Self-sustained activity in balanced networks with low firing-rate

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Abstract

The brain can display self-sustained activity (SSA), which is persistent firing of neurons in the absence of external stimuli. This spontaneous activity shows low neuronal firing rates and is observed in diverse in vitro and in vivo situations. In this work, we study the influence of excitatory/inhibitory balance, connection density, and network size on the self-sustained activity of a neuronal network model. We build a random network of adaptive exponential integrate-and-fire (AdEx) neuron models connected through inhibitory and excitatory chemical synapses. The AdEx model mimics several behaviours of biological neurons, such as spike initiation, adaptation, and bursting patterns. In an excitation/inhibition balanced state, if the mean connection degree ($K$) is fixed, the firing rate does not depend on the network size ($N$), whereas for fixed $N$, the firing rate decreases when $K$ increases. However, for large $K$, SSA states can appear only for large $N$. We show the existence of SSA states with similar behaviours to those observed in experimental recordings, such as very low and irregular neuronal firing rates, and spike-train power spectra with slow fluctuations, only for balanced networks of large size.

Keywords: self-sustained activity, neuronal networks, irregular firing activity

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1. Introduction

Self-sustained activity (SSA), where neurons display persistent activity even in the absence of external stimuli (Greicius, 2003; Fox, 2005), is observed in diverse situations such as in vitro cortical culture and slice preparations (Plenz, 1996; Sanchez-Vives, 2000; Shu, 2003), in vivo cortical slab preparations (Timofeev et al., 2000), slow-wave sleep (Steriade, 2001), anesthesia (Steriade, 1993), and resting state (Arieli, 1995; Manti, 2003). Electrophysiological recordings of SSA states show irregular neuronal spiking, typically with low average frequencies of a few Hz but obeying long-tailed distributions (Hromádka, 2008; O’Connor, 2010; Buzsáki & Mizuseki, 2014). Many works have modeled neuronal networks with SSA by using random networks composed of excitatory and inhibitory leaky integrate-and-fire (LIF) neurons with external background input (Brunel, 2000; Vogels, 2005a; Parga, 2007; Kumar, 2008; Kriener, 2014; Ostojic, 2014). Other studies have considered networks with non-random architectures, composed of LIF neurons (Renart, 2007; Kaiser, 2010; Wang, 2011; Litwin, 2012; Potjans, 2014) or non-linear two-dimensional integrate-and-fire neuron models (Compte, 2004; Izhikevich, 2008; Destexhe, 2009; Stratton, 2010; Tomov, 2014; 2016). Both the architecture and the neuron types that comprise the network play an impor...
tant role in SSA states. SSA states are generated and maintained by recurrent interactions within networks of excitatory and inhibitory neurons. A stable SSA state is related to strong recurrent excitation within the neuronal network, which is restrained by inhibition to prevent runaway excitation. The balance between excitation and inhibition but also the connection density and connectivity are necessary to have SSA states. In our simulation (Borges, 2017). Here, we show some conditions in unstructured, sparsely connected networks of AdEx neurons can exhibit a transition from spiking to bursting synchronisation (Borges, 2017). Here, we show some conditions in that unstructured, sparsely connected networks of AdEx neurons can display low frequency self-sustained activity.

We show that not only the balance between excitation and inhibition but also the connection density and the network size are both important for low frequency self-sustained activity. In balanced networks, large values of mean node-degree connectivity are necessary to have low mean neuronal firing-rates, and for such values, large networks are necessary to have SSA states. In our simulations, we obtain similar results to the ones observed in experimental recordings, such as very low and irregular neuronal firing-rates ($\approx 1$ Hz) and spike-train power spectra with slow fluctuations (Litwin, 2012).

The paper is organised as follows: the introduction of the coupled AdEx network model were given in Section 2. In Section 3, we discuss the methodology for quantifying SSA in networks. Our results were presented in Section 4. Finally, the discussion and conclusion were given in Section 5.

