Dynamics of Sparsely Connected Networks of Excitatory and Inhibitory Spiking Neurons

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Overview

Introduction

Basics:

- Analytical Examination
- 2 states (**Synchronous** and **Asynchronous**)
- 2 types of network oscillation (**Fast** Oscillation State and **Slow** Oscillation State)

Introduction

Following were determined analytically:

- The characteristics and the region of stability of the **Asynchronous** states
- Oscillations frequency on the various Hopf bifurcation lines (the boundary of the region of stability)
- The phase lag between **excitatory** and **inhibitory** populations on the Hopf bifurcation line
- The auto-correlation function, or equivalently the power spectrum, of the global activity in a finite network, in the **Asynchronous** region.

Introduction

Beyond Hopf bifurcation lines, the behaviour of the system is studied through:

- Numerical Integration of coupled non-linear PDE's
- Numerical simulations of the model

Introduction

The balance between excitation, inhibition and temporal characteristics of synaptic procession (depending on the values of external frequencies), they found:

- **Synchronous regular** (SR) states - excitation dominates inhibition and synaptic time distribution are sharply peaked
- **Asynchronous regular** (AR) states - with stationary global activity, excitation dominates inhibition and synaptic time distribution are broadly peaked
- **Asynchronous irregular** (AI) states - with stationary global activity but strongly irregular firing at low rate when inhibition dominates excitation in an intermediate range of external frequencies
- **Asynchronous irregular** (AI) states - with oscillatory global activity but strongly irregular firing at low rate (compared to

Model

The dynamics of a network composed of:

- N integrate-and-fire (IF) - N_E(excitatory) and N_I(inhibitory)
- Each neuron receives C randomly chosen connections from other neurons in the network:
  - $C_E = \epsilon N_E$ (from excitatory networks)
  - $C_I = \epsilon N_I$ (from inhibitory networks)
- The network also receives C_{ext} connections from excitatory neurons outside the network.

Model

The depolarization $V_i(t)$ of neuron $i, (i = 1, 2, ..., N)$ at its soma obeys the equation :

- $\tau V_i(t) = -V_i(t) + RI_i(t)$ (1)
- $RI_i(t) = \tau \sum_j J_{ij} \sum_k \delta(t - t_j^k - D)$ (2)

$\sum_j J_{ij}$ - sum on different synapses ($j = 1, ..., C + C_{ext}$) with postsynaptic potential (PSP) J_{ij}.
$\sum_k \delta(t - t_j^k - D)$ - is the sum on different spikes arriving at synapse j, at time $t = t_j^k - D$, where t_j^k is the emission time of kth spike at neuron j with a transmission delay D.

Model - Cases

- Model A- When excitatory and inhibitory neurons have identical characteristics
- Model B- (Taking psychological data under consideration) when inhibitory and excitatory neurons have different characteristics.

Model - A

- PSP amplitude equal at each synapse
  - $J_{ij} = J > 0$ for excitatory (recurrent and external) synapses
  - $J_{ij} = gJ > 0$ for inhibitory synapses.
- External synapses are activated by independent Poisson process with rate ν_{ext}
- frequency that is needed for neuron to reach threshold $\nu_{thr} = \frac{\theta}{JC_E\tau}$

Model - B

- memberane time constants:
  - τ_E for excitatory
  - τ_I for inhibitory
- Synaptic efficacies:
  - $J_{EE} = J_E$ from excitatory - excitatory
  - $J_{EI} = g_E J_E$ from inhibitory - excitatory
  - $J_{IE} = J_I$ from excitatory - inhibitory
  - $J_{II} = g_I J_I$ from inhibitory - inhibitory
- PSP amplitude:
  - J_E for excitatory (recurrent and external) synapses
  - J_I for inhibitory synapses
- External frequencies: $\nu_{E,ext}$ (excitatory) and $\nu_{I,ext}$ (inhibitory)
- Delays: D_{ab} for synapses connecting population b to a for $a, b = E, I$

Model - A (identical excitatory and inhabitory neurons)

Consider a regime in which individual neurons recieves a large no of inputs per integration time τ, and each input makes a small contribution compared to the firing threshold ($J \ll \theta$). In this situation, the synaptic current of a neuron can be approximated by an average part plus a fluctuating gaussian part:

