Working memory and spontaneous activity of cell assemblies. A biologically motivated computational model

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Abstract— **Many cognitive tasks require the ability to maintain and manipulate simultaneously several chunks of information. Numerous neurobiological observations have reported that this ability, known as the working memory, is strongly associated with the activity of the prefrontal cortex. Furthermore, during resting state, the spontaneous activity of the cortex exhibits exquisite spatiotemporal patterns sharing similar features with the ones observed during specific memory tasks.**

Here, we propose a computational model of the prefrontal cortex within the framework of the cell assembly theory. In that framework, 'a chunk of information' refers to an associative memory and consists of an ensemble of neurons which activates coherently due to their strong interconnections. Our model consists of a recurrent network of cells whose dynamics results from the interplay between the membrane potential and the theta local field potential.

I. Introduction

In recent years, different groups have reported that in absence of any tasks or stimuli, the resting brain is not silent, but spontaneously active, and that this spontaneous activity exhibits exquisite spatiotemporal patterns of activity [1], [2], [3]. Their data suggests that during spontaneous activity, the brain activity itinerates through previously learned 'memories' or 'thoughts', in a way similar to chaotic itinerancy [4] or frustrated dynamics [5].

More than fifty years ago, Hebb proposed the cell assembly theory of cortical associative memory [6]. In this theory, each memory is defined by a cell assembly, i.e. a set of cells having strong synaptic weights between each other due to the well-known Hebbian rule of synaptic plasticity. The functional principles underlying that theory of memory has been formalized mathematically as attractor neural networks and is still a working concept in the neuroscience community for the understanding of how the brain works. In the mammalian brain, evidence suggests that memories are first formed in the hippocampus and then are transferred for long time storage in the cortex, and more specifically in the prefrontal cortex (pfc). Following that view, we assume here that the prefrontal cortex is characterized by the presence of multiple 'stored memories' in term of cell assemblies; each cell assembly being defined by a set of cells having strong interconnections. Within this framework, we expect that during spontaneous activity, i.e. in absence of any external stimulation, the different cell assemblies will be selectively reactivated in an unpredictable way.

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The working memory appears as a fundamental component in the realization of higher cognitive functions, and defines the ability to hold and manipulate a limited amount of information during short time periods [7]. The neural basis of the working memory has been widely investigated in primates by using delay to matching tasks. In these tasks, the primate has to retain specific information during a short period of time to guide a forthcoming response. Single cell recordings has shown that during this delay period, some cells located in specific brain areas had increased firing rates [8], [9], [10]. By using neuro-imaging tools, the same brain areas were observed to have increased activity [11]. Among the different areas which are believed to take part in the working memory, we count the prefrontal cortex, the posterior parietal cortex and parts of the basal ganglia and of the thalamus [12].

This paper focuses on one of the defining features of the working memory: the ability to actively hold a limited amount of information in memory for a short time, which is also called the short term memory. Following diverse biological evidence [13], we suppose here that the short term memory is an intrinsic feature of the prefrontal cortex. In that view, the manipulation of the sustained information (the second defining feature of the working memory) would result from the interplay between different brain areas. The numerous observations concerning the neural basis of the working memory have inspired many neurobiologically based computational models (e.g. [14], [15]). However, here, for the first time, we propose a model of working memory where each information is held by a specific cell assembly.

Ten years ago, Lisman and Idiart proposed a single cell model of working memory where a fundamental role was attributed to the rapid oscillations observed in the local field potential (LFP) [16] (oscillations are commonly observed in most brain areas, in association with specific brain states; e.g. the hippocampal theta (4-8Hz) observed during REM sleep). In their model, the subthreshold oscillations provided by the theta LFP combined with the after-depolarization of the membrane potential occurring after emission of an action potential can sustain the repeated firing activity of the cell at each cycle. Recently, converging evidence highlights the important role played by the theta local field potential in higher cognitive functions and more specifically in working memory [17]. Following this view, the authors introduced in a previous paper an oscillator model for a single cell characterized by two variables: the membrane potential and its phase relative to the theta LFP [18]. It was demonstrated that a large range of different dynamics resulted from the

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interplay between these two variables and it was proposed that specific dynamics could serve as the basis for a working memory.