2. Neuronal network model

We build a random neuronal network of $N$ neurons by connecting them with probability $p$, where $p$ is the probability that any two neurons in the network are connected, excluding autapses. The $N$ neurons in the network are split into excitatory and inhibitory neurons according to the ratio $4:1$, respectively. The connection probability $p$ and the mean connection degree $K$ of the network are associated by means of the relation

$$p = \frac{K}{(N-1)}. \quad (1)$$

The dynamics of each AdEx neuron $i = 1, \ldots, N$ in the network is given by (Destexhe, 2009)

$$C d V_i \frac{dt}{dt} = -g_L (V_i - E_L) + g_L \Delta_T \exp \left( \frac{V_i - V_T}{\Delta_T} \right) - \frac{1}{S} \left( w_i + \sum_{j=1}^{N} g_{ij} (V_i - E_j) + \Gamma_i \right), \quad (2)$$

$$\tau_w \frac{dw_i}{dt} = a (V_i - E_L) - w_i$$

where $V_i$ and $w_i$ are, respectively, the membrane potential and adaptation current of neuron $i$, $g_{ij}$ is the synaptic conductance of the synapse from neuron $j$ to neuron $i$, and $\Gamma_i$ is the external perturbation applied to neuron $i$. The meanings of the other parameters in Eq. (2) are defined in Table I. The synaptic conductance $g_{ij}$ has exponential decay with synaptic time-constant $\tau_s$. The values of the parameters were selected in order to reproduce the spiking characteristics of RS (excitatory) and FS (inhibitory) neurons (Destexhe, 2009). They are shown in Table I.

When the membrane potential of neuron $i$ is above a threshold potential ($V_i(t) > V_{\text{thres}} = 20$ mV), the neuron is assumed to generate a spike and the following update conditions are applied

$$V_i \rightarrow V_r = -60 \text{ mV}, \quad w_i \rightarrow w_i + b, \quad g_{ji} \rightarrow g_{ji} + g_s, \quad (3)$$

where $V_r$ is the reset potential. The update parameters $b$ and $g_s$ have different values for excitatory ($b = 0.01$ nA and $g_{\text{ex}}$) and inhibitory ($b = 0$ and $g_{\text{in}}$) neurons. The $g_{ji}$ updates have synaptic delays $1.5$ ms and $0.8$ ms for excitatory and inhibitory synapses, respectively. After the update, $g_{ji}$ decays exponentially with a fixed time constant $\tau_s$ (5 ms for excitatory and 10 ms for inhibitory synapses (Wang, 2011)). We define the relative inhibitory conductance $g = g_{\text{in}}/g_{\text{ex}}$ to be used as a parameter in the investigation of network dynamics. In each simulation, we apply external stimuli $\Gamma_i$ to $5\%$ of the $N$ neurons (randomly chosen) for $50$ ms to initiate network activity, and then we stop the external stimuli to observe the activity, which can be persistent (SSA) or transient. For each neuron $i$, the external stimulus $\Gamma_i$ has the same characteristics: it consists of excitatory current pulses with synaptic conductances that rise instantaneously to $0.01 \mu$S and decay exponentially afterwards with decay time $5$ ms, generated by a homogeneous Poisson process with rate $400$ Hz.

3. Methodology

3.1. Coefficient of variation

In this work, we use different statistical measures based either on time intervals or spike times. Those based on time intervals exploit the inter-spike interval (ISI) where the $m$th interval is defined as the difference between two
3.2. Firing-rate

We define the spike-train of neuron $i$ as the sum of delta functions (Gabbiani, 1998)

$$x_i(t) = \sum_{t_i^m} \delta(t - t_i^m),$$

where $[t_i^m]$ is the set of all spike times of neuron $i$ for $t \in [0, T]$. Based on Eq. (5), we calculate the mean firing rate of neuron $i$ over the time interval $[0, T]$ as

$$\bar{F}_i = \frac{1}{T} \int_0^T x_i(t) dt.$$  

For all neurons in the network, we define the time-varying network firing rate (in Hz) in intervals of 1 ms as

$$F(t) = \frac{1000}{N} \sum_{i=1}^{N} \left( \int_0^{t+1ms} \delta(t - t_i^m) dt \right),$$

and the mean network firing rate over the time interval $[0, T]$ as

$$\bar{F} = \frac{1000}{N} \sum_{i=1}^{N} \left( \frac{1}{T} \int_0^{T} \delta(t - t_i^m) dt \right) = \frac{1}{\langle\text{ISI}\rangle}. $$

where $\langle\text{ISI}\rangle$ is the mean ISI of the network given by

$$\langle\text{ISI}\rangle = \frac{1}{N} \sum_{i=1}^{N} \langle\text{ISI}_i\rangle. $$