- $RI_i(t) = \mu(t) + \sigma\sqrt{\tau}\eta_i(t)$ (3)

Average part $\mu(t)$

$\mu(t)$ is related to the firing rate ν at time $t - D$ and is a sum of local and external inputs:

- $\mu(t) = \mu_I(t) + \mu_{ext}$
- $\mu_I(t) = C_E J(1 - \gamma g)\nu(t - D)\tau$ (4)
- $\mu_{ext} = C_E J \nu_{ext} \tau$

Fluctuating part $\sigma\sqrt{\tau}\eta_i(t)$

The fluctuating part, $\sigma\sqrt{\tau}\eta_i(t)$, is given by the fluctuation in the sum of internal excitatory, internal inhibitory, and external Poisson inputs of rates $C_E\nu$, $\gamma C_E\nu$ and $C_E\nu_{ext}$:

- $\sigma^2(t) = \sigma_I^2(t) + \sigma_{ext}^2$
- $\sigma_I(t) = J\sqrt{C_E(1+\gamma g^2)\nu(t-D)\tau}$ (5)
- $\sigma_{ext} = J\sqrt{C_E\nu_{ext}\tau}$

$\eta_i(t)$ is a gaussian white noise, with $\langle\eta_i(t)\rangle = 0$ and $\langle\eta_i(t)\eta_i t'\rangle = \delta(t-t')$

Fokker-plank Equation

The Fokker-plank Equation describing the evolution of the probability distribution: distribution:

- $\tau \frac{\partial P(V,t)}{\partial x} = \frac{\sigma^2(t)}{2} \frac{\partial^2 P(v,t)}{\partial V^2} + \frac{\partial}{\partial V}[(V - \mu(t))P(V,t)]$ (6)
- $\frac{\partial P(V,t)}{\partial x} = \frac{\partial S(V,t)}{\partial V}$ (7)
- $\frac{\partial S(V,t)}{\partial V} = \frac{-\sigma^2(t)}{2\tau} \frac{\partial P(v,t)}{\partial V} - \frac{(V-\mu(t))}{\tau} P(V,t)$ (8)
- $\frac{\partial P(\theta,t)}{\partial V} = -\frac{2\nu(t)\tau}{\sigma^2(t)}$ (9)

The difference between the probability currents $S(V_r^+, t) - S(V_r^-, t) = \nu(t - \tau_{rp})$

- $\frac{\partial P(V_r^+,t)}{\partial V} - \frac{\partial P(V_r^-,t)}{\partial V} = -\frac{2\nu(t-\tau_{rp})\tau}{\sigma^2(t)}$ (10)
- $\lim_{V\to-\infty} P(V,t) = 0 \qquad \lim_{V\to-\infty} VP(V,t) = 0$ (11)
- $\int_{-\infty}^{\theta} P(V,t)dV + p_r(t) = 1$ (12)
  in which $p_r(t) = \int_{t-t_{rp}}^{t} \nu d\nu$

When excitatory and inhibitory cells have different characteristics, we need to study the statistical properties of the populations separately:

- $\mu_a = C_E J_a \tau_a[\nu_{a,ext} + \nu_E(t - D_{a,E}) - \gamma_a g_a \nu_I(t - D_{a,E})]$ (13)
- $\sigma^2 = J_a^2 C_E \tau_a[\nu_{a,ext} + \nu_E(t - D_{a,E}) - \gamma_a g_a \nu_I(t - D_{a,I})]$ (14)

The system is now described by the distribution of the neuron depolarization $P_a(V,t)$, that is; the probability of finding the depolarization of a randomly chosen neuron of population $a = E, I$ at V at time t:

- $\tau_a \frac{\partial P_a(V,t)}{\partial t} = \frac{\sigma_a^2(t)}{2} \frac{\partial^2 P_a(V,t)}{\partial V^2} + \frac{\partial}{\partial V}[(V - \mu_a(t))P_a(V,t)],$ $a = E, I$ (15)
- $\frac{\partial P_a}{\partial t}(\theta, t) = -\frac{2\nu_a(t)\tau_a}{\sigma_a^2(t)}$ (16)
- $\frac{\partial P_a}{\partial V}(V_r^+, t) - \frac{\partial P_a}{\partial V}(V_r^-, t) = -\frac{2\nu_a(t-\tau_{rp})\tau_a}{\sigma_a^2(t)}$ (17)
- $\lim_{V \to -\infty} P_a(V,t) = 0 \qquad \lim_{V \to -\infty} VP_a(V,t) = 0$ (18)