In this paper we investigate the dynamics of these cells when bundled in large networks where cell assemblies were previously stored in the synaptic weights. After reproducing the classical paradigm of associative memory, i.e. that the partial stimulation of a cell assembly entails its entire activation, we will investigate how a cell assembly can sustain its activity when the stimulation is over. Furthermore, we will investigate the possibility to maintain simultaneously the activity of multiple cell assemblies (which can overlap) without mixing their information.

II. Methods and model

In our model, the prefrontal cortex is represented by a set of N excitatory cells, each cell having excitatory connections to the other cells (i.e. positive weights). To maintain sparse activity in this excitatory network, global inhibition is further simulated. The connectivity of the network is defined by the presence of cell assemblies. A cell assembly aims to store a piece of information in the form of an associative memory and is here defined by a subset of cells having large connecting weights. To link our study to neurophysiological considerations, a single cell model motivated by biological observations and based on oscillatory dynamics is introduced. In previous studies, we demonstrated that the dynamics of this system is characterized by the presence of a Milnor attractor. To escape from it, random noise is further simulated.

This system enables to analyze the reactivation of previously stored patterns during spontaneous activity. During working memory tasks, one or multiple cell assemblies are first reactivated during a short period of time. Then, these cell assemblies are supposed to be hold in memory; i.e. to be selectively reactivated. To facilitate their reactivation, short time plasticity is phenomenologically simulated during periods of external stimulation.

A. Creation of cell assemblies

We phenomenologically assume the presence of M cell assemblies. It implies first the identification of the cells composing each cell assembly, then the specification of the synaptic weights between cells lying in same or in different cell assemblies.

1) Identifications of the cell assemblies: To define cell assemblies in a straightforward and reproducible way, the following parameters were introduced.

- r, number of cells composing a cell assembly;
- p, proportion of cells in a cell assembly having overlap with other cell assemblies;
- q, the proportion of cells lying in two cell assemblies;
- the proportion of cells lying in more than two cell assemblies. Since in our experiments, the network size, and the number of cell assemblies were maintained small, no cells were allowed to participate to more than two cell assemblies.

For this paper, the following values were used for the parameters: $r = 10$, $p = 70\%$ and $q = 20\%$. It means that a cell assembly is composed of 10 cells and that 7 of them are shared with other cell assemblies. However, no more than two cells can be common to two cell assemblies.

2) Specifications of the weights: The connectivity of the network followed a bimodal distribution. Each synaptic weight between cells lying in a same cell assembly was assigned to a value obtained from a first normal distribution (parameters: $\mu = 0.8$, $\sigma = 0.15$) while other synaptic weights were assigned to a value from a second normal distribution (parameters: $\mu = 0.2$, $\sigma = 0.1$). Then, the total amount of synaptic weights impinging each cell was normalized to unity.

B. Definition of PFC excitatory cells

To characterize pfc cells, two phenomenological models are combined. First, following the attractor neural network theory, each cell i is characterized by its membrane potential S_i, which is modulated by the activity of the other cells through recurrent connections (Eq.1-up). Second, following the tradition of oscillator networks, each cell i is characterized by a phase ϕ_i which is driven by the intracellular membrane potential and modulated by additional inputs (Eq.1-down). As a result, each cell's dynamics results from the non linear coupling between two simpler dynamics having different time constant, in a reminiscent way of the working memory cell proposed by Lisman and Idiart [16]. The state of each cell is defined by $\{S_i, \phi_i\} \in \Re \times [0, 2\pi[$ $(i \in [1, N])$ and evolves according to the following equations:

$$\begin{cases} \frac{dS_i}{dt} &= -S_i + \sum_{j=1}^{N} w_{ij} R(S_j) + \Gamma_i \\ \frac{d\phi_i}{dt} &= \omega + (\beta - \Lambda_i) \sin \phi_i \end{cases} \quad (1)$$

with w_{ij}, the synaptic weight between cells i and j, $R(S_j)$, the spike density of the cell j, and Γ_i, the sum of all inputs to the cell. In the second equation, ω and β are respectively the frequency and the stabilization coefficient of the internal oscillation. Λ_i represents the variation of the membrane potential according to impinging inputs.