3.3. Power spectrum

From the definition of spike-train in Eq. (5), we define the power spectrum of neuron $i$ by

$$S^{xx}_i(f) = \frac{\langle \tilde{x}_i(t) \tilde{x}_i^*(f) \rangle}{T},$$

where $\langle \cdot \rangle$ indicates ensemble average, $[0, T]$ is the time window of the simulation, $\tilde{x}_i(f)$ is the Fourier transform of neuron $i$, given by $\tilde{x}_i(f) = \int_0^T e^{2\pi if t} x_i(t) dt$, and $\tilde{x}_i^*(f)$ is the complex conjugate of $\tilde{x}_i(f)$. The power spectrum of a set of $M$ ($M \leq N$) neurons, $S^{xx}(f)$ is then defined as the average power spectrum of these $M$ neurons

$$\bar{S}^{xx}(f) = \frac{1}{M} \sum_{i=1}^{M} S^{xx}_i(f).$$

In order to keep consistency over the different simulations (see Fig. 5), we choose $M = 5 \times 10^4$ independently of the values of $N$ and $K$. Here, we will use two measures related to $S^{xx}$ to describe spike-train characteristics (Lindner, 2004; Grün, 2010; Wieland, 2015; Pena, 2018).

The first one is the Fano factor $FF = \langle \Delta n^2 \rangle / \langle n \rangle$, which is defined as the ratio of the variance to the mean of the spike count of the $M$ neurons over the time window $[0, T]$, $n = \sum_{i=1}^{M} \int_0^T dx_i(t)$. Its relation to $S^{xx}$ is meaningful in the vanishing frequency limit of $S^{xx}(f)$, i.e., $\lim_{f \rightarrow 0} S^{xx}(f) = F \cdot FF$. The Fano factor $FF$ is a standard measure of neuronal variability ($FF = 1$ for a Poisson process) and is related to the CV of the ISIs of the $M$ neurons (Cox, 1966) by $\lim_{f \rightarrow 0} S^{xx}(f) = F \cdot CV^2 (1 + 2 \sum_{k=1}^{N} r_k)$, where $r_k$ is the serial correlation coefficient between ISIs that are lagged by $k$.

The second one is the mean firing-rate of the $M$ neurons, $F_M$ (defined as in Eq. 6 but dividing by $M$), which is related to $\bar{S}^{xx}(f)$ by its limit to infinity, i.e. $\lim_{f \rightarrow \infty} \bar{S}^{xx}(f) = \bar{F}_M$.

3.4. Synaptic input

The instantaneous synaptic conductance of neuron $i$ due to all excitatory and inhibitory synapses arriving to it will be written here as

$$G_i(t) = \sum_{j \in \mathcal{E}} g_{ej}(t) + \sum_{j \in \mathcal{I}} g_{ij}(t) = G_{ex,i}(t) + G_{in,i}(t),$$

where $\mathcal{E}$ and $\mathcal{I}$ are the sets of all excitatory and inhibitory neurons in the network, respectively.
The instantaneous synaptic input to neuron $i$ will be written as

$$ I_{syn,i}(t) = \sum_{j \in \mathcal{E}} g_{ij}(E_j - V_i) + \sum_{j \not\in \mathcal{E}} g_{ij}(E_j - V_i) $$

$$ = I_{syn,i}^E(t) + I_{syn,i}^I(t), $$

and the mean synaptic input $I_{syn}$ to all neurons in the network over simulation time $[0, T]$ as

$$ \bar{I}_{syn} = \frac{1}{N} \sum_{i=1}^{N} \frac{1}{T} \int_{t=0}^{T} (I_{syn,i}^E(t) + I_{syn,i}^I(t)) \, dt. $$

### 3.5. Network decay-time

Finally, for a given neuron $i$ we define the time of its last spike as the maximum time in its spike train

$$ t_{last}^{i} = \max \{ t_{m}^{i} \}. $$

The network decay-time (DT) is defined as the last spike in the network during simulation time, i.e. the maximum $t_{last}^{i}$ comparing all neurons in the network,

$$ DT = \max \{ t_{last}^{i}, i = 1, \ldots, N \}. $$

### 3.6. Implementation details

All simulations were implemented in C. The ordinary differential equations were integrated by the fourth order Runge-Kutta method with step size 0.01 ms.