Model - A

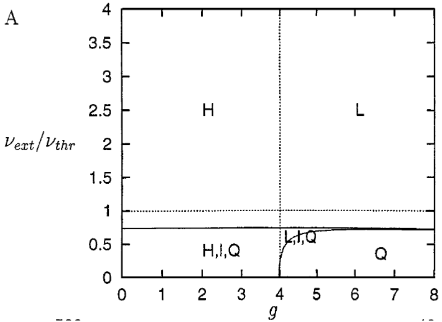
In a stationary solution, $P(V,t) = P_0(V), p_r = p_{r,0}$. Time independent solutions of (6) satisfying the boundary conditions (9), (10), and (11) given by:

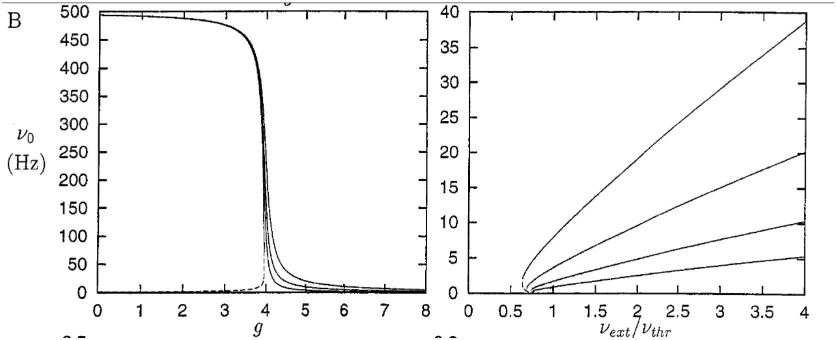
- $$P_0(V) = 2\frac{\nu_0\tau}{\sigma_0} exp(-\frac{(V-\mu_0)^2}{\sigma_0^2}) x \int_{\frac{V-\mu_0}{\sigma_0}}^{\frac{\theta-\mu_0}{\sigma_0}} \Theta(u-V_r)e^{u^2}du,$$
  $$p_{r,0} = \nu_0\tau_{rp} \qquad (19)$$
- $\mu_0 = C_E J\tau[\nu_{ext} + \nu_0(1-g\gamma)]$
- $$\sigma_0^2 = J_a^2 C_E\tau[\nu_{ext} + \nu_0(1+g^2\gamma)] \qquad (20)$$

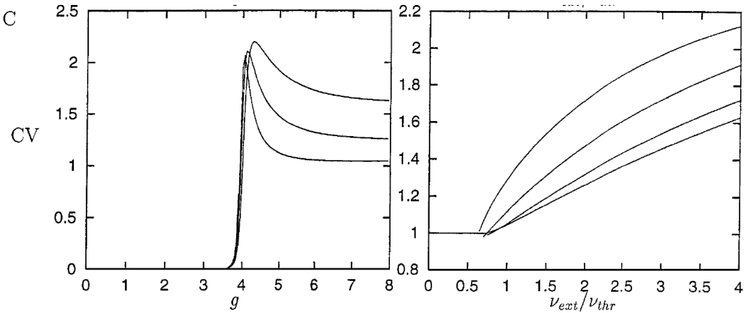
The normalization condition (12) provides the self consistent condition, which determines ν_0

- $$\frac{1}{\nu_0} = \tau_{rp} + 2\tau \int_{\frac{V_r-\mu_0}{\sigma_0}}^{\frac{\theta-\mu_0}{\sigma_0}} du e^{u^2} \int_{-\infty}^{u} dv e^{-v^2}$$
  $$= \tau_{rp} + \tau\sqrt{\pi} \int_{\frac{V_r-\mu_0}{\sigma_0}}^{\frac{\theta-\mu_0}{\sigma_0}} du e^{u^2}(1 + erf(u)) \qquad (21)$$
- $$\nu_0\tau \simeq \frac{(\theta-\mu_0)}{\sigma_0\sqrt{\pi}} exp(-\frac{(\theta-\mu_0)^2}{\sigma_0^2}) \qquad (22)$$
- $$\nu_{thr} = \frac{\theta}{C_E J\tau}$$

