Introduction to neural dynamics

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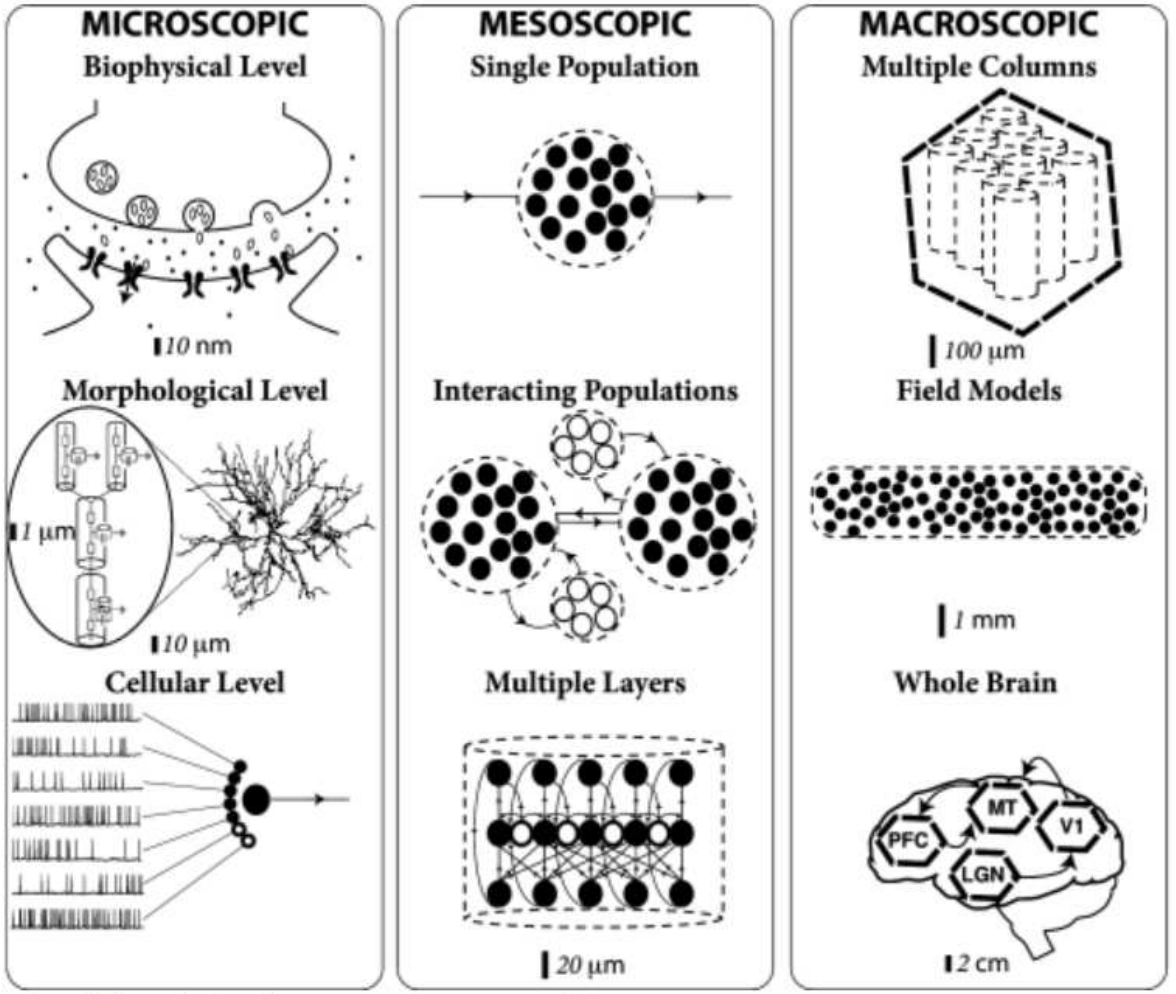
Summary

- Neuronal Populations
- Columnar Organization
- Homogeneous Population of LIF Neurons
- Heterogeneous Populations
- Connectivity Schemes
  - Fully Coupled Networks
  - Random Coupling : Massive versus Sparse
- Balanced Networks
- Dynamical Balance
- Experimental Verifications

Neuronal Populations



The brain contains millions of neurons which are organized in different brain areas, within a brain area in different subregions, inside each small region into different layers, inside each layer into various cell types.

Neuronal Populations

- Suppose a subject receives a visual, auditory, or somatosensory stimulus.
- What is the activity of all the cells in this layer of this subregion that are of type ‘pyramidal’ in response to the stimulus?
- What is the response of this subregion as a whole?
- What is the response of a brain area?
- In other words, at any of the scales of spatial resolution, we may be interested in the response of the neuronal population as a whole, rather than in the spikes of individual neurons.

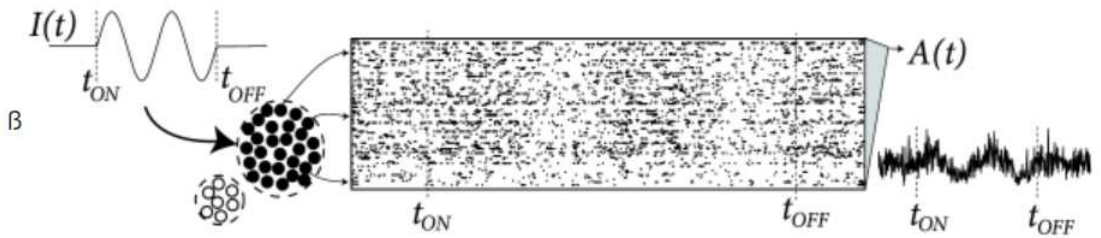


Fig. 12.2: Input and output of a population. A signal $I(t)$, represented by a sinusoidal modulation of the input starting at t_{ON} and ending at t_{OFF}, stimulates the population of 8 000 excitatory neurons in a randomly coupled network of 8 000 excitatory and 2 000 inhibitory neurons (left). Each neuron produces a spike train (middle) illustrated here by lines of dots, each dot corresponding to a spike. Only 1% of the population is shown. The population activity $A(t)$ (right) counts spikes in time bins of 1ms averaged over the 8 000 excitatory neurons.

Columnar Organization

The cortex in the brain is organized in cortical columns, these are organised in cortical layers : a plausible biological candidate of a neuronal population is a group of neurons of the same type in one layer of a cortical column .

