\[
\text{Cov}(n_i, n_j) = \frac{1}{N(L-T)} \sum_{m=1}^{N} (n_i^m(t) - \bar{n}_i^m)(n_j^{m+1}(t) - \bar{n}_j^{m+1}) 
\]

where the interior sum ranges over time bins. The variance was \( \text{Var}(n_i) = \text{Cov}(n_i, n_i) \) and the correlation coefficient \( \rho_{ij} \) was computed as in equation (2). In Fig. 1 we used \( T = 40 \text{ ms} \) (see Supplementary Information for analysis using \( T = 10–300 \text{ ms} \)).

We used a leaky integrate-and-fire (LIF) neural model\(^{23}\) which obeyed:

\[
\begin{align*}
\tau_m \frac{dI_m}{dt} &= -I_m + \mu + \sqrt{\tau_m(V_m - \xi_i(t)) + \xi_j(t)} \quad \text{where } V_m \text{ is the membrane potential of the } \text{th neuron}, \text{ and } \tau_m = 10 \text{ ms, threshold } = 20 \text{ mV and reset } = 0 \text{ mV. Here } \xi_i(t) \text{ and } \xi_j(t) \text{ are white noise currents. Correlation coefficients } \rho_{ij} \text{ defined as the limit of } \rho_{ij} \text{ as } T \text{ increases, were estimated from the ratio of the area of the cross-correlation function normalized by the areas of the auto-correlation functions (see Supplementary Information). Other parameters that differ from the experimental data analysis were } L = 100 \text{ s and } \Delta t = 0.5 \text{ ms. The network in Fig. 3 had } N_0 = 800 \text{ excitatory and } N_0 = 200 \text{ inhibitory cells in layer 1 randomly connected (probability } = 0.25) \text{ with 100 cells in layer 2. Layer 1 neurons fired Poisson trains with a rate of } 10 \text{ spikes } s^{-1}. \text{ Layer 2 neurons were LIF models with conductance-based excitatory and inhibitory synapses with time constants } \tau_e = 4 \text{ ms, } \tau_i = 8 \text{ ms, mean weights } \bar{w}_i = 0.32 \text{ nS, } \bar{w}_e = 1.4 \text{ nS and standard deviation } \Delta_2 = 0.35 \bar{w}_i (x = e, i). \end{align*}
\]

Full Methods and any associated references are available in the online version of the paper at www.nature.com/nature.

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METHODS

Experiments. Surgical, slicing and recording techniques were done as described previously and followed guidelines established by the NYU Animal Welfare Committee. Slices were made from auditory and somatosensory cortices of young mice (postnatal day P14 to P21). Whole-cell somatic recordings were made from layer 5 neurons identified under an infrared video microscopy. During recordings, slices (300 μm thick) were perfused at room temperature or at 32 °C with ACSF (with 95% O₂, 5% CO₂). Pipettes were filled with 100 mM K-glucuronate, 20 mM KCl, 10 mM phosphocreatine, 10 mM HEPES, 4 mM ATP-Mg, and 0.3 mM GTP at pH 7.3. Filled electrode resistances were in the range of 5 to 10 MΩ and recordings were performed under current-clamp conditions. Voltage and current signals were filtered at 10 kHz and digitized at 5 kHz.

Stimulus protocol. Computer-generated currents (equation (2)); duration L = 1 s after removing the initial 100 ms) were delivered to 1–4 neurons every 1–5 s. The common ξ(t) and the independent terms ξ_i(t) were gaussian noises low-pass filtered with a time constant of 5 ms. We presented each N = 100 trials stimulus block for 1–7 repetitions (see Supplementary Information for details).

Data analysis. Voltage traces were differentiatied and action potential times were detected by setting a threshold on the differential traces. The spike trains emitted by the jth cell in the ith trial were represented by a binary time series, y_i,j(t), which equaled 1 if there was a spike at time t and zero otherwise. The spike count, n(t), was simply the number of spikes that occurred in a window of length T; that is, n(t) = \sum_{t'=t}^{t+T} y_i,j(t'). Time was discretized with Δt = 1.2 ms resolution and the first 100 ms of the spike trains were removed to correct for spike frequency adaptation. The spike count shift-corrected covariance was computed as:

\[ \text{Cov}(n_i,n_j) = \frac{1}{N(L-T)} \sum_{t=1}^{L-T} \sum_{t'=0}^{T} n_i(t) n_j(t') - \frac{1}{N(L-T)} \sum_{t=1}^{L-T} \sum_{t'=0}^{T} n_i(t) n_j(t+\Delta t) \]  

where the interior sums range over time bins. Note that in the second term, named the shift-corrector, spike counts from consecutive trials were multiplied.