To probe the nature and number of stationary states in the plane (g, ν_{ext}), (20) and (21) were solved numerically. The results for $C_E = 4000, J = 0.2mV$ with parameters $\nu_{thr} = 1.25Hz, g = 4$ is shown in the fig A:




B
ν_0 (Hz)
500
450
400
350
300
250
200
150
100
50
0
0 1 2 3 4 5 6 7 8
g
40
35
30
25
20
15
10
5
0
0 0.5 1 1.5 2 2.5 3 3.5 4
ν_{ext}/ν_{thr}


C
CV
2.5
2
1.5
1
0.5
0
0 1 2 3 4 5 6 7 8
g
2.2
2
1.8
1.6
1.4
1.2
1
0.8
0 0.5 1 1.5 2 2.5 3 3.5 4
ν_{ext}/ν_{thr}

Simple Estimates of the stationary firing rates for Large C_E

- $\nu_0 = \frac{1}{\tau_{rp}}[1 - \frac{\theta - V_r}{C_E J(1-g\gamma)}]$ (23)
- $\nu_0 = \frac{\nu_{ext} - \nu_{thr}}{g\gamma - 1}$ (24)

Stationary states for Model B

Considering the stationary solutions for the two probability distributions $P_a(V,t) = P_{a0}(V), p_{ra}(t) = p_{ra0}$ a = E,I.

- $P_{a0}(V) = 2\frac{\nu_{a0}\tau_{a0}}{\sigma_{a0}} exp(\frac{(V-\mu_{a0})^2}{\sigma_{a0}^2}) x \int_{\frac{V-\mu_{a0}}{\sigma_{a0}}}^{\frac{\theta-\mu_{a0}}{\sigma_{a0}}} \Theta(u - V_r) e^{u^2} du,$

  $p_{r,0} = \nu_{a0} \tau rp$ (25)
- $\mu_{a0} = C_E J_a \tau_a [\nu_{ext} + \nu_{E0} - \gamma g \nu_{I0}]$
- $\sigma_{a0}^2 = J_a^2 C_E \tau_a [\nu_{ext} + \nu_{E0} + \gamma g^2 \nu_{I0}]$ (26)
- $$\frac{1}{\nu_{a0}} = \tau_{rp} + 2\tau_a \int_{\frac{V_r-\mu_{a0}}{\sigma_{a0}}}^{\frac{\theta-\mu_{a0}}{\sigma_{a0}}} due^{u^2} \int_{-\infty}^{u} d\nu e^{-\nu^2}$$

  (27)

Linear Stability Analysis

Rescaling of P, V and ν:

- $P = \frac{2\tau\nu_0}{\sigma_0} Q$
- $y = \frac{V-\mu_0}{\sigma_0}$
- $\nu = \nu_0(1 + n(t))$

Linear Stability Analysis

After rescaling the Fokker-Planck equation becomes:

- $\tau \frac{\partial Q}{\partial t} = \frac{1}{2} \frac{\partial^2 Q}{\partial y^2} + \frac{\partial}{\partial y}(yQ) + n(t - D)(G \frac{\partial Q}{\partial y} + \frac{H}{2} \frac{\partial^2 Q}{\partial y^2})$

with

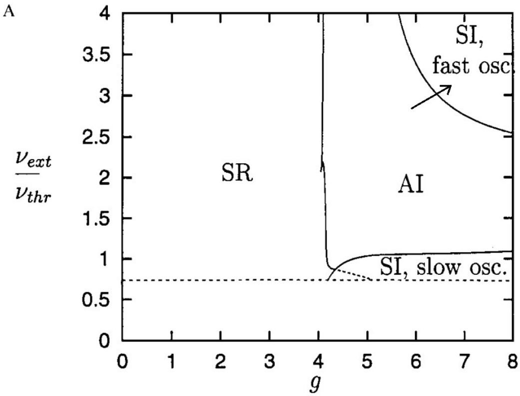
- $G = \frac{C_E J \tau \nu_0 (g\gamma - 1)}{\sigma_0} = \frac{-\mu_{0,l}}{\sigma_0}$
- $H = \frac{C_E J^2 \tau \nu_0 (g^2\gamma - 1)}{\sigma_0^2} = \frac{\sigma_{0,l}^2}{\sigma_0^2}$

