Mean-field Theory and Synchronization in Random Recurrent Neural Networks

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Abstract. In this paper, we first present a new mathematical approach, based on large deviation techniques, for the study of a large random recurrent neural network with discrete time dynamics. In particular, we state a mean field property and a law of large numbers, in the most general case of random models with sparse connections and several populations. Our results are supported by rigorous proofs. Then, we focus our interest on large size dynamics, in the case of a model with excitatory and inhibitory populations. The study of the mean field system and of the divergence of individual trajectories allows to define different dynamical regimes in the macroscopic parameters space, which include chaos and collective synchronization phenomenons. At last, we look at the behavior of a particular finite-size system submitted to gaussian static inputs. The system adapts its dynamics to the input signal, and spontaneously produces dynamical transitions from asynchronous to synchronous behaviors, which correspond to the crossing of a bifurcation line in the macroscopic parameters space.

Keywords: Asymmetric Networks, Chaos, Mean Field theory, Stochastic Dynamics, Synchronization, Two-population models.

1. Introduction

In a living system, short and long-distance synchronization is a transient phenomenon. It corresponds to a mutual increase in coherency between different brain regions. Such *dynamical phenomena* are conjectured to have an active influence on perception-action processes, and to condition the ability of performing recognition (Gray and Singer, 1987; Neuenschwander et al., 1996; Mac Leod and Laurent, 1996). A recognition process seems to occur through a *bifurcation* from one dynamical regime to the other (Skarda and Freeman, 1987; Rodriguez et al., 1999).

Synchronous behavior has been reproduced in artificial biologically plausible random neural networks (Hansel and Sompolinsky, 1996; Brunel and Hakim, 1999). Yet, a drawback of these models is that their biological relevance and their complexity prevent any rigorous proof of the simulation supported assertions. Moreover, it is not sure that the dynamical properties of large assemblies of neurons stand on complex cellular mechanisms. It is therefore relevant to focus one's interest on the behavior of less sophisticated networks and to wonder whether they exhibit dynamical properties that can relate to biology.

Thus, the quite elementary model introduced by Amari (Amari, 1972; Amari et al., 1977) has been the focus of several studies these last years. The neurons are modelled by N localized continuous variables, and the connections do not change with time. The occurence of chaos in this network has been obtained in (Crisanti et al., 1990). Moreover, statistical physics arguments have been used (Sompolinsky et al., 1988) to derive mean-field equations, which give a statistical description of the behavior of the network in the thermodynamic limit : the long term dynamics only depend on a few number of statistical parameters. These mean-field equations have also been obtained if the connections are chosen independently at each time step (Bresslof and Taylor, 1990), or in a two population network where the respective influences of excitation and inhibition are balanced (Van Vreeswijk and Sompolinsky, 1998). In these works, the mean-field equations admit fixed points when time grows to infinity. Their stability is used to predict with a good accuracy the dynamics of individual large-size neural networks.

Nevertheless, the methods used to obtain the mean-field equations don't constitute a rigorous proof from mathematical point of view. Moreover, they only describe the statistical behavior of the network, and don't give any information about the neurons of a given network with fixed connection weights. Mathematically, this means that these dynamic mean-field methods lead to weak convergence results, and not to any almost sure convergence properties.

This paper is dedicated to the study of a discrete time recurrent neural network model which takes its inspiration from (Amari et al., 1977). The connection weights and thresholds are randomly drawn under some probabilistic law. The design of this model includes recurrent connection pattern, sparsity of connections and large size approach.

The main interest of this paper is triple :

- All of our mean-field results are supported by rigorous proofs, which use a large deviation methodology (Ben Arous and Guionnet, 1995). Large deviation techniques have already been used for the symetric Hopfield model (Bovier and Gayrard, 1996), but are new in the context of recurrent asymmetric neural networks (Section 2.3.2).

- We prove a law of large numbers, which describes the asymptotic behavior of the mean activity of the neurons when the size of the network grows to infinity. This result is obtained for almost any values of the random connection weights and not statistically (Section 2.3.3).