Receptive fields

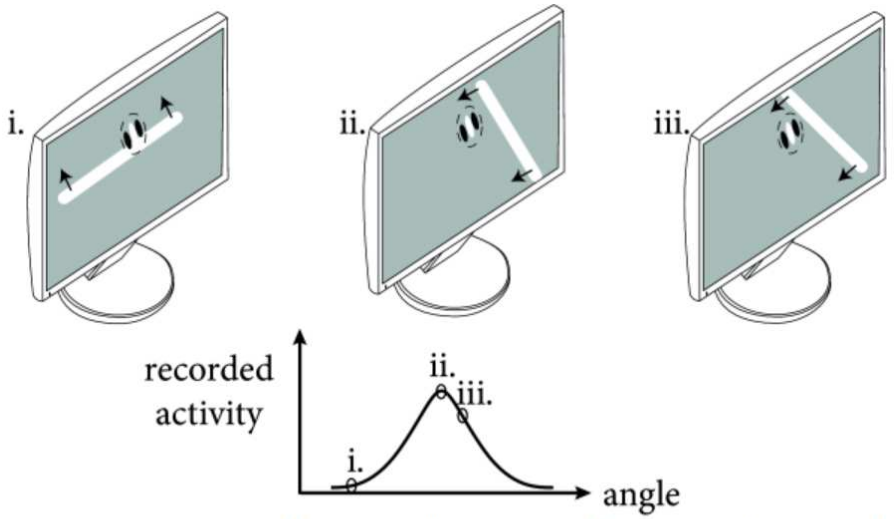


Fig. 12.3: Orientation tuning. The receptive fields of simple cells in visual cortex have positive and negative subfields. To test orientation tuning, a light bar is slowly moved across the screen (i). The neuron responds maximally if the light bar with an orientation aligned with that of the receptive field moves into the positive subfield (ii) and responds slightly less, if the orientation of the bar is not optimal (iii). The response as a function of the orientation of the bar is shown at the bottom (schematic figure).

- Neurons in sensory cortices can be experimentally characterized by the stimuli to which they exhibit a strong response.
- The neuron responds maximally to a moving light bar with an certain orientation aligned with the elongation of the positive subfield.
- If the orientation of the stimulus changes, the activity of the cell decreases.

Columnar Organization

Cortical Columns

Neighboring neurons in visual cortex have similar receptive fields.

- If the experimentalist moves the electrode vertically down from the cortical surface to deeper layers, the location of the receptive field and its preferred orientation does not change substantially.
- If the electrode is moved to a neighboring location in cortex, the location and preferred orientation of the receptive field of neurons at the new location changes only slightly compared to the receptive fields at the previous location.
- This observation has led to the idea that cortical cells can be grouped into 'columns' of neurons with similar properties.
- Each column contains several thousand neurons with similar receptive fields

In other sensory cortices:

- In the auditory cortex neurons can be characterized by stimulation with pure tunes. Each neuron has its preferred tone frequency and neighboring neurons have similar preferences.
- In the somatosensory cortex neurons that respond strongly to touch on, e.g., the index finger are located close to each other.

Columnar Organization

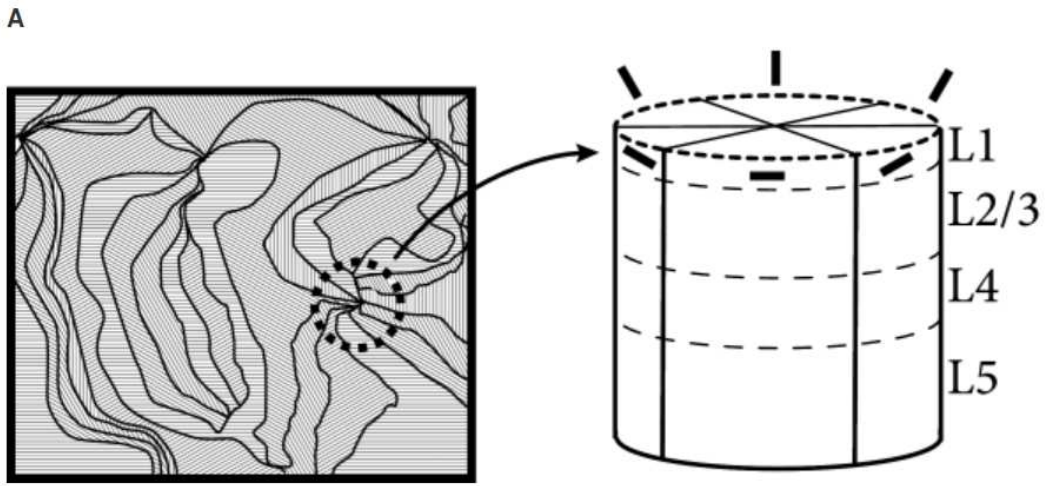


Fig. 12.4: Orientation maps and columns. **A**. Top view onto the surface of visual cortex. Neurons that are optimally activated by a moving grating with an orientation of, e.g., 60^o, form bands. Direction of hash-line texture in the image indicates the preferred orientation. Iso-orientation contour lines converge to form pinwheels. One of the pinwheels is highlighted by the dashed circle. **B**. Side view of a pinwheel (dashed circle in A). Orientation selectivity is indicated by thick bars. Neurons with the same orientation form vertical columns; schematic representation following experimental data shown in Bressloff and Cowan (65).

- Inside a column neurons are organized in different layers.
- Each layer contains one or several types of neurons: excitatory and inhibitory neurons (different types of inhibitory interneurons)
- The concept of cortical columns suggests that localized populations of neurons can be grouped together into populations, where each population (e.g., the excitatory neurons in layer 4) can be considered as a homogeneous group of neurons with similar intrinsic properties and similar receptive fields.

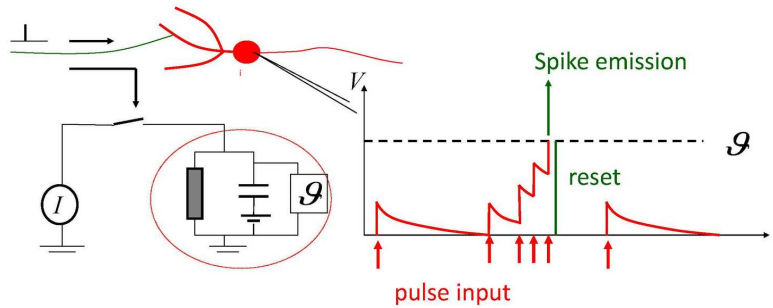
Homogeneous Population of LIF neurons

For a Homogeneous population of N Leaky Integrate-and-Fire neurons the sub-threshold evolution is given by

$$\tau_m \frac{du}{dt} = -u + RI_i(t) \quad \text{for} \quad u_i < \theta \qquad i = 1, \ldots, N$$