The spike density is defined by a sigmoid function:

$$R(x) = \frac{1}{2} \left(\tanh \left(g(x - 0.5) \right) + 1 \right), \quad (2)$$

Following the oscillator dynamics theory, the cosine of the phase stands for the oscillation current. Here, this current will modulate the dynamics of the membrane potential S, and the couplings between the two equations appear as follows:

$$\Gamma_i = \sigma(\cos \phi_i - \cos \phi_0) + I_i \quad (3)$$

$$\Lambda_i = \rho S_i \quad (4)$$

where ρ and σ modulates the coupling between the internal oscillation and the membrane potential, and ϕ_0 is

the equilibrium phase obtained when all cells are silent ($S_i = 0$); i.e. $\phi_0 = \arcsin(-\omega/\beta)$. I_i represents the driving stimulus which enables to selectively activate a cell. In our simulations, to observe how the information is sustained in the network, stimuli will be applied during very short time periods.

For this paper, the following values were used for the parameters: $\omega = 1$, $\beta = 1.2$, with $g = 10$, $\rho = 1$ and $\sigma = .96$.

C. Inhibition and noise

The dynamics of a one-cell network can be fully theoretically characterized, and the dynamics of two coupled cells can be characterized in limit cases [18]. However, for a growing number of cells, mathematics becomes intractable and it appears difficult to tune the different parameters to avoid the saturation of this excitatory network. Here, to provide robust dynamics in a wider range of the parameter space, one inhibitory cell is introduced which prevents saturation and provides sparse coding.

The inhibitory cell receives synaptic connections from all pfc cells, summates them linearly and, according to some threshold, inhibits all pfc cells. The following additional term is added to Eq.3:

$$\Gamma_i \rightarrow \Gamma_i - \Delta\left(\gamma\left(\sum_{i=1}^{N} R(S_i) - \kappa N\right)\right) \tag{5}$$

where $\Delta(x) = x$ for $x > 0$ and 0 elsewhere; κ (in $\%$) defines a threshold triggering the inhibition and γ defines the strength of the inhibitory cell. Here we have $\kappa = 0.03$ and $\gamma = 0.1$

In Eq.1, it appears that the system is in a stable state when $S_i = 0$ and $\phi_i = \phi_0$. To avoid global stabilization, random noise continuously excites the network and Eq.3 becomes:

$$\Gamma_i \rightarrow \Gamma_i + \Upsilon_i \tag{6}$$

Noise is obtained from a gaussian distribution ($\mu = 0.02$ and $\sigma = 0.01$) and is applied only to a small subset of the network (6% of cells). Every 200 computational steps new noise values are assigned to a new subset of cells chosen randomly.

D. Working memory tasks

It has been demonstrated in previous papers that these cells could serve as the neural correlate for working memory in small network and in the absence of cell assemblies [18]. We experienced that the story becomes more complex when facing large networks containing multiple cell assemblies.

Here, we observed that if the transient stimulation of part of a cell assembly can sustain the activity of the entire cell assembly, it can also propagate to other cell assemblies having overlapping cells. While this could be an acceptable and viable choice, here, our goal was different: We wanted specific stimuli to drive only the sustained activity of their associated cell assemblies.

To this aim, the presentation of the input was associated with a period of Hebbian short term plasticity. Since the stimulus forces the activation of specific cell assemblies and since inhibition tends to prevent the simultaneous activation of multiple cell assemblies, that period of short term plasticity resulted in increasing the weights between cells of the stimulated cell assembly. We believe that the proposed mechanism can be related with growing evidence showing that attention-like processes are associated with period of short term plasticity [19], [20]. In that view, the period of external stimulation should be associated with a state of attention.