- Last, there is a parametric domain where the mean-field equations don't admit a fixed point for large time : in the case of a model with excitatory and inhibitory populations, mean-field periodic and quasi-periodic regimes occur asymptotically. So, these dynamics are qualitatively different from previous studies about mean-field equations in neural networks (Van Vreeswijk and Sompolinsky, 1998; Cessac, 1995). Note that Amari (Amari, 1974) pointed out the existence of oscillations in a two-population model for a different normalization of the dispersion of the weights. Moreover, the study of the divergence of individual trajectories allows to distinguish stable and chaotic behaviors in the thermodynamic limit. These large scale theoretical behaviors can be interpreted in terms of finite-sized systems. In particular, various regimes of collective oscillations are described at finite size. In presence of a static input, we observe dynamical transitions that may relate to natural processes of perception (Section 3).

2. Mathematical advances

2.1. Model

The network contains two populations of neurons, each population being labelled by index p. They might for instance represent excitatory ($p = 1$) and inhibitory ($p = 2$) populations. For $p \in \{1, 2\}$, there are N_p neurons in population p. $N = N_1 + N_2$ is the total number of neurons in the network. For $p, q \in \{1, 2\}^2$, the $(J_{ij}^{pq})'s$ represent the connection weights relative to the influence of population q towards population p. The θ_i^p are thresholds (or, equivalently, inputs remaining constant), and $W_i^p(t)$ is a synaptic noise.

We consider the following discrete time dynamics: for $p \in \{1,2\}$ and $1 \leq i \leq N_p$,

$$\begin{cases} x_i^p(t) = f(u_i^p(t)) \\ u_i^p(t) = \sum_{j=1}^{N_1} J_{ij}^{p1} x_j^1(t-1) + \sum_{j=1}^{N_2} J_{ij}^{p2} x_j^2(t-1) + \sigma W_i^p(t) - \theta_i^p \end{cases} \tag{1}$$

f is an arbitrary bijective, continuous sigmoidal function from $\mathbb{R}$ to $]0,1[$. For numerical simulations, we use $f(x) = \frac{1+\tanh(x)}{2}$. $x_i^p(t)$ represents the activation state of neuron i of population p at time t. $u_i^p(t)$ is the local field of neuron i of population p at time t. All neurons are supposed to be independent at time 0. The distribution of $x_i^p(0)$ is μ_0^p.

The connections are zero with probability $(1-\rho)$, where $\rho \in [0,1[$. We suppose that the distributions of the non zero connection weights, the thresholds and the synaptic noise are respectively gaussian laws $\mathcal{N}(\frac{\bar{J}^{pq}}{\rho N_q}, \frac{(J^{pq})^2}{\rho N_q} + \frac{(\bar{J}^{pq})^2(\rho-1)}{N^2\rho^2})$ (so that the global mean of the connections weights is $\frac{\bar{J}^{pq}}{N}$, and their variance $\frac{(J^{pq})^2}{N}$), $\mathcal{N}(\bar{\theta}^p, (\sigma_\theta^p)^2)$ and $\mathcal{N}(0,1)$. All those random variables are supposed to be independent.

We study the statistical behavior of such systems in the *thermodynamic limit* framework, i.e. when the sizes of the populations grow to infinity. Moreover, there is no change in their proportion (i.e $\exists \lambda \in]0,1[, \lim_{N\to+\infty} \frac{N_1}{N} = \lambda$).

2.2. The methodology

We now describe the method we use to get our mathematical results.

We first focus our interest on the case of gaussian connection weights ($\rho = 1$). We adapt the large deviation techniques developed by Ben Arous and Guionnet (Ben Arous and Guionnet, 1995; Guionnet, 1997) in a one population continuous time spin glass context.