Whenever the neuron goes supra-threshold i.e. $u_i > \theta$

- a spike is emitted and transmitted to all the connected neurons
- u_i is resetted to u_r



Homogeneous population : all neurons have the same input resistance R, the same membrane time constant τ_m, the same threshold θ and same reset potential u_r

We assume that a neuron is coupled to all others with coupling strength w_0. The input current is

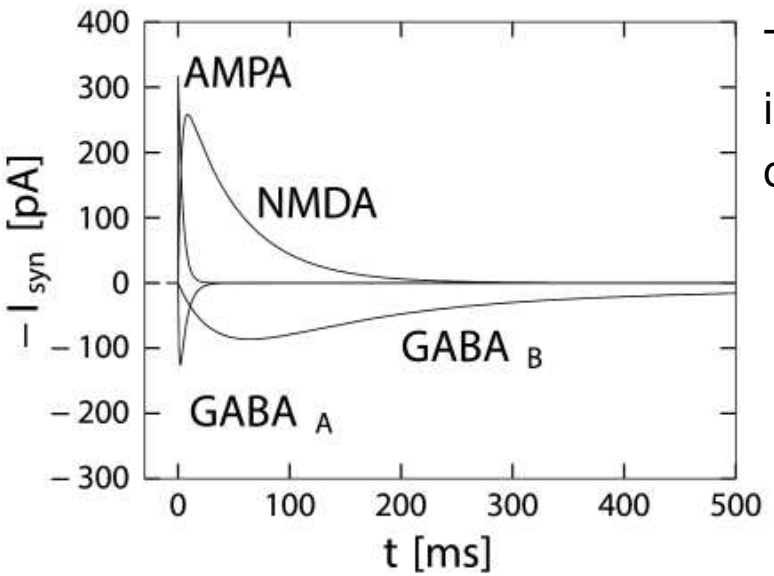
$$I_i(t) = w_0 \sum_{j=1}^{N} \sum_f \alpha(t - t_j^{(f)}) + I_{ext}(t)$$

Synaptic current

$$I_i(t) = I_{syn}(t) + I_{ext}(t) = w_0 \sum_{j=1}^{N} \sum_f \alpha(t - t_j^{(f)}) + I_{ext}(t)$$

The input current to neuron i has 2 components:

- an external drive
- a synaptic current that is the superposition of all the post-synaptic currents (PSCs) $\alpha(t - t_j^{(f)})$ received from all the neurons at previous times $t_j^{(f)}$



The PSCs have different time durations depending on the aminoacid neurotrasmitters in the chemical synapses:

- $w_0 > 0$ excitatory :
  AMPA (2-5 ms) and NMDA (40-100 ms)
- $w_0 < 0$ inhibitory :
  GABA_A (6-7 ms) and GABA_B (100-400 ms)

Population Activity

Due to the all-to-all coupling the synaptic current is the same for all neurons and it can be writtent in terms of the population activity

$$A(t) = \lim_{\Delta t \to 0} \frac{1}{\Delta t} \frac{n_{act}(t, t+\Delta t)}{N} = \frac{1}{N} \sum_{j=1}^{N} \sum_{f} \delta(t - t_j^{(f)})$$

which is a the population average of all the spikes emitted in the network within a time bin Δt.

Therefore the synaptic current is given by the convolution of the population activity A and of the specific PSC α

$$I_{syn}(t) = w_0 N \int_0^\infty \alpha(s) A(t-s) ds = \sum_{j=1}^{N} \sum_{f} \int_0^\infty \alpha(s) \delta(t - t_j^{(f)} - s) ds = \sum_{j=1}^{N} \sum_{f} \alpha(t - t_j^{(f)})$$

The important thing to notice is that $I_{syn}(t)$ grows with the number of neurons N

- A possibility to avoid this is to rescale the synaptic coupling $w_0 = a/N$: a sort of synaptic homeostatic mechanism when neurons receive too much inputs

Turrigiano, G. Annual review of neuroscience 34 (2011) 89.

Heterogeneous Population

Each neuron in a population can fire in a different manner to the same external drive I for two reasons:

- different values of the neuron parameters (excitability, threshold tec)
- different pre-synaptic neurons - different connectivity;

The parameters θ_i of each neuron are different, therefore

$$\nu_i = g_{\theta_i}(I) \qquad i = 1, \ldots, N$$

For a symmetric distribution $P(\theta_i)$ of the parameters (e.g. a Gaussian ditribution) , we can write

$$< \nu >= \frac{\sum_i \nu_i}{N} \simeq g_{\bar{\theta}}(I) + \frac{d^2 g}{d\theta^2}|_{\bar{\theta}}(\theta_i - \bar{\theta})^2 + \ldots$$

where $\bar{\theta}$ is the average value of the parameters.

For a smooth distribution of heterogeneitis and if the distribution is sufficiently narrow we can neglect the second term and higher order terms.

Connectivity Schemes

- The real connectivity between cortical neurons of different types and different layers, or within groups of neurons of the same type and the same layer is still partially unknown.
- At most, some plausible estimates of connection probabilities exist.
- In simulations of spiking neurons, there are a few coupling schemes that are frequently adopted.