E. Experiments

This paper aimed to observe the activity of the cells, and more specifically of the cell assemblies, following two different scenario. First, the spontaneous activity of the network was observed in absence of external stimuli. Second, to analyze if our network can work as a working memory, selective inputs I_i were transiently applied to a subset of one or several cell assemblies. In this latter case, first we specified the number of cell assemblies to externally stimulate, then each cell assembly was sequentially stimulated during 10 computational time steps. However, in agreement with the cell assembly theory, only a small proportion of cells composing the cell assemblies were stimulated (only 40% of cells).

At each computational time step the activity of all cells was monitored and we computed the proportion of reactivation of the different cell assemblies.

III. Simulation Results

Before focusing on the main topic of this paper, i.e. the analysis of the activity of cell assemblies during spontaneous activity and during working memory tasks, we first discuss briefly the dynamics characterizing the activity of one single cell (see also [18]).

A. One cell dynamics and the Milnor attractor

In absence of external stimuli ($I = 0$ in Eq.3), the dynamics stabilizes to the resting state $M_0 = (S_0 = 0, \phi_0)$ (with $\beta > w$, such that $\phi_0 = \arcsin(-w/\beta)$). The linear stability of that resting state is obtained by analyzing the Jacobian of the system:

$$\left\{\begin{array}{lcl} 0 & = & -S + \sigma(\cos\phi - \cos\phi_0) \\ 0 & = & \omega + (\beta - \rho S)\sin\phi \end{array}\right. \tag{7}$$

at the resting point M_0:

$$DF|_{M_0} = \left(\begin{array}{cc} -1 & -\sigma\sin\phi_0 \\ -\rho\sin\phi_0 & \beta\cos\phi_0 \end{array}\right) \tag{8}$$

The stability of the system depends of the product $\mu = \sigma\rho$. Below a critical value $\mu < \mu_c$ ($\mu_c = \beta\cos\phi_0/\sin^2\phi_0 \approx 0.96$), M_0 is stable. Above the critical value, M_0 becomes unstable. Further analyses show that the system has a second fixed point $M_1 = (S_1, \phi_1)$ which is unstable when M_0 is stable (with $S_1 > 0$), and which becomes stable when M_0

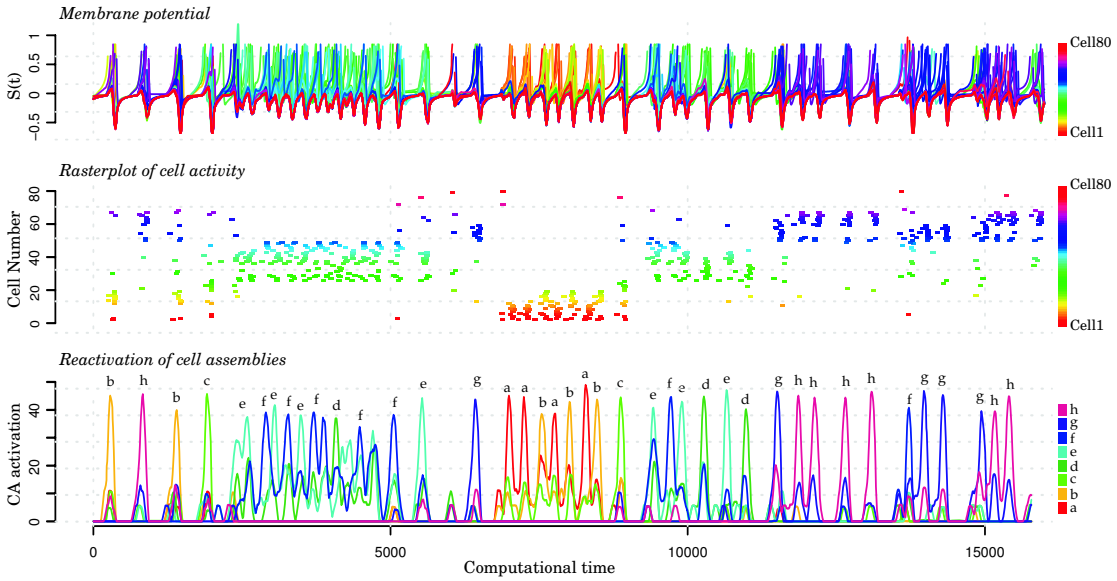

Fig. 2. Spontaneous activity in a 80 units network containing 8 overlapped cell assemblies of 10 cells each. The upper figure shows the membrane potential of each individual cell (80 cells, each cell one color). The middle figure shows a rasterplot of the activity of each individual cell. The cell has activity if its spike density is larger than 0.5. The lower figure shows the reactivation of the different cell assemblies (each assembly has its own color and letter). It appears that periods of no-activity alternates with periods of activity during which specific cell assemblies are preferentially activated.

is unstable (with $S_1 < 0$). At the critical value, the stability of the two fixed points switches which defines a transcritical bifurcation.