We consider the evolution of the system (1) between time 0 and a fixed time T. For technical reasons, we suppose $\sigma > 0$. Notice that the results are valid for σ arbitrarily small. We denote by P_N the law of the set of individual trajectories $x = (x_1^1, ..x_{N_1}^1, x_1^2, .., x_{N_2}^2)$, with $x_i^p = (x_i^p(t))_{t=0..T}$. Let $(\hat{\mu}_N^1, \hat{\mu}_N^2)$ be defined for $p \in \{1,2\}$ by :

$$\hat{\mu}_N^p = \frac{1}{N_p} \sum_{i=1}^{N_p} \delta_{x_i^p}$$

Here, δ_a is the Dirac measure. We denote by π_N the image law of P_N by $(\hat{\mu}_N^1, \hat{\mu}_N^2)$. It is a probability on $(\mathcal{M}_1^+(]0,1[^{[\![0,T]\!]}))^2$, which is the set of couples of probabilities on $]0,1[^{\{0,1,..,T\}}$.

Recall that a family of probabilities π_N on a space E satisfies *a large deviation principle* if there exists a function I from E to $[0, +\infty]$ such that for any subset A in E, if $\inf_A I > 0$, $\pi_N(A)$ converges exponentially fast towards zero when N grows to infinity, with rate approximated by $\inf_A I$. I is called the *rate function* of the large deviation principle. For any precise definitions and properties about large deviations, see (Deuschel and Stroock, 1989).

In (Moynot and Samuelides, 2001; Moynot, 2000), we proved that when N grows to infinity, the family π_N defined above satisfies a large deviation principle, whose rate function I admits a unique minimum $P = (P^1, P^2)$ such that $I(P) = 0$.

The expression of the rate function I is directly related to the evolution law of the network. We omit here its analytic expression, because it is very intricate and useless to understand the main results of this paper.

In particular, we thus established that : $\forall r > 0, \exists a > 0, \exists N_0 \in \mathbb{N}, \forall N \geq N_0$,

$$P_N\left((\hat{\mu}_N^1(x), \hat{\mu}_N^2(x)) \notin B(P, r)\right) \leq \exp(-Na) \tag{2}$$

where B is a ball with center P and radius r. Notice that for gaussian couplings, one can take $a = \alpha . \inf_{B(P,r)} I$ for any $0 < \alpha < 1$.

If we don't suppose $\rho = 1$, the connection weights are not gaussian any more. It is not possible to derive a large deviation principle by using the ideas of Ben Arous and Guionnet. However, using a Lindeberg's argument, we are able to deduce that equation (2) remains true for our diluted networks (Moynot and Samuelides, 2001).

We are therefore able to infer the exponentially fast convergence of π_N towards δ_P (Dirac measure of P) and to deduce our main mathematical results :

2.3. The main results

2.3.1. *Propagation of chaos*

The symmetry properties of P_N (all the activation states of the neurons of a given population have the same distribution) give the following *propagation of chaos result* by using the same arguments as in (Sznitman, 1984) :

THEOREM 1.

Let k, m be two integers, and $h_1^1, .., h_k^1, h_1^2, .., h_m^2$ be bounded continuous functions from $(]0,1[^{[\![0,T]\!]})$ *to* $\mathbb{R}$*, then :*

$$\lim_{N\to+\infty}\int [h_1^1(x_1^1)...h_k^1(x_k^1)h_1^2(x_1^2)...h_m^2(x_m^2)]dP_N(x) = \prod_{i=1}^{k}\int h_i^1(x)dP^1(x)\prod_{i=1}^{m}\int h_i^2(x)dP^2(x)$$

Let us now explain this propagation of chaos result in concrete terms : at time 0, the activation states of the neurons are chosen independent from each other. But from time 1 to T, many relations take place between the neurons. We call propagation of chaos the property of the activation states (x_i^p) to tend to become **independent** random vectors when the size N of the network grows to infinity.

This propagation of chaos property is related to Boltzmanian local chaos hypothesis stated by Amari (Amari, 1972) : it gives the asymptotic independence of the activation states of the neurons.