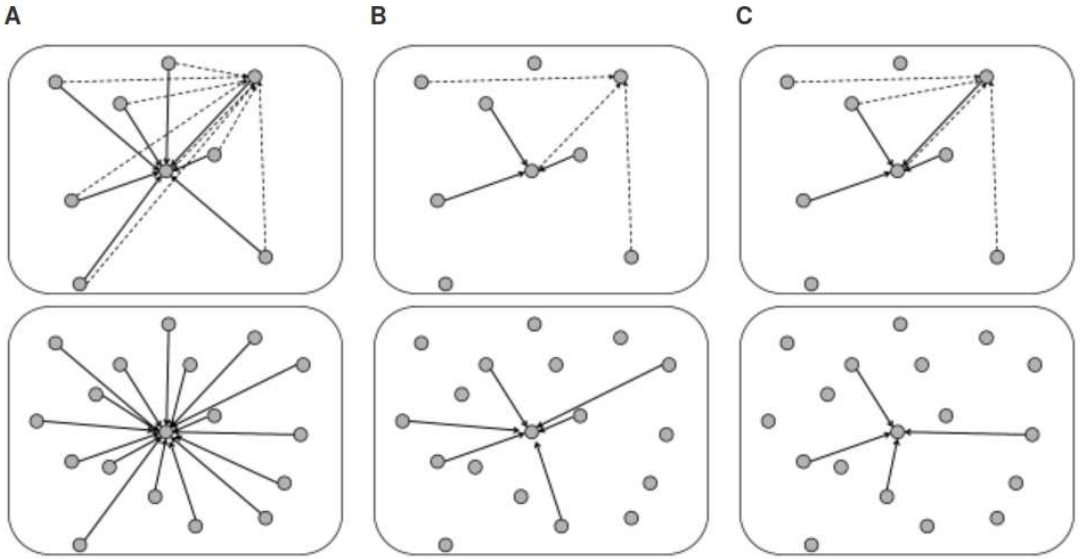


Fig. 12.6: Coupling Schemes. **A**. Full connectivity: Top: A network of 9 neurons with all-to-all coupling. The input links are shown for two representative neurons. Self-couplings are not indicated. Bottom: The number of input links (indicated for one representative neuron) increases, if the size of the network is doubled. **B**. Random coupling with fixed connection probability. In a network of 18 neurons (bottom) the number of input links is larger than in a network of 9 neurons (top). **C**. Random coupling with fixed number of inputs. The number of links from presynaptic neurons (top: input links to two representative neurons) does not change when the size of the network is increased (bottom: input links to one representative neuron).

Scaling Behaviour

- For each choice of the coupling scheme we should discuss how to rescale the synaptic coupling w_0 with the number of neurons.
- This because if I double the number of neurons N in a fully coupled or randomly coupled network with fixed probabilility the synaptic input to a a neuron will double and its property will change with the number of neurons N
- From a simulation and physical point of view I want that a certain network with $N = 1000$ or $N = 100000$ neurons will have the same properties, to obtain this I should properly rescale the coupling
- Real populations of neurons have a fixed size because, e.g., the number of neurons in a given cortical column is given and, at least in adulthood, does not change dramatically from one day to the next. Typical numbers, counted in one column of mouse somatosensory cortex (barrel cortex, C2) are 5750 excitatory and 750 inhibitory neurons.
- Tipically one has 80 % excitatory neurons and 20 % inhibitory ones

Full Connectivity

All neuron connected with all the others with constant synaptic coupling w_0:

$$\tau_m \frac{du}{dt} = -u + w_0 \sum_{j=1}^{N} \sum_f \delta(t - t_j^{(f)}) + I_{ext} = -u + w_0 N < \nu >_N + I_{ext} \qquad i = 1, \ldots, N$$

where the mean firing rate is given by

$$< \nu >_N = \frac{1}{N} \sum_{i=1}^{N} \nu_i \longrightarrow \nu_0 \quad \text{for} \quad N \to \infty$$

The synaptic current $I_{syn} = w_0 N < \nu >_N$ should not diverge for large N, therefore an appropriate scaling is

$$w_0 = \frac{J_0}{N}$$

and the current becomes

$$I_{syn} = J_0 < \nu >_N \longrightarrow J_0 \nu_0 \quad \text{for} \quad N \to \infty$$

Full Connectivity - Finite Size Fluctuations

For finite N the synaptic current can present finite size fluctuations.

The Central Limit Theorem says that the averages $< \nu >_N$ are Gaussian distributed

$$P(< \nu >_N) = \frac{1}{\sigma\sqrt{2\pi}} \mathrm{e}^{-\frac{1}{2}(\frac{<\nu>_N - \nu_0}{\sigma})^2}$$

with mean ν_0 and standard deviation $\sigma \propto \frac{1}{\sqrt{N}}$

Therefore also the synaptic currents $I_{syn} = J_0 < \nu >_N$ are Gaussian distributed with mean $J_0\nu_0$ and standard deviation $\sigma_I \propto \frac{J_0}{\sqrt{N}}$

Due this fluctuations even for constant I_{ext} the firing rate will oscillate randomly in time and also the inter-spike intervals (ISI) we expect:

$$C_v = \frac{STD(ISI)}{< ISI >} = \frac{a}{\sqrt{N}}$$

for $N = 1000$ we already have $C_v \simeq 0.03$ too small in the cortex $C_v \simeq 1$

Random coupling: Fixed coupling probability

Experimentally the probability p that a neuron inside a cortical column makes a functional connection to another neuron in the same column is in the range of 10 %

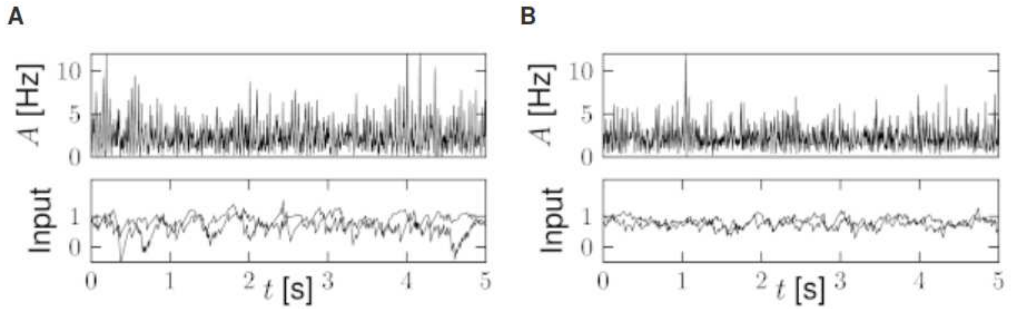


Fig. 12.7: Simulation of a model network with a fixed connection probability $p = 0.1$. **A**. Top: Population activity $A(t)$ averaged over all neurons in a network of 4 000 excitatory and 1 000 inhibitory neurons. Bottom: Total input current $I_i(t)$ into two randomly chosen neurons. **B**. Same as A, but for a network with 8 000 excitatory and 2 000 inhibitory neurons. The synaptic weights have been rescaled with a factor of $1/2$ and a common input current I^{ext} is given to all neurons to ensure that the same population activity is obtained. All neurons are leaky integrate-and-fire units with identical parameters interacting by short current pulses.