Solution of the first order linear equation

- $Q_1 \;=\; exp(\omega t)\hat{Q}_1 \;\; and \;\; n_1 \;\sim\; exp(\omega t)\hat{n}_1$

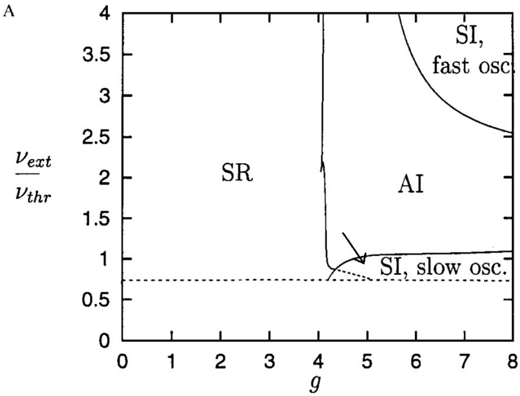
Transition to a fast oscillation of the global activity:

- the Hopf bifurcation line in the high g, high ν_{ext} region shows a transition to a synchronous irregular state with fast oscillation



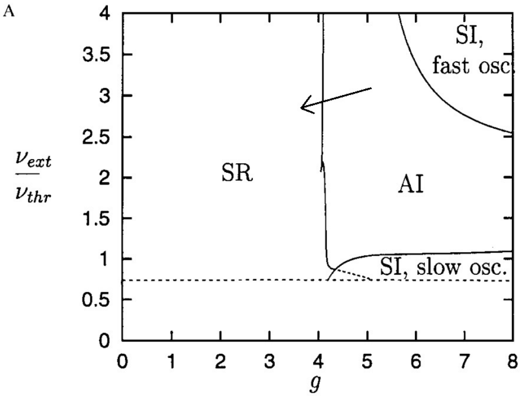
Transition to a slow oscillation of the global activity:

- the Hopf bifurcation line where ν_{ext} is almost equal to ν_{thr} shows a transition to a synchronous irregular state with slow oscillation

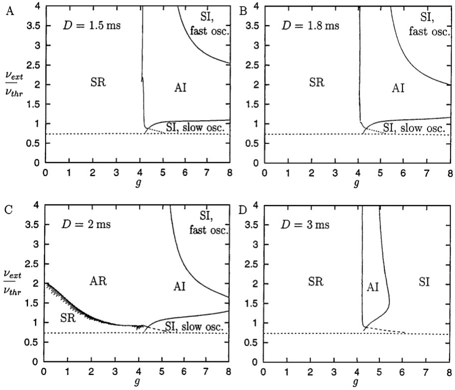


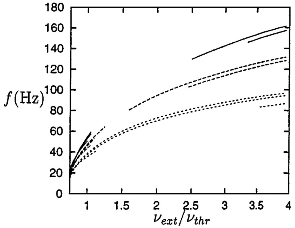
Transition to a very fast Oscillation (near saturation):

- near g = 4 another Hopf bifurcation line appears wich divides the asynchronous irregular state from the synchronous regular state



if D/τ_{rp} is an integer the instability line will be pushed towards lower values of g