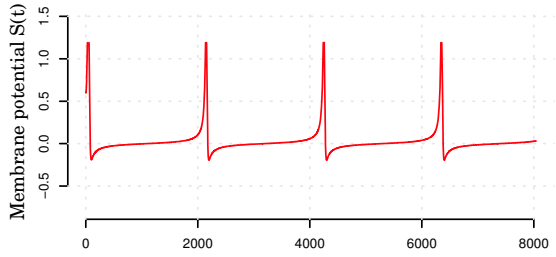

Fig. 1. Evolution of the membrane potential of one cell at the Milnor attractor. The parameters are tuned such that the dynamics lies exactly at the Milnor attractor, at the bifurcation point between two fixed point dynamics.

Since the stable and the unstable fixed points merge at the critical point, the topology of the attractor no longer defines a closed set: orbits can escape from the resting state. Accordingly, most of initial conditions are attracted by the resting state but then they can be kicked away from it by infinitesimal perturbation. At that point, a complete cycle of the phase occurs and the state is attracted again by the resting state. It means that the unstable orbits cross the attractive set. This particular dynamics defines a Milnor attractor, i.e. an attractor according to the extended definition proposed by Milnor [21] (see also [22], [23]).

Figure 1 shows the evolution of the membrane potential of one single unit when $\mu = \mu_c$ (here, 0.96). As explained, the dynamics is successively attracted to the Milnor attractor ($S = 0$), then escapes from it because of infinitesimal perturbation, to fall again in the basin of attraction of the resting state. In the simulation, a tiny perturbation ($I = 0.0001$) is constantly applied to the cell, to force the escape of the resting state.

B. Spontaneous activity

In the previous section, we have shown that the single cell dynamics is characterized by a Milnor attractor. In this section, a network of $N = 80$ cells is simulated. We suppose that this network contains $M = 8$ cell assemblies, each cell assembly containing $r = 10$ cells. Cell assemblies are overlapping in such a way that $p = 70\%$ of their cells belong to other cell assemblies, with no more than $q = 20\%$ of cells in common between two cell assemblies (see Section II-A for more details).

Figure 2 shows the spontaneous activity of the network, i.e. in absence of any external stimuli. The upper part of the figure shows the evolution of the membrane potential for the 80 cells (each cell is represented by a different color). From comparisons with Figure 1, it appears that the Milnor attractor is still playing an important role here: while global inhibition tends to stabilize the network, the presence of diverging orbits at the resting state is such that any tiny perturbation is likely to kick out one cell out of the attractor, and an oscillation occurs.

The middle part in Fig. 2 shows cells activity. Each period of reactivation is associated with the activation of a specific subset of cells. Lower part in Fig. 2 quantifies the proportion of each cell assembly which is activated at each time step. It appears that individual cell assemblies (characterized in the

figure by a different color and letter) are likely to be activated separately. During the observed period, all cell assemblies are reactivated. It has to be noted that since cell assemblies are overlapping, the total reactivation of a cell assembly is necessarily associated with the partial reactivation of other cell assemblies.

The activity of the network could be explained in the following way: when one cell is activated, it tends to activate the cells belonging to the same cell assembly. The simultaneous activation of multiple cell assemblies is prevented by the global inhibition. The activated cells will undergo one oscillation before going back to the resting state, nearby to the Milnor attractor. At that time, any cell can be kicked out of the attractor leading to the activation of a different or the same cell assembly. As a result, the spontaneous activity of our system is characterized by the Milnor attractor which provides a kind of reset of our network, enabling the activation of the different stored memories. However, since cell assemblies are overlapping, the reactivation of one specific cell assembly will tend to reactivate the cell assemblies sharing common cells and a visible sequential structure is visible. Still, the pathway followed along the different memories defines a chaotic itinerancy [24], [25], [26].