In the simulations we fix the probability p that a neuron j is connected to a pre-synaptic neuron, then the number of pre-synaptic inputs C_j has

$$\text{mean} \quad < C >= pN \qquad \text{variance} \quad \sigma^2(C) = p(1-p) * N$$

This is called a massively coupled random network, since $< C_j >$ grows proportionally to N

Random coupling: Fixed coupling probability

The input synaptic current is therefore

$$I_{syn} = w_0 C < \nu >_N$$

to avoid the divergence of the current the synaptic coupling should be rescaled as

$$w_0 = \frac{J_0}{C} = \frac{J_0}{pN}$$

In the current there are two sources of fluctations one due to the random distribution of the number C_j of the links and one to finite size effects, to the leading order the variance of I_{syn} is given by

$$\sigma^2(I_{syn}) = w_0^2(a\frac{p-1}{C} + b\frac{1}{N}) \simeq \frac{1}{pN}$$

The leading fluctuations are those associated to the random links since $p << 1$, but by increasing the number of neurons N also this decreases

Random coupling:Fixed number of presynaptic neurons

- The number of synapses of a single pyramidal neuron is of the order of a few thousand.
- Thus, when one simulates networks of a hundred thousand neurons or millions of neurons, a modeling approach based on a fixed connection probability in the range of 10 % cannot be correct.
- Moreover, in an animal participating in an experiment, not all neurons will be active at the same time. Rather only a few subgroups will be active, the composition of which depends on the stimulation conditions and the task.
- In other words, the number of inputs converging onto a single neuron may be of order thousand

A possible strategy is to consider neurons with a constant number C of pre-synaptic input neurons randomly chosen among the whole N population

- If $N >> 1$ the finite size effects are negligible
- If $C << N$ each neuron receives completely independent inputs

Random coupling:Fixed number of presynaptic neurons

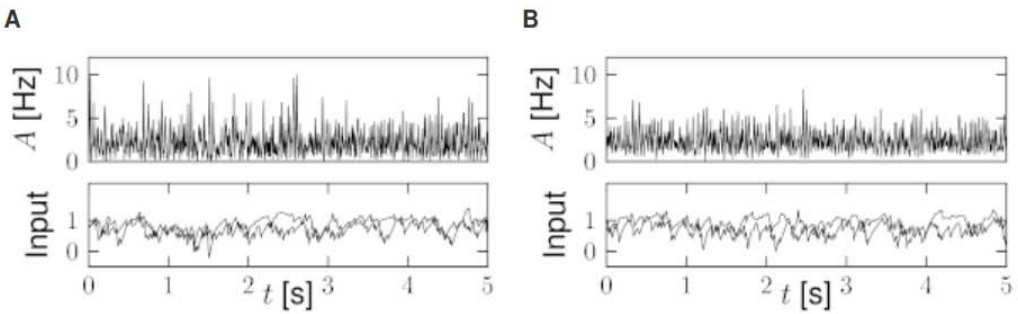


Fig. 12.8: Simulation of a model network with a fixed number of presynaptic partners (400 excitatory and 100 inhibitory cells) for each postsynaptic neuron. **A**. Top: Population activity $A(t)$ averaged over all neurons in a network of 4 000 excitatory and 1 000 inhibitory neurons. Bottom: Total input current $I_i(t)$ into two randomly chosen neurons. **B**. Same as A, but for a network with 8 000 excitatory and 2 000 inhibitory neurons. The synaptic weights have not been rescaled. While the fluctuations of the population activity $A(t)$ decrease compared to the smaller network (top), the mean and variance of the synaptic input do not change with the size of the network (bottom). All neurons are leaky integrate-and-fire units with identical parameters interacting by current pulses; cf. (79).

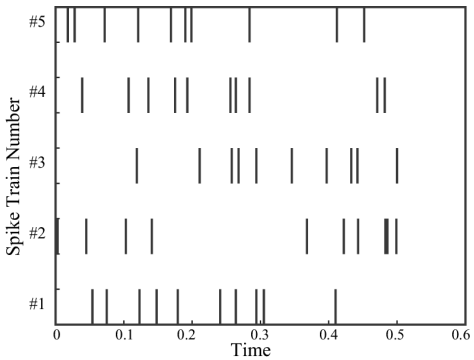
We do not need to rescale the synaptic coupling , the mean and variance of the synaptic current do not depend on N (for $N >> 1$) and are given by

$$< I_{syn} >= w_0 C \nu_0 \qquad \sigma^2(I_{syn}) = w_0^2 C \nu_0$$

this because one can assume that the train of spikes reaching one neuron is the superposition of K independent Poissonian spike trains

Poissonian Spike Trains

- C independent neurons emitting spikes via a Homogeneous Poisson Process with rate ν_0;
- the spike trains are inputs for a post-synaptic neuron.



- each neuron emits a spike with probability $\nu_0 \Delta t$ in a time interval Δt
- all neurons together emits spikes with probability $C\nu_0 \Delta t$, since they are independent

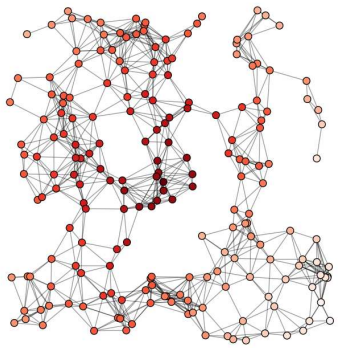
The post-synaptic neurons receives a Poissonian Spike train with rate $C\nu_0$

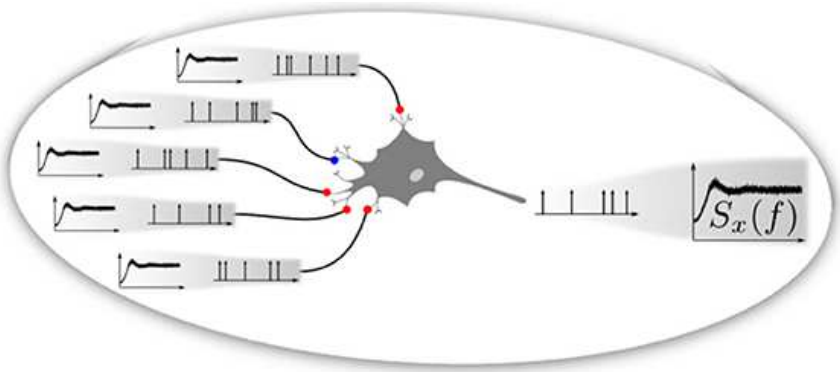
- the average number of spikes received in Δt is the spike count $< n^{sp}(\Delta t) >= C\nu_0 \Delta t$
- the average synaptic current is therefore $< I_{syn} >= w_0 \frac{<n^{sp}(\Delta t)>}{\Delta t} = w_0 C\nu_0$
- the variance of the spike count is $(\Delta n^{sp})^2 >= C\nu_0 \Delta t$
- the variance of the synaptic current is $\sigma^2(I_{syn}) = w_0^2 \frac{(\Delta n^{sp})^2}{\Delta t} = w_0^2 C\nu_0$

Cortical Dynamics

A cortical area can be seen as a recurrent random network made of a large number N of excitatory and inhibitory neurons, each one receiving many inputs ($C \simeq 1,000 - 10,000$):

- the output of a neuron is an input to another neuron of the same area, the input and output firing rate should be quite similar;
- the inputs are uncorrelated due to the high dilution in the connections $\frac{C}{N} << 1$, each neuron share
- neurons emit spikes in an irregular fashion $C_v \simeq 1$ and not too frequently $\nu_0 \simeq 1 - 10$ Hz despite the many excitatory and inhibitory inputs. Why ?