### 3.7. Experimental details

#### 3.7.1. Animals

Electrophysiological experiments were conducted using male Wistar rats with 20-25 postnatal days. All animals were kept cycles in an animal facility in a 12:12h lightdark cycle with temperature adjusted for 23°C ± 2°C with free access to food and water. All procedures were approved by the Institutional Animal Care Committee of the Institute of Biomedical Sciences, University of São Paulo (CEUA ICB/USP n. 090, fls. 1°).

#### 3.7.2. Brain slices preparation

After animals were deeply anesthetised through isoflurane inhalation (AErrane; Baxter Pharmaceuticals) they were decapitated and the brain was quickly removed and submerged in cooled (0°C) oxygenated (5% CO$_2$ - 95% O$_2$) cutting solution (in mM): 206 sucrose, 25 NaHCO$_3$, 2.5 KCl, 10 MgSO$_4$, 1.25 Na$_2$PO$_4$, 0.5 CaCl$_2$, and 11 D-glucose. After removing the cerebellum, brain hemispheres were separated by a single sagittal cut. Both brain hemispheres were trimmed up and glued in a metal platform and sectioned using a vibratome (Leica - VT1200). 350-400 μm brain slices were obtained by advancing the vibratome blade from anterior-posterior orientation. Slices were rapidly transferred to a holding chamber containing artificial cerebrospinal fluid (ACSF; in mM): 125 NaCl, 25 NaHCO$_3$, 3 KCl, 1.25 Na$_2$PO$_4$, 1 MgCl$_2$, 2 CaCl$_2$, and 25 D-glucose. Slices were kept oxygenated at room temperature (20-25°C) for at least one hour before proceeding with electrophysiological recordings.

#### 3.7.3. Electrophysiological recordings

Brain slices containing the hippocampal formation were placed in a submersion-type recording chamber upon a modified microscope stage and maintained at 30°C with constant perfusion of oxygenated ACSF (5% CO$_2$-95% O$_2$). Whole-cell recordings were made from neurons located in the pyramidal layer of CA1. Recording pipettes were fabricated from borosilicate glass (Garner Glass) with input resistances of ~ 4-6 MΩ and were filled with intracellular solution (in mM): 135 K-gluconate, 7 NaCl, 10 HEPES, 2 Na$_2$ATP, 0.3 Na$_3$GTP, 2 MgCl$_2$; at a pH of 7.3 obtained with KOH and osmolality of 290 mOsm. All experiments were performed using a visualised slice setup under a differential interference contrast-equipped Nikon Eclipse E600FN microscope. Recordings were made by using a Multiclamp 700B amplifier and pClamp software (Axon Instruments). Only recordings from cells that presented spontaneous activity with membrane potentials lower than -60 mV, access resistance lower than 20 MΩ, and input resistance higher than 100 MΩ and lower than 1000 MΩ were included in our data. We injected depolarising currents to identify regular, tonic, or bursting spike patterns. Neuronal spontaneous activity was assessed by 10 minutes of continuous recordings in current clamp mode.

Figure 1: Intracellular recordings demonstrating a high variability of spontaneous activity pattern. Twenty seconds of recordings are shown (horizontal black bar denotes 1 s and vertical black bar denotes 50 mV).