Moreover, by taking all the functions h_j^1 and h_j^2 equal to 1, except h_i^p, one can deduce from theorem 1 that the distribution of every activation x_i^p converges towards P^p. It follows that the distribution of local fields $(u_i^p(t))_{1\leq t\leq T}$ converges towards a law $\tilde{P}^p$, which is the image of the restriction of P^p by the sigmoid function f for $t \geq 1$. This means that all the neurons of each population tend to behave as a generic asymptotic neuron, whose local field's law is $\tilde{P}^p$.

2.3.2. *Mean Field equations*

Furthermore, we proved that every law $\tilde{P}^p$ is gaussian. These laws are given explicitely in our discrete time context. We are thus able to compute their characteristics, in order to obtain *the mean-field equations.*

More precisely, we consider $(\mu^p(t), \Delta^p(t,s))_{1\leq t,s\leq T}$ the mean and covariance of $\tilde{P}^p$. In particular, we note $\nu^p(t) = \Delta^p(t,t)$.

We also consider

$$m^p(t) = \int x(t)dP^p$$

$$q^p(t) = \int (x(t))^2 dP^p$$

We note $Dh = \frac{1}{\sqrt{2\pi}}\exp(-h^2/2)$. Then we have :

THEOREM 2.

The mean and covariance of the limit gaussian distribution are given by the following recurrence relations :

$$m^p(0) = \int_{-\infty}^{+\infty} x_0 d\mu_0^p(x_0)$$

$$q^p(0) = \int_{-\infty}^{+\infty} (x_0)^2 d\mu_0^p(x_0)$$

for $1 \leq t \leq T$, *and* $p \in \{1, 2\}$,

$$\bar{\mu}^p(t) = \bar{\theta}^p + \bar{J}^{p1} m^1(t-1) + \bar{J}^{p2} m^2(t-1)$$

$$\nu^p(t) = (\theta^p)^2 + (J^{p1})^2 q^1(t-1) + (J^{p2})^2 q^2(t-1) + (\sigma)^2$$

$$m^p(t) = \int_{-\infty}^{+\infty} f(\sqrt{\nu^p(t)}h + \bar{\mu}^p(t)) Dh$$

$$q^p(t) = \int_{-\infty}^{+\infty} f^2(\sqrt{\nu^p(t)}h + \bar{\mu}^p(t)) Dh$$

for $2 \leq t, t' \leq T$, *with* $t \neq t'$:

$$\Delta^p(t, 1) = (J^{p1})^2 m^1(0) m^1(t-1) + (J^{p2})^2 m^2(0) m^2(t-1) + (\sigma_\theta^p)^2$$

$$\Delta^p(t, t') = (J^{p1})^2 C^1(t-1, t'-1) + (J^{p2})^2 C^2(t-1, t'-1) + (\sigma_\theta^p)^2$$

with, for $1 \leq t, t' \leq T - 1$,

$$C^p(t, t') = \iint f\left(\frac{\sqrt{\nu^p(t)\nu^p(t') - (\Delta^p)^2(t, t')}}{\sqrt{\nu^p(t')}} h + \frac{\Delta^p(t, t')}{\sqrt{\nu^p(t')}} h' + \bar{\mu}^p(t)\right) f(h'\sqrt{\nu^p(t')} + \bar{\mu}^p(t')) Dh Dh'$$

Those mean-field equations contain the characteristics of the limit distribution of every activation state of the neurons of the network. They depend on a small set of parameters (in particular they don't depend on the size N), and constitute a small system whose asymptotic behavior (i.e. fixed point, cycle, chaos) corresponds to the global dynamics of a broad class of large finite-size random systems.

2.3.3. *Law of large numbers*

We now state a strong law of large numbers. For our model, this result can't be derived with the dynamic mean-field approach usually used in statistical physics (Sompolinsky et al., 1988; Crisanti et al., 1990). It is the consequence of the exponentially fast convergence given by the large deviation theory. We have :

THEOREM 3.