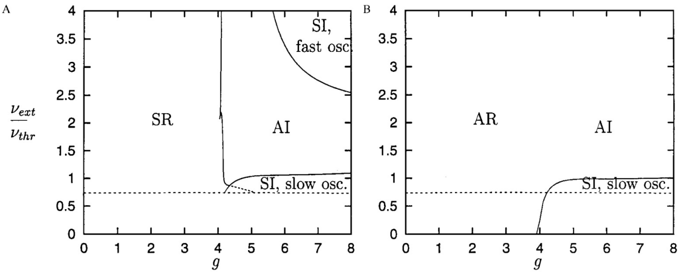




(a) D = 1.5ms, 2ms, 3ms; g = 8, 6, 5

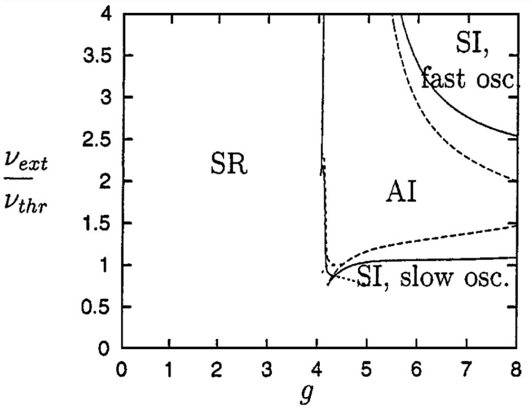
Wide Distribution of Delays

As we can see a uniformly distributed transmission delay D between 0ms and 3ms changes the behaviour of the system drasticly



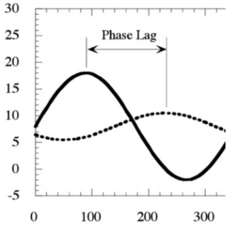
External Noise

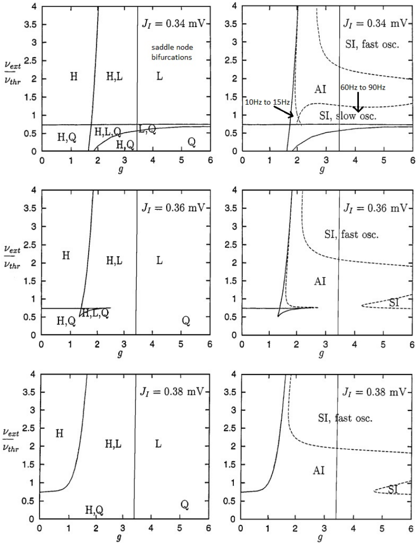
The phase diagram depends only weakly on the external noise



Analysis of Model B

- the linear stability analysis can be studied as much as in Model A
- if excitatory and inhibitory cells have different characteristics a phase lag appears
- the phase lag can be well approximated by the imaginary part of the eigenvalues with $\omega(D_{EI} - D_{II})$

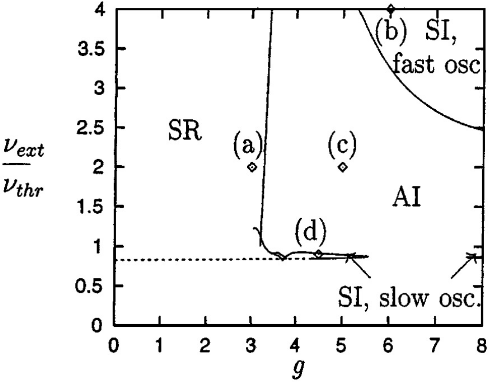




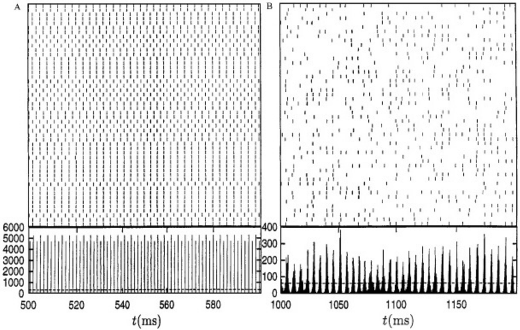
$J_I = 0.34$ mV
saddle node bifurcations
H
H,L
L
H,Q
H,L,Q
L,Q
H,Q
Q
$\frac{\nu_{ext}}{\nu_{thr}}$
g
SI, fast osc.
AI
60Hz to 90Hz
10Hz to 15Hz
SI, slow osc.
$J_I = 0.36$ mV
H,L,Q
SI
$J_I = 0.38$ mV

Simulating

- we choose J = 0.1 mV, $N_E = 10000$, $N_I = 2500$, $C_e = 1000$, $C_I = 250$ and D = 1,5ms



- (a) the network is strongly synchronized with a fast oscillation
- (b) it settles in a state where the global activity exhibits a fast oscillation close to 180Hz, where Neurons fire irregularly at a lower rate, about 60Hz.