The auto-correlation function was obtained by making i = j in equation (7). Note that, as in the covariance, we have corrected for ‘chance correlations’ in C_{ii}(t) by subtracting the corresponding shift-corrector.

Computational model. We used a leaky integrate-and-fire (LIF) neural model integrating white noise currents:

\[ \tau_m \frac{dV_{i}}{dt} = -V_{i} + \mu + \sigma \sqrt{\tau_m} \left( \sqrt{1 - c(t)} + \sqrt{c(t)} \right) \]  

where \( V_i \) is the membrane potential of the ith neuron, and \( \tau_m = 10 \text{ ms} \) threshold = 20 mV and reset = 0 mV. Correlation coefficients \( \rho \) defined as the limit of \( \rho(c) \) as \( T \) increases, were estimated by computing the ratio of the area of the cross-correlation function, \( C_{ii}(t) \), normalized by the areas of the auto-correlation functions, \( C_{ii}(t) \) (see Supplementary Information). Other parameters in the simulations that differ from the data analysis were \( L = 100 \text{ s} \) and \( \Delta t = 0.5 \text{ ms} \). \( \rho \) in Fig. 2c was numerically obtained from a linear regression of \( \rho(c) \) in the interval \( c = 0–0.12 \). Parameter values in Fig. 2: \( \mu = 10, 14 \text{ and } 26 \text{ mV} \), \( \sigma = 1.3 \text{ mV} \) (panel a); \( \mu = 10 \text{ mV} \), \( \sigma = 1.3, 2.4 \text{ and } 8.8 \text{ mV} \) (panel b); \( \mu = 10–38 \text{ mV} \) and \( \sigma = 1.3 \text{ mV} \) (red in panel c); \( \mu = 10 \text{ mV} \) and \( \sigma = 1.3–15.8 \text{ mV} \) (blue in panel c). The theoretical curves in Fig. 2 were computed from standard first passage time formulae appropriate for a LIF neuron receiving white noise inputs (see Supplementary Information).

Network simulation. The network in Fig. 3 had \( N_E = 800 \) excitatory (E) and \( N_I = 200 \) inhibitory (I) cells in layer 1 and 100 cells in layer 2. Connections were randomly established from layer 1 to layer 2 with a probability \( P = 0.25 \). Layer 1 cells fired Poisson spike trains at rate \( \lambda = 10 \text{ spikes s}^{-1} \). Each neuron in layer 2 was a LIF neuron model with conductance-based E and I synapses described by:

\[ \frac{dC_{m}}{dt} = g_L (E_k - V) + g_L (E_k - V) + g_L (E_l - V) \]  

\[ \frac{d}{dt} = \frac{-g_{E}}{\tau_E} + \sum_{j} J_{kj} \sum_{k} \delta(t - t_{kj}) \]  

where \( \delta(t) \) are the times of the spikes emitted by the jth pre-synaptic cell and \( J_{kj} \) is the synaptic weight which was zero if the jth cell did not establish a connection or was randomly chosen from a gaussian distribution with mean \( f_k = 0.32 \text{ nS} \) (\( f_l = 1.4 \text{ nS} \)) and standard deviation \( \Delta \) (a = 0.35) if there was one. The synaptic time constants were \( \tau_E = 4 \text{ ms} \), \( \tau_I = 8 \text{ ms} \) resembling AMPA and GABA_A kinetics (see Supplementary Information for a slow synapses analysis). We also set threshold \( = -55 \text{ mV} \), reset \( = -65 \text{ mV} \), refractory period \( = 2 \text{ ms} \), membrane capacitance \( C_m = 0.12 \text{ nF} \), leak conductance \( g_L = 4.5 \text{ nS} \), excitatory reversal potential \( E_k = 0 \text{ mV} \), and inhibitory reversal potential \( E_l = -80 \text{ mV} \).