For any $N \in \mathbb{N}$, *let* $(x_i^1, x_j^2)_{1 \leq i \leq N_1, 1 \leq j \leq N_2}$ *be a family of random variables, defined by (1). Let* ϕ *be a bounded continuous function.*

Then for $p \in \{1,2\}$, almost surely (i.e with probability 1 on the choice of the random couplings and thresholds),

$$\lim_{N \to +\infty} \frac{1}{N_p} \left(\sum_{i=1}^{N_p} \phi\left(x_i^p(t)\right) \right) = \int \phi(x) dP^p$$

In particular, for $\phi(x) = x$, we deduce the asymptotic behavior of the mean activation state of the network.

$$\lim_{N \to +\infty} \frac{1}{N_p} \left(\sum_{i=1}^{N_p} x_i^p(t) \right) = m^p(t)$$

Theorem 3 gives a convergence result for almost all the choices of the parameters of the network. This property allows us to predict the behavior of macroscopic observables of a particular realization of a finite-size network.

2.3.4. *Divergence of individual trajectories*

Moreover, the large deviation techniques allow to study the *mean quadratic distance* between two given trajectories. For $p \in \{1,2\}$, we consider $u^p(t) = (u_i^p(t))_{1 \leq i \leq N_p}$, and $v^p(t) = (v_i^p(t))_{1 \leq i \leq N_p}$, satisfying (1).

We use our large deviation methods to study the covariance between u_i^p and v_i^p. The mean quadratic distance between u^p and v^p is defined as in (Cessac et al., 1994) and (Derrida and Pommeau, 1986) :

$$(d^p(t))^2 = \lim_{N \to +\infty} \frac{1}{N_p} \sum_{i=1}^{N_p} \int [u_i^p(t) - v_i^p(t)]^2 d\tilde{Q}_N$$

Where $\tilde{Q}_N$ is the global law of the system $(u_i^1, u_i^2, v_i^1, v_i^2)$.

We prooved the following theorem :

THEOREM 4.

$$(d^p(t))^2 = 2(\nu^p(t) - \Delta^p(t))$$

$$\Delta^p(t+1) = (J^{p1})^2 C^1(t) + (J^{p2})^2 C^2(t) + (\sigma_\theta^p)^2$$

with

$$C^p(t) = \int \int DhDh' f\left(\frac{\sqrt{(\nu^p(t))^2 - (\Delta^p(t))^2}}{\sqrt{\nu^p(t)}} h + \frac{\Delta^p(t)}{\sqrt{\nu^p(t)}} h' + \bar{\mu}^p(t) \right) f(h'\sqrt{\nu^p(t)} + \bar{\mu}^p(t))$$

This result will help to understand the behavior of our networks : the time evolution of this mean quadratic distance, for close initial conditions, allows to know whether the dynamical regime of the system is chaotic.

3. Dynamics and synchronization

3.1. A model with excitatory and inhibitory populations

In order to describe excitatory and inhibitory populations in our system, we define a statistics on the weights. Parameters J (reference standard deviation) and d (mean shift) describe the synaptic weights, according to the following statements (see fig. 1):

$$\bar{J}^{11} = Jd, J^{11} = J, \bar{J}^{12} = -2Jd, J^{12} = \sqrt{2}J, \bar{J}^{21} = Jd, J^{21} = J, \bar{J}^{22} = 0, J^{22} = 0$$

Include fig. 1

For such parameters, inhibitory population has no connections towards inhibitory population. Excitatory population sends connections towards both excitatory and inhibitory populations. Inhibitory population connections have a negative shift $\bar{J}^{12}$ which is twice stronger than excitatory positive shifts $\bar{J}^{11}$ and $\bar{J}^{21}$. This is supposed to globally balance excitatory and inhibitory influences.

Notice that in our model, the probability of an excitatory connection to be negative is not zero. Thus, the real meaning of excitation and inhibition is statistical and stands on global influence of a whole population towards its receptors. This point differs from biological networks, where every neuron is either purely excitator or purely inhibitor.