### 4. Self-sustained activity

SSA assessed by electrophysiological recordings in physiological brain states is characterised by irregular neuronal spiking, normally with low average frequency fitted in long-tailed distribution (Buzsáki & Mizuseki, 2014). Interestingly, some brain regions are able to produce spontaneous network activity after slicing procedure including hippocampal sharp waves (Maier, 2003; Giannopoulos, 2013; Bazelolet, 2016). Once hippocampus slices present...
SSA represented by spontaneous activity, we used CA1 neurons whole cell recording to demonstrate the possible variability of firing rate pattern observed in the brain. Our intracellular recordings demonstrated a high variability of spontaneous activity pattern including low neuronal firing-rates and small bursts activity in distinct recorded neurons. In Fig. 1 we show a representation of five traces obtained by whole-cell patch clamp from CA1 neurons for 20 seconds. Recorded neurons presented distinct firing pattern including very low firing (0.018 Hz) to small bursts of spikes (see the second and last trace in Fig. 1). In the whole record (600 s), the mean firing rate over the five neurons is \( \approx 1.172 \text{ Hz} \) (0.028, 0.157, 3.403, 0.018, 2.252). Similar firing patterns, with high variability and low neuronal firing-rates, are found in different recordings in the hippocampus and cortex in the rat brain during slow-wave sleep [Mizuseki 2013], and recordings of human middle temporal gyrus during sleep [Pevrache 2012].

In order to reproduce SSA firing patterns with low neuronal firing-rates, we analyse the parameter space \( g_{ex} \times g \) for a neuronal network of \( N = 10^4 \) neurons and connection probability \( p = 0.02 \). We focus on an area of parameter space where we found a balanced regime between excitation and inhibition. In this area we do not observe SSA for \( g_{ex} < 0.004 \mu \text{S} \), which is the weak coupling region. Examples of time-dependent network firing rates in this region are shown in Fig. 2(a), for \( g_{ex} = 0.0035 \mu \text{S} \) and \( g = 16 \). The initial stimulus applied in the first 50 ms generates short-lived activity in the network. The mean synaptic input to the network neurons \( I_{syn} \) is essentially negative (left-hand inset in Fig. 2(a)) and the network decay-time DT is always less than 3 s (right-hand inset in Fig. 2(a)). Examples of time-dependent network firing rates in the region of SSA are shown in Figure 2(b), corresponding to parameters \( g_{ex} = 0.008 \mu \text{S} \) and \( g = 16 \). The mean synaptic input is approximately balanced (\( I_{syn} \approx 0 \)) and the decay-time is higher than the maximum time used in our simulations (\( DT \geq 5 \text{ s} \)).

For \( N = 10^4 \) neurons, the lowest mean network firing rates are approximately 2 Hz. The region where these rates occur is shown in purple in Fig. 2(c). A particular case for \( g_{ex} < 0.003 \mu \text{S} \) is indicated by a turquoise square, where the activity is not self-sustained. For the random network and parameter space considered, the region with SSA is roughly determined by \( g_{ex} > 0.004 \mu \text{S} \) (indicated in yellow in Fig. 2(e)). The lowest mean network firing rate of an SSA state is around 4 Hz (red region in Fig. 2(c)). Within this region, one can identify the

Figure 2: (Colour online) Self-sustained activity (SSA) emerges due to the excitatory/inhibitory balance in the network. Time-dependent network firing rate \( F \) in two different regimes: (a) non-SSA and (b) SSA. Each trace corresponds to a different simulation. The two neuronal networks have the same set of parameters except that the excitatory conductance is \( g_{ex} \).

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region of excitation/inhibition balance by considering the region with $I_{\text{syn}} \approx 0$ (see Eq. (14)). This is shown in red in Fig. 2(d). The black and yellow regions in Fig. 2(d) correspond to slightly predominant inhibitory and excitatory mean synaptic input, respectively. A SSA case with $\bar{F} \approx 4$ Hz and excitation/inhibition balance is indicated by the green circle in Figs. 2(c)-(e) and corresponds to $g_{\text{ex}} = 0.008 \, \mu S$ and $g = 16$.