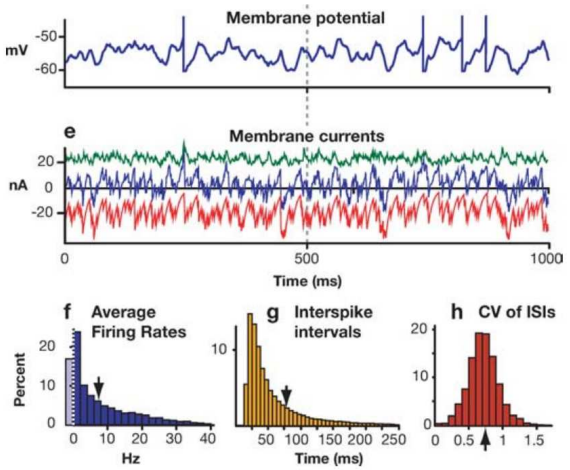


The Balanced Network

In a network of two populations, one excitatory and one inhibitory, it is possible to adjust parameters such that the mean input current into a typical neuron vanishes.

$$< I_{syn} >= I_e + I_i \simeq 0$$

- The condition is that the total amount of excitation and inhibition cancel each other, so that excitation and inhibition are 'balanced'.
- The resulting network is called a balanced network or a population with balanced excitation and inhibition
- The neurons fire irregularly and with low frequency due to current fluctuations



Vogels, Rajan, Abbott, Annu. Rev. Neurosci. (2005)

The Balanced Network: a Mathematical Model

Each neuron in a random recurrent network receives spikes of amplitude w_0 from C neurons firing with frequency ν_0.

- Average Excitatory and Inhibitory Synaptic Currents $I_e = w_0 C\nu_0$ $I_i = -w_0 C\nu_0$
- Fluctuations of the total current $I_{syn} = I_e + I_i \qquad \sigma^2(I_{syn}) = 2w_0^2 C\nu_0$

The neurons in the cortex fire quite irregularly with a finite frequency ($\simeq \nu_0$), therefore $< I_{syn} >= I_e + I_i$ and $\sigma^2(I_{syn})$ should be both FINITE for large C, this is possible if

- Excitatory and Inhibitory Inputs cancel each other $< I_{syn} >= I_e + I_i = 0$: the neurons fire due to current fluctuations
- The synaptic coupling scales as $w_0 = \frac{J_0}{\sqrt{C}}$ therefore the variance

$$\sigma^2(I_{syn}) = 2w_0^2 C\nu_0 = 2\frac{J_0^2}{C} C\nu_0 = 2J_0^2\nu_0$$

does not depend on the number C of synaptic input

The Balanced Network: a Mathematical Model

By adjusting the parameters to obtain $< I_{syn} >= I_e + I_i = 0$ and by rescaling the synaptic coupling as $w_0 = \frac{J_0}{\sqrt{C}}$ we obtain a fluctuation driven dynamics with low neuronal firing rate ν_0 for $1 << C << N$

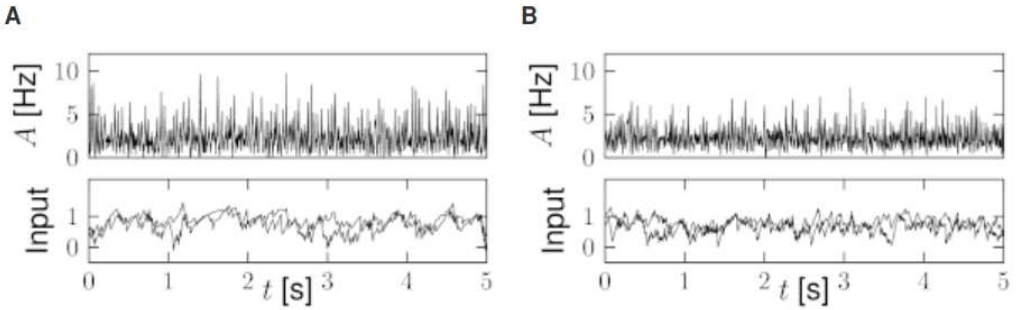


Fig. 12.9: Simulation of a model network with balanced excitation and inhibition and fixed connectivity $p = 0.1$ **A**. Top: Population activity $A\,(t)$ averaged over all neurons in a network of 4 000 excitatory and 1 000 inhibitory neurons. Bottom: Total input current $I_i\,(t)$ into two randomly chosen neurons. **B**. Same as A, but for a network with 8 000 excitatory and 2 000 inhibitory neurons. The synaptic weights have been rescaled by a factor $1/\sqrt{2}$ and the common constant input has been adjusted. All neurons are leaky integrate-and-fire units with identical parameters coupled interacting by short current pulses.

The nature does not adjust the parameters to obtain the balanced state, is it possible to obtain a dynamical balance, without adjusting the parameters ?

YES

van Vreeswijk and Sompolinsky, Science (1996)

The Balanced Network: a Mathematical Model

A simple non-linear integrate and fire model for an excitatatory and inhibitory population can be written as

$$\tau_m \frac{du_i}{dt} = F(u_i) + \frac{J_0}{\sqrt{C}} \left[\sum_{j \in pre_e(i)}^{N} \sum_f \delta(t - t_j^{(f)}) - \sum_{k \in pre_i(i)}^{N} \sum_f \delta(t - t_j^{(f)}) \right] \qquad i = 1, \ldots, N$$

therefore by assuming ν_0 as the average firing rate

$$\tau_m \frac{du_i}{dt} = F(u_i) + \frac{J_0}{\sqrt{C}} \left[C\nu_0 - C\nu_0 \right] = F(u_i) + J_0 \left[\sqrt{C}\nu_0 - \sqrt{C}\nu_0 \right] \qquad i = 1, \ldots, N$$

In order to explain the dynamical balance mechanism we can simplify the model to a single inhibitory population with a constant excitatory drive $I_e = \sqrt{C} i_0$ thus

$$\tau_m \frac{du_i}{dt} = F(u_i) + \sqrt{C} i_0 - \frac{J_0}{\sqrt{C}} \sum_{k \in pre_i(i)}^{N} \sum_f \delta(t - t_j^{(f)}) \qquad i = 1, \ldots, N$$