3.2. Bifurcation map

3.2.0.1. *Stability* A chaotic regime regime is characterized by initial conditions sensitivity. The analysis of the divergence between two trajectories can be made at the thermodynamic limit. Such a method has been introduced by Derrida and Pomeau on the Kauffman model (Derrida and Pommeau, 1986), and extended by B. Cessac (Cessac, 1995) for one-population random recurrent fully connected neural networks. We consider two close initial conditions. We use the expression given in theorem 4 and say that our dynamics are *stable* if the mean quadratic distance $(d^p(t))^2 = 2(\nu^p(t) - \Delta^p(t))$ converges towards zero when t grows to infinity. We talk about *destabilized dynamics* if $d^p(t)$ grows in the same conditions. Such a behavior is of course directly connected to chaos.

3.2.0.2. *Synchronization* Random recurrent networks with excitatory and inhibitory populations are known to spontaneously exhibit global oscillatory signals (Amari, 1972). When the number of inhibitory neurons is large, the whole inhibitory population acts as a global inhibitory feedback loop which tends to produces synchronous collective oscillations in excitatory population. This synchronous behavior can be found in a broad class of non-linear distributed systems, including biological systems (Mac Leod and Laurent, 1996).

In our mean-field dynamical system (2), we will say that the system is in a synchronous mode as soon as the signal $m^p(t)$ (mean over neuronal activations) is not static for long time. The transition from static dynamics towards cyclic dynamics appears with a Hopf bifurcation when increasing the dispersion on the weights (parameter J). Then, the mean signal describes a periodic or semi-periodic cycle.

Include fig. 2

So the limit dynamics of the two-population model can be described twofold, i.e. as stable/destabilized dynamics and as an asynchronous/synchronous dynamics. Given some parameters (namely $\bar{\theta}^1 = 0, \sigma_\theta^1 = 0$, $\bar{\theta}^2 = 0.3$, $\sigma_\theta^2 = 0$), we iterate the mean-field dynamics for different values of J and d. The frontiers of bifurcations found in this range of parameters are displayed on Figure 2. Remark here that there is no contradiction for the neurons to be synchronous and independent in the same time : the synchronization is directly related to the temporal oscillations of $m^p(t)$, while independence corresponds to random individual fluctuations of the activation states around $m^p(t)$.

The overlapping between those two dynamical regions gives rise to four dynamical regimes. The *fixed point* regime correspond to a system which is dynamically quiescent. Every neuron tends to produce a static output. In a regime of *stationary chaos*, every neuron is dynamically active, and individual signals are aperiodic and temporally desynchronized. Such regime corresponds to a stationary gaussian process in the limit of large sizes. In a regime of *synchronized oscillations*, individual neurons produce phase-locked periodic or semi periodic signals, so that the mean signal oscillates with the same period and phase. At last, a regime of *cyclostationary chaos*, one have global oscillations on the mean signal and initial conditions sensitivity on the individual trajectories. Such regime corresponds to a cyclostationary gaussian process in the limit of large sizes.

3.3. Finite-size dynamics

We now consider the dynamics of large finite-size system. As stated in theorem 3, the dynamical regime of one given system tends to obey to the dynamics of the mean field equations. This adequation can be checked with good accuracy for systems whose size is of the order of 100 to 1000 neurons.

We ask here the question of dynamical adaptation when a change occurs in the environment of the system. We will take the simple case of a static centered gaussian random input of standard deviation σ_I. Every neuron of excitatory population is receptive so that sending an input corresponds to a change on the thresholds parameters σ_θ^1, which corresponds to a shift in the bifurcation map. The bifurcation map thus predicts the *dynamical reactivity* of the system on a given class of inputs.

Figure 3 gives an example of such reactivity, corresponding to a transition from stationary chaos to cyclostationary chaos. One can see that the system tends to spontaneously produce synchronous oscillations in presence of this static input. As long as the pattern is present in the field of receptive neurons, the system maintains its synchronized dynamics; when the pattern is put off, the system turns back to an asynchronous chaotic regime. Finally, our system spontaneously adapts its dynamics when input signal changes.