To study the effect of the size of the network on SSA, we considered two networks of different sizes but same mean degree. The first has $N = 10^4$ and $p = 0.04$ and the second $N = 2 \times 10^4$ and $p = 0.02$, with both having $K = 400$. The parameters ($g_{\text{ex}} = 0.005 \, \mu S$ and $g = 8$) are the same for the two networks, and put them close to the balanced state. In Fig. 3(a), we show the time evolution of the firing rates of the two networks. For $N = 10^4$ (turquoise line), the activity decays before 2 s, and for $N = 2 \times 10^4$ (green line) SSA is observed. The distributions of $I_{\text{syn}}$ for the two cases (see the insets in Fig. 3(a)) have similar (positive) average values but the one for $N = 10^4$ is broader and left-skewed. The decay-time distribution (Figs. 3(b), (c)) clearly shows that the networks with $N = 2 \times 10^4$ have SSA, while the ones with $N = 10^4$ are predominantly short-lived. A comparison of networks with the two sizes in the $g_{\text{ex}} \times g$ parameter space is shown in Figs. 3(d)-(i). The mean network firing rate and mean synaptic input display similar behaviour in the parameter space for the two network sizes (Figs. 3(d)-(g)). However, this similarity is not seen in the diagram for DT (Figs. 3(h)-(i)). The region corresponding to SSA (yellow in the diagrams) is larger for $N = 2 \times 10^4$ than for $N = 10^4$. Moreover, the shape of this region for $N = 2 \times 10^4$ discloses almost absent sensitivity of SSA duration to the relative inhibitory synaptic conductance $g$. On the other hand, for $N = 10^4$ the SSA lifetime is sensitive to $g$ for $0.006 \, \mu S \lesssim g_{\text{ex}} \lesssim 0.008 \, \mu S$, and only for strong coupling ($g_{\text{ex}} \gg 0.008 \, \mu S$), it becomes insensitive to $g$.

Next, we investigate the influence of the network parameters $N$, $p$, and $K$ (Eq. (11)) on the network firing rate of SSA states. In order to do so, we fix $g_{\text{ex}} = 0.008 \, \mu S$ and $g = 16$ to keep the network around excitatory/inhibitory balance. Figure 4 shows the mean network firing rate of SSA states (colour scale) in the parameter space $N \times p$. Moreover, the white area in the diagram corresponds to non-SSA states. We can see that the mean network firing rate of SSA states depends on both $N$ and $p$. In particular, low firing rate SSA states appear when the network size $N$ increases. The black solid line in Fig. 4 represents networks with mean node-degree $K = 1500$, in which case there are SSA states for $N \geq 1.5 \times 10^4$ neurons. The inset in Fig. 4(a) shows the dependence of the mean network firing rate $\bar{F}$ of SSA states on the mean connection node-degree $K$ for constant $N$. We can observe that lower rates are obtained as $K$ increases. However, very low rates ($\approx$
1 Hz) are present only for very large network sizes. In the inset in Fig. 4(b), we can observe that the network size does not alter the mean network firing rate when $K$ is kept constant. Therefore, large $K$ plays an important role in the occurrence of low network firing rates, and for such low rates, large networks are necessary to support SSA states.

Figure 5 displays statistical results for three different combinations of network size and node-degree, namely $(N = 10^5; K = 1300), (N = 3 \times 10^5; K = 1500)$, and $(N = 5 \times 10^5; K = 1700)$. The time-varying network firing rate is non-periodic and its mean value decreases as both $N$ and $K$ increase (Fig. 5(a)). In the three cases, the spiking variability, characterised by the ISI distribution, is very well described by a Poisson distribution (Figs. 5(c)-(e)).

Moreover, as the network size $N$ increases, $FF$ decreases, indicating tendency to converge to a Poisson process and is linked to a standard irregularity measure and CV. We see that in the limit of very small frequencies, the value of the power spectra decreases as both $K$ and $N$ increases, confirming the behaviour observed by the CV, i.e. the spiking times are becoming more regular. The power spectra display slow fluctuations, which can be explained by the low neuronal firing rates and bursting spiking patterns (as discussed below). Slow power spectrum fluctuations are also a feature of spontaneous activity in cortical networks (Mantini, 2007; Wieland, 2013; Mastrogiuseppe, 2017).

In Fig. 6 we show characteristics of the SSA firing patterns with low rate exhibited by the network (a particular case with $N = 5 \times 10^5$ and $K = 1700$ is shown as a representative example). The patterns have sparse and non-synchronous activity (Fig. 6(a)), akin to what has been termed the heterogeneous variant (Ostojic, 2014) of the asynchronous irregular (AI) regime (Brunel, 2000; Vogels, 2005). In the homogeneous AI regime, all neurons fire with the same mean rate, but in the heterogeneous AI regime, the mean firing rates fluctuate in time and across neurons. In some cases, neurons can even exhibit bursting periods (as can be seen in Fig. 6(b)). Neurons have low firing rates, with a right-skewed distribution that peaks around 1 Hz (Fig. 6(d)). Irregularity is confirmed by the CV of ISIs and power spectra analysis (Fig. 6). The similarity between the firing regime observed in our simulations and the heterogeneous AI state can be further verified by a comparison of the respective spike-train power spectra. Spectra for networks in heterogeneous AI states have been calculated elsewhere (Penal, 2018) and display slow fluctuations as shown here (Fig. 5(b)).