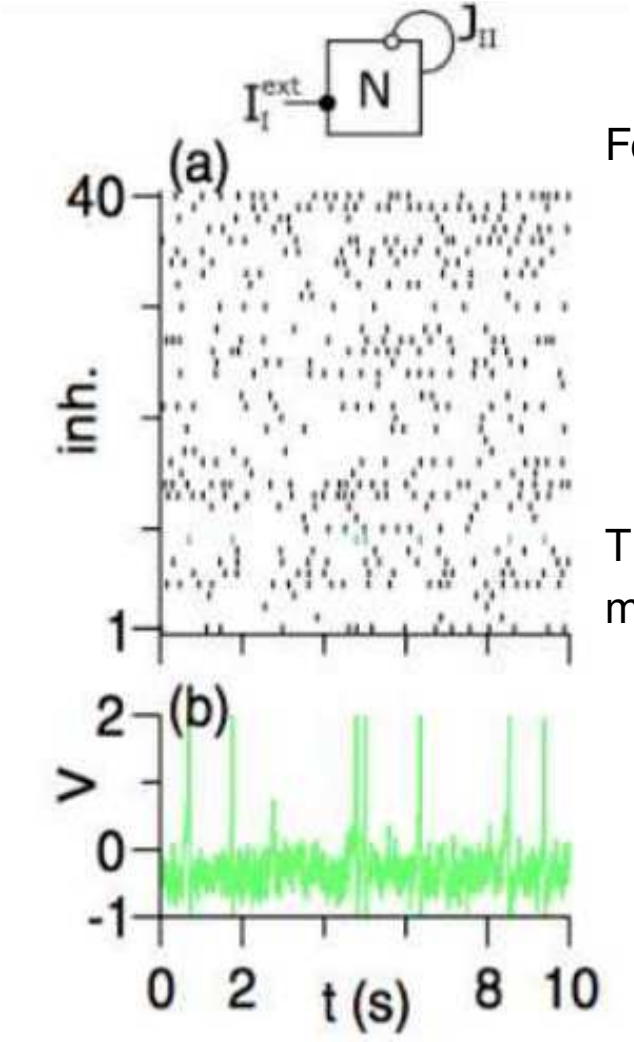
The Balanced Network: Inhibitory Network

$$\tau_m \frac{du_i}{dt} = F(u_i) + \sqrt{C} i_0 - \frac{J_0}{\sqrt{C}} \sum_{k \in pre_i(i)}^{N} \sum_f \delta(t - t_j^{(f)}) \simeq F(u_i) + \sqrt{C}[i_0 - J_0 \nu_0]$$

- The average synaptic current is $\boxed{I_{syn} = \sqrt{C}[i_0 - J_0 \nu_0]}$
- For $C >> 1$ I want that I_{syn} remains finite as in the cortex :
  - $I_{syn} > 0 \Rightarrow \nu_0 \to \infty \Rightarrow I_{syn} < 0$
    Contraddiction
  - $I_{syn} < 0 \Rightarrow \nu_0 \to 0 \Rightarrow I_{syn} > 0$
    Contraddiciton
- The only possible solution is $I_{syn} = 0 \Rightarrow \boxed{\nu_0 = \frac{i_0}{J_0}}$
- Dynamical balance without adjusting the parameters

Monteforte & Wolf, PRL (2010)

The Balanced Network: Inhibitory Network

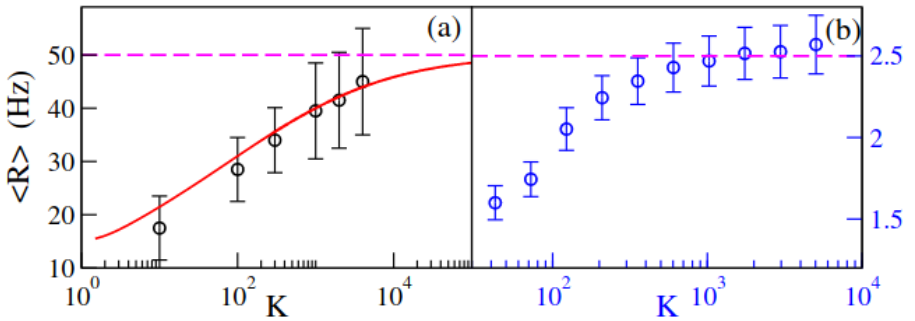


For sufficiently large C

$$I_{syn} = \sqrt{C}[i_0 - J_0\nu_0] = 0$$

$$\lim_{C\to\infty} \nu_0 = \frac{i_0}{J_0}$$

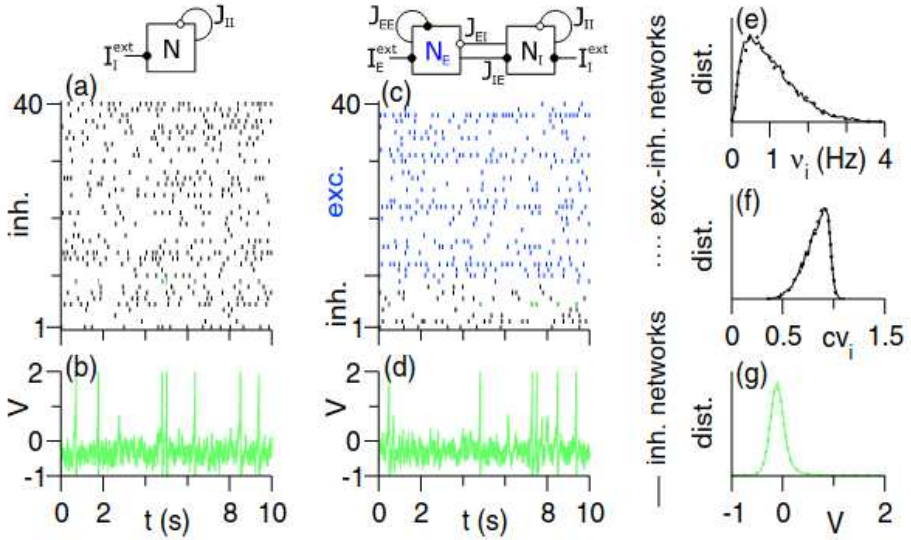
This works independently from the considered neuronal model : QIF or Morris-Lecar



di Volo & Torcini, PRL (2018)

The Balanced Network: Chaotic Dynamics

The dynamics of a neuron in a balanced network is completely irregular



- The distribution of the firing rates $\nu_i = \frac{1}{ISI}$ is exponential
- The values of the $C_{v_i} \simeq 0.5 - 1.0$
- The dynamics is Poissonian as the neurons in the cortex