- (c) it settles in a state in which the global activity exhibit strongly damped oscillation and neurons fire irregularly
- (d) it exhibits a slow oscillation, around 20Hz, with very low neuron firing rates, about 5Hz.(external frequency is below but near threshold)

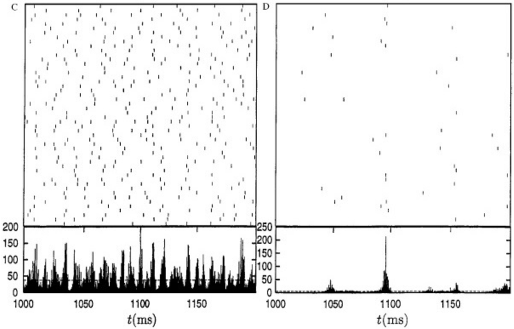


Table 1. Comparison between simulations and theory in the inhibition-dominated irregular regimes: Average firing rates and global oscillation frequency.

	Firing rate		Global frequency	
	Simulation	Theory	Simulation	Theory
B. SI, fast	60.7 Hz	55.8 Hz	180 Hz	190 Hz
C. AI	37.7 Hz	38.0 Hz	—	—
D. SI, slow	5.5 Hz	6.5 Hz	22 Hz	29 Hz

- the analysis developed for the SR state is not adequate, because correlations are beyond the once induced by a time varying firing rate $\nu(t)$
- in the inhibition dominated regime the analysis predicts it quite well
- the global activity is in the AI state not quite stationary as predicted

Not Predicted Behaviour

- for finite neuron size two new affects appear
- in the stationary AI regime a strongly damped oscillatory component appears
- in the SI regimes it creates a phase diffusion of the global activity

Finite Size Effects

- when the dynamics are stochastic, sharp transitions occur only for N $\rightarrow \infty$
- analysing it by simplifying the Model with 2 processes
- the spike emission process S(t)
- the connectivity process $\rho_i(t)$
- $RI_i(t) \ = \ -J\tau\rho_i(t)S(t-D)$

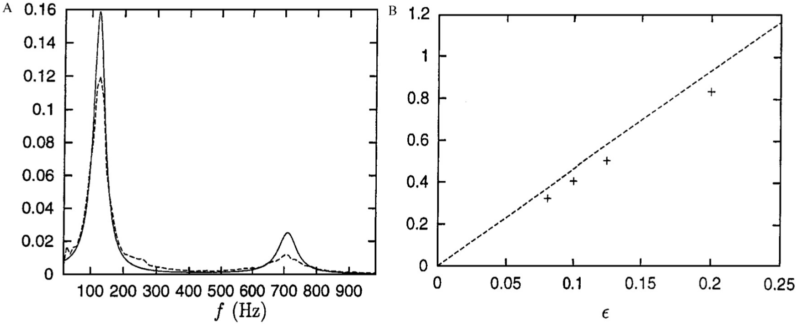
Decomposing both processes between their mean and their fluctuation

- $\rho_i(t) \;=\; \frac{C}{N} + \delta\rho_i(t),\;\; S(t) \;=\; N\nu(t) + \delta S(t)$

$$\Rightarrow \;\; RI_i(t) \;=\; \mu(t) - J\tau N\nu(t)\delta\rho_i(t) - J\tau\frac{C}{N}\delta S(t)$$

Since we can model the fluctuations with Gaussian White Noise, the mean synaptic input becomes

$$\Rightarrow \;\; CJ\tau\nu(t) + J\tau\sqrt{\epsilon C\nu_0}\,\xi(t) + \mu_{ext}$$



Power spectrum of the global activity in $g = 5$, $\frac{\nu_{ext}}{\nu_{thr}} = 2$

Conclusion

- first time analytical picture of dynamics of randomly interconnected excitatory and inhibitory spiking neurons
- the model outlines the importance of the transmission delay D
- similar behaviour of Model A to Model B was shown
- the finite neuron size Model predicted quite well the behaviour of the simulation
- in the inhibitory regime the system switches from the stationary to the oscillatory state through changes of the external input only

Relationships with Neurophysiological Data

- the behaviour of the synchronous irregular state is shown in Neurophysiological data for example in the visual cortex of mammals
- collected data of the Neo-Cortex showing a similar behaviour like the asynchronous irregular state

Thanks for your attention

Any questions?