Include fig. 3

Every different input pattern will produce a different pattern of activation. Such pattern of activation both depends on the values of the input pattern and on the inner constraints of the system. The nature of the regime (stationary chaos, cyclostationary chaos, synchronized oscillations) depends on the effective distribution of the input pattern.

4. Conclusion

The introduction of a stochastic computation technique allows to derive new properties in the framework of mean-field equations of random recurrent neural networks, such as the strong law of large numbers. Then we derive dynamical properties of the mean field : the design of random recurrent networks with several populations leads to a great variety of dynamical regimes, richer in comparison with the dynamical regimes found in a single population model. In particular, one have different regimes of global synchronization, one of which including local chaotic behavior. At finite size, we finally study the dynamical reactivity of the system when presenting random gaussian input patterns. In that case, our system displays a simple example of collective organization under the influence of this changing environment.

The description of these behaviors is accurate in the limit of large sizes, and supported by rigorous convergence results. It would be of great interest to extend our mathematical proofs to more complex biologically plausible models.

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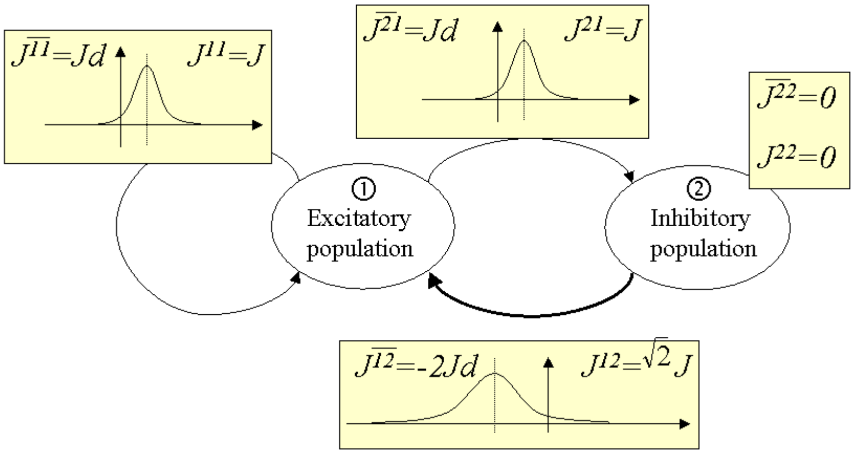

Figure 1. **Architecture of the model.** The weights have random gaussian values. Inhibitory population has no recurrent links. Inhibitory feedback is twice stronger than excitatory one.