The models are completely deterministic, the dynamics of a single neuron inside the network can be seen as due to

- Gaussian fluctuations of variance $\sigma^2(I_{syn}) \propto J_0^2 \nu_0$

self-induced in the network, without the addition of any noise. Therefore the irregularity are due to chaotic dynamics.

van Vreeswijk and Sompolinsky, Science (1996)

Experimental Confirmations

nature neuroscience

Synaptic scaling rule preserves excitatory–inhibitory balance and salient neuronal network dynamics

Jérémie Barral[1] & Alex D Reyes[1]

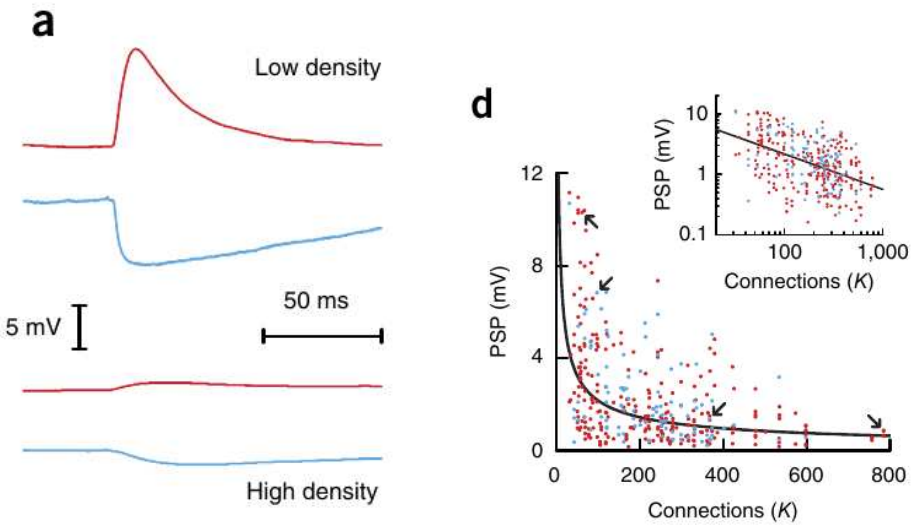
The balance between excitation and inhibition (E–I balance) is maintained across brain regions though the network size, strength and number of synaptic connections, and connection architecture may vary substantially. We use a culture preparation to examine the homeostatic synaptic scaling rules that produce E–I balance and *in vivo*-like activity. We show that synaptic strength scales with the number of connections K as $\sim 1/\sqrt{K}$, close to the ideal theoretical value. Using optogenetic techniques, we delivered spatiotemporally patterned stimuli to neurons and confirmed key theoretical predictions: E–I balance is maintained, active decorrelation occurs and the spiking correlation increases with firing rate. Moreover, the trial-to-trial response variability decreased during stimulation, as observed *in vivo*. These results—obtained in generic cultures, predicted by theory and observed in the intact brain—suggest that the synaptic scaling rule and resultant dynamics are emergent properties of networks in general.

Barral & Reyes in 2016 have shown in experiments on a in vitro culture of neurons of the cortex that the main theoretical predictions done by van Vreeswijk & Sompolinsky in 1996 were correct

Experimental Confirmations

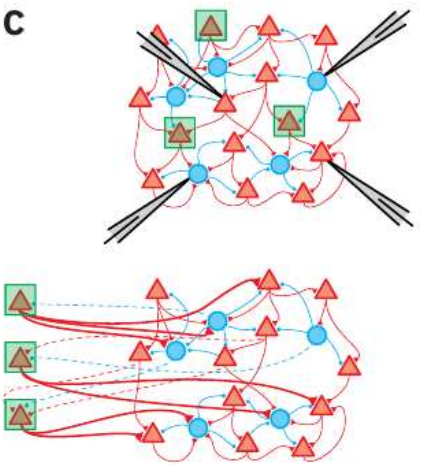
Barral & Reyes considered a culture of cortical neurons composed of 77 % of excitatory and 23 % of inhibitory neurons.
They measured the Postsynaptic Potential amplitudes of excitatory (EPSP) and inhibitory (IPSP) neurons and observed that

- Amplitudes of EPSP and IPSP decrease with the density of neurons, therefore with the number of pre-synaptic connections K
- The amplitude of EPSP (IPSP) scales as $K^{-0.60}$ ($K^{-0.52}$) with the connectivity K in good agreement with the prediction of van Vreeswijk & Sompolinsky $\propto 1/\sqrt{K}$



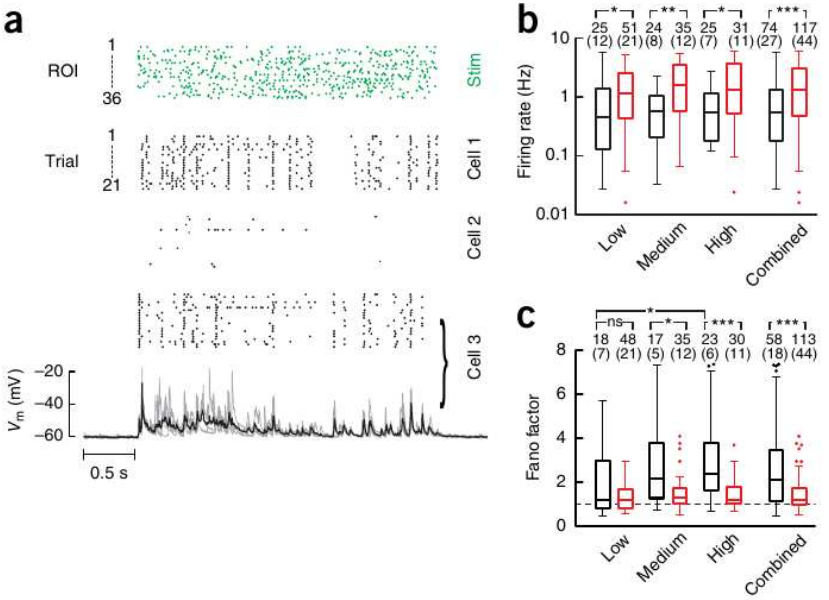
Experimental Confirmations

Barral & Reyes stimulated with light a small group of neurons that were optogenitically modified to induce fring activity in the network, the activity of these neurons represented the excitatory drive to the network.



The evoked activity in the network is consistent with key predictions:

- The Fano factor $\simeq 1$ indicating Poissonian dynamics;
- The spiking variability does not vary with the number of neurons N, as expected since $\sigma^2(I_{syn})$ does not depend on K or N
- The distribution of the firing rates has a long-tail approximately log-normal.



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