The distributions over neurons of the excitatory ($G_{ex}$) and inhibitory ($G_{in}$) synaptic conductances (panels (e) and (f) in Fig. 6) have nearly symmetric shapes, with the $G_{in}$ distribution slightly right skewed, according to experimental evidence (Rudolph, 2007), though the conductance values are higher than in the experimental recordings. The distributions of excitatory ($I_{syn}^{ex}$) and inhibitory ($I_{syn}^{in}$) synaptic inputs are nearly identical and vary over nearly the same range of absolute values, though the upper end of the range is slightly higher for the inhibitory synaptic inputs (panels (g) and (h) in Fig. 6). This is a hallmark of a balanced state, and the neuronal spikes happen due to the large synaptic conductance variability. This
allows necessary conditions for the appearance of SSA.

5. Conclusion

In this paper, motivated by the self-sustained activity that is observed in the brain in the absence of external stimuli, we sought to study necessary conditions by which irregular and low-frequency self-sustained dynamics emerge in neuronal networks with random connectivity. We build neuronal networks of excitatory (80%) and inhibitory (20%) neurons where the neurons are randomly connected through chemical synapses and are mathematically described by the adaptive exponential integrate-and-fire model. This model mimics the behaviour of biological neurons, exhibiting spiking and bursting patterns of activity. We studied the network features by varying (i) the balance between excitation and inhibition, and (ii) the topological characteristics of the network. Results were obtained by running a large number of simulations in order to obtain valid statistical results and their firing rates.

Our results allowed us to observe neural activity with slow fluctuations in the absence of external perturbation. We found that the pattern of low firing-rate self-sustained activity is asynchronous and irregular as depicted in raster plots. We showed that the irregular spikes with low rate depend on the mean node degrees of the neurons, and that low-rate self-sustained activity occurs for a tight balance between inhibition and excitation and large network sizes. When the connection mean degree \( K \) is fixed, the mean network firing rate does not change with the network size \( N \). In an excitation/inhibition balanced network, if \( N \) is fixed, the mean network firing rate decreases when \( K \) increases. However, there is a maximum \( K \) value for which it is possible to maintain SSA states, and this value is proportional to the network size. Therefore, large networks are necessary to have SSA states when \( K \) is large. Moreover, the spikes are due to the synaptic-conductance variability, and the inhibitory synaptic input is slightly higher than the excitatory. Our simulation results resemble those obtained from experimental recordings, such as irregular firing with network firing rate approximately equal to 1 Hz.

Finally, we have been able to verify the existence of low neuronal firing-rate SSA in the balanced network. This phenomenon is characterised by irregular network oscillations and shows that low frequency self-sustained activity can be found in large balanced random networks.

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Figure 6: (Colour online) Characteristics of low firing-rate SSA states. All plots refer to a single simulation of a network with $N = 5 \times 10^5$, $K = 1700$, and parameters $g_{ex} = 0.008 \, \mu S$ and $g = 16$. (a) Raster plot of 2 s of simulation time (only 500 randomly chosen neurons from the network are shown). (b) membrane potential of three randomly chosen neurons from the network (horizontal black bar denotes 0.2 s and vertical black bar denotes 50 mV). (c) Time-varying synaptic conductances ($G_{ex,i}(t)$ in green and $G_{in,i}(t)$ in cyan) of neurons represented by blue line in (b). (d) distribution of mean firing rates $F_i$ (Eq. (6)) of all neurons in the network. Distribution of excitatory ($G_{ex,i}$) (e) and inhibitory ($G_{in,i}$) (f) synaptic conductances of all network neurons at the end of the simulation. Distribution of excitatory ($I_{syn,i}^E$) (g) and inhibitory ($I_{syn,i}^I$) (h) synaptic inputs of all network neurons at the end of the simulation.
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