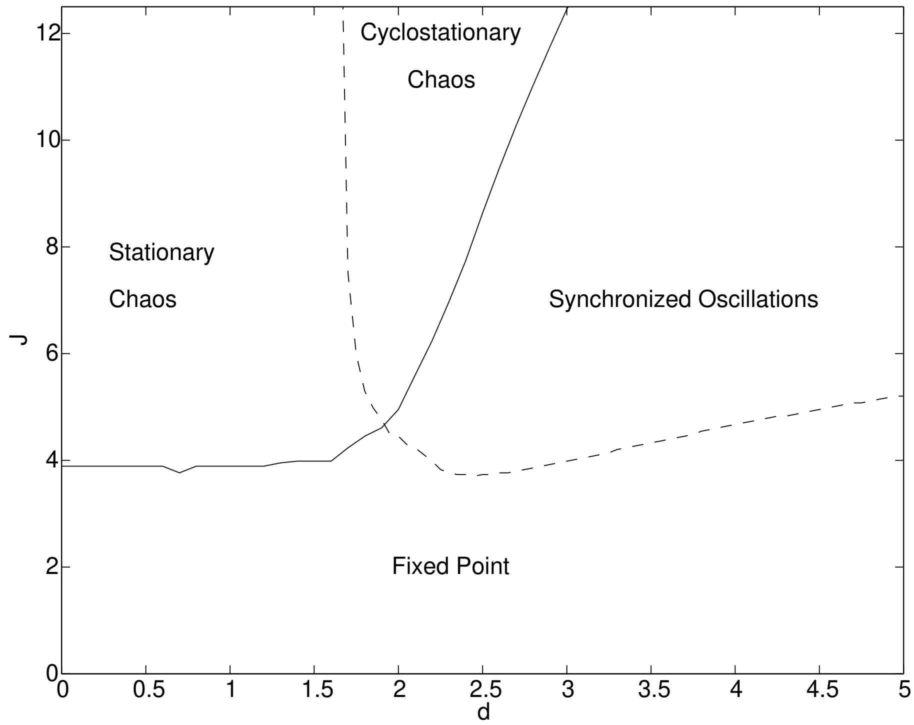

Figure 2. **Bifurcation map derived from mean-field equations**. Plain line : bifurcation between stable dynamics and destabilized dynamics. Dashed line : bifurcation between asynchronous and synchronized dynamics. 4 different dynamical regions can be described. Mean shift d is on the x-axis, and weight dispersion J on the y-axis. Other parameters are $\bar{\theta}^1 = 0$, $\sigma_\theta^1 = 0$, $\bar{\theta}^2 = 0.3$, $\sigma_\theta^2 = 0$.

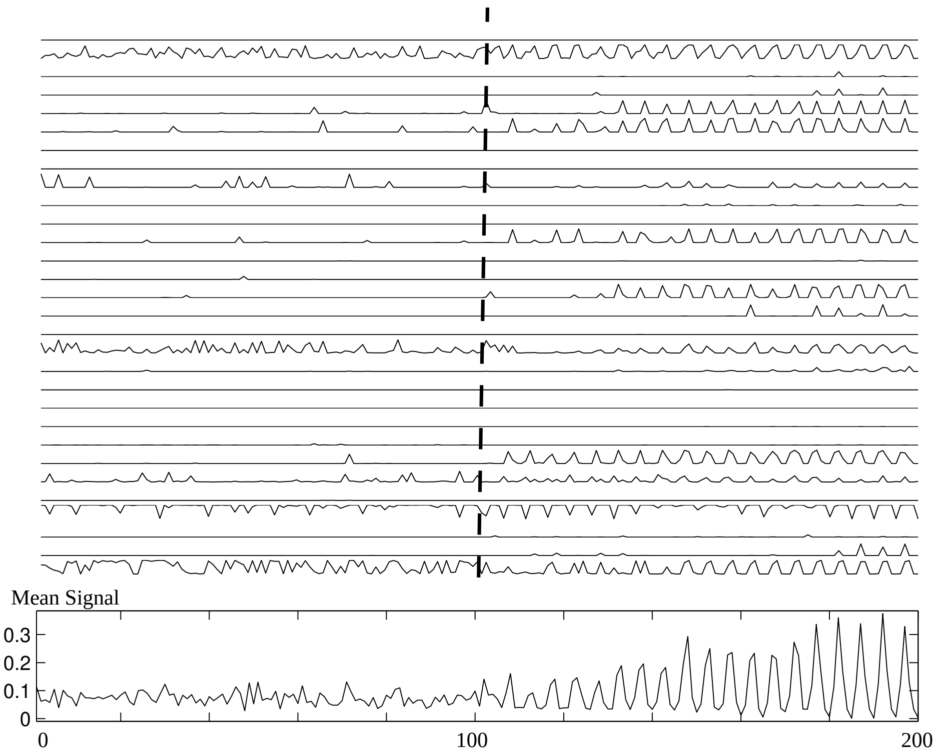

Figure 3. **Reactivity of the system to a static input in excitatory population.** 30 signals are extracted from excitatory population, and the mean signal over these 30 signals is given on lower figure. At time $t = 100$, a gaussian input is sent on excitatory population. Parameters are $N_1 = 500$, $N_2 = 500$, $J = 4.5$, $d = 2$, $\rho = 0.02$, $\bar{\theta}^1 = 0$, $\sigma_\theta^1 = 0$, $\bar{\theta}^2 = 0.3$, $\sigma_\theta^2 = 0.1$, $\sigma_I^1 = 0.3$.