C. Working memory

In this section, after applying external stimulus during a short time period to part of one or several cell assemblies embedded in the network, we observe the network's ability to sustain the activity of these cell assemblies in agreement with the theory of working memory. Figure3 shows the activity of the 80 cells and the level of activity of the 8 cell assemblies, after applying an external stimulation during a few (10) computational steps. In Fig.3a, part (40%) of one cell assembly was stimulated. In Fig.3b, part (40%) of three cell assemblies were sequentially stimulated.

In agreement with the theory of associative memories, in both scenario, rasterplot figures indicate that the stimulation of 40% of a cell assembly results in its complete reactivation. During the first activation, the cells composing the cell assembly are activated at slightly different phases. However, in their subsequent activations, all cells are activated at same phases. Again, global inhibition prevents the simultaneous activity of a large number of cells (here, the simultaneous activity of 3 cells ($\kappa N = 0.03 * 80$) triggers inhibition). As a result, we observe that only one cell assembly can be activated at a given time step.

Figure 3a shows the sustain activity of one cell assembly. After the external stimulation, a cycle of oscillation occurs and the network is attracted again to its resting state. When reaching the resting state, some cells are kicked away to diverging orbits, attracting with them their associated cell assembly. As explained Section II-D, during the stimulation period, short time plasticity was simulated, and the weights of the activated cells (i.e. of the selected cell assembly) were slightly increased. As a result, the cell assembly having the largest weights is activated and our model successfully leads to the sustained activity of the desired memory.

Figure 3b shows the sustain activity of three cell assemblies (noted by the letters a, d, g). After the external stimulation, the network is attracted to its resting state, and again, we can observe the dynamics generated by the presence of diverging orbits crossing attracting orbits. The balance between excitation and inhibition results in the competition of these 3 cell assemblies which leads to complex patterns of activity. As a result, the three cell assemblies show clear sustained activity, and these reactivations occur at different times. This result appears important since their simultaneous reactivation would mix them and would prevent any possibility to decode the utile information embedded in the network. Together, these results confirm that our model satisfies one important defining feature of the working memory: The ability to maintain short term memory.

IV. Discussion

Converging evidence are suggesting that the prefrontal cortex plays an important role both in the storing of long term memories, and in the short-term maintenance and manipulation of information in goal-directed tasks, i.e. in working memory. Here, a computational theory of the pfc is proposed focusing only on the ability to maintain information during short periods after stimulation.

In a previous study [18], we developed the equations of a biologically motivated cell characterized by two variables, the cell's membrane potential and the cell's phase of firing activity relatively to the theta local field potential. Theoretical analyzes of the one-cell and two-cell networks revealed the presence of numerous dynamics, one of them being the presence of a Milnor attractor. When the cell is in the resting state, a simple perturbation activates an oscillation of the membrane potential followed by its attraction to the resting state. As a result, in presence of tiny noise, this leads to successive cycle of reactivation, i.e. to the maintenance of the information (Figure 1). This leaded us to propose that a simple unit could serve as the basis for a working memory.

Still, the complex dynamics provided by a single cell is not enough to have a working memory. Here, we developed the previous model to a more realistic application. In agreement with the cell assembly theory of cortical associative memories [6], we hypothesize that each memory, or chunk of information, is represented by an ensemble of cells interconnected with large synaptic weights [29]. As a result, multiple overlapping cell assemblies were phenomenologically embedded in the network.

First simulations showed that the spontaneous activity of the network is dynamically switching between the different cell assemblies previously stored (Figure 2). This is supported by recent biological reports; for example, the spatiotemporal pattern of activity of cortical neurons observed during thalamically triggered events are similar to the ones observed during spontaneous events [2]. In our model, the Milnor attractor plays a crucial role: First, the diverging orbits prevent the network from stabilizing to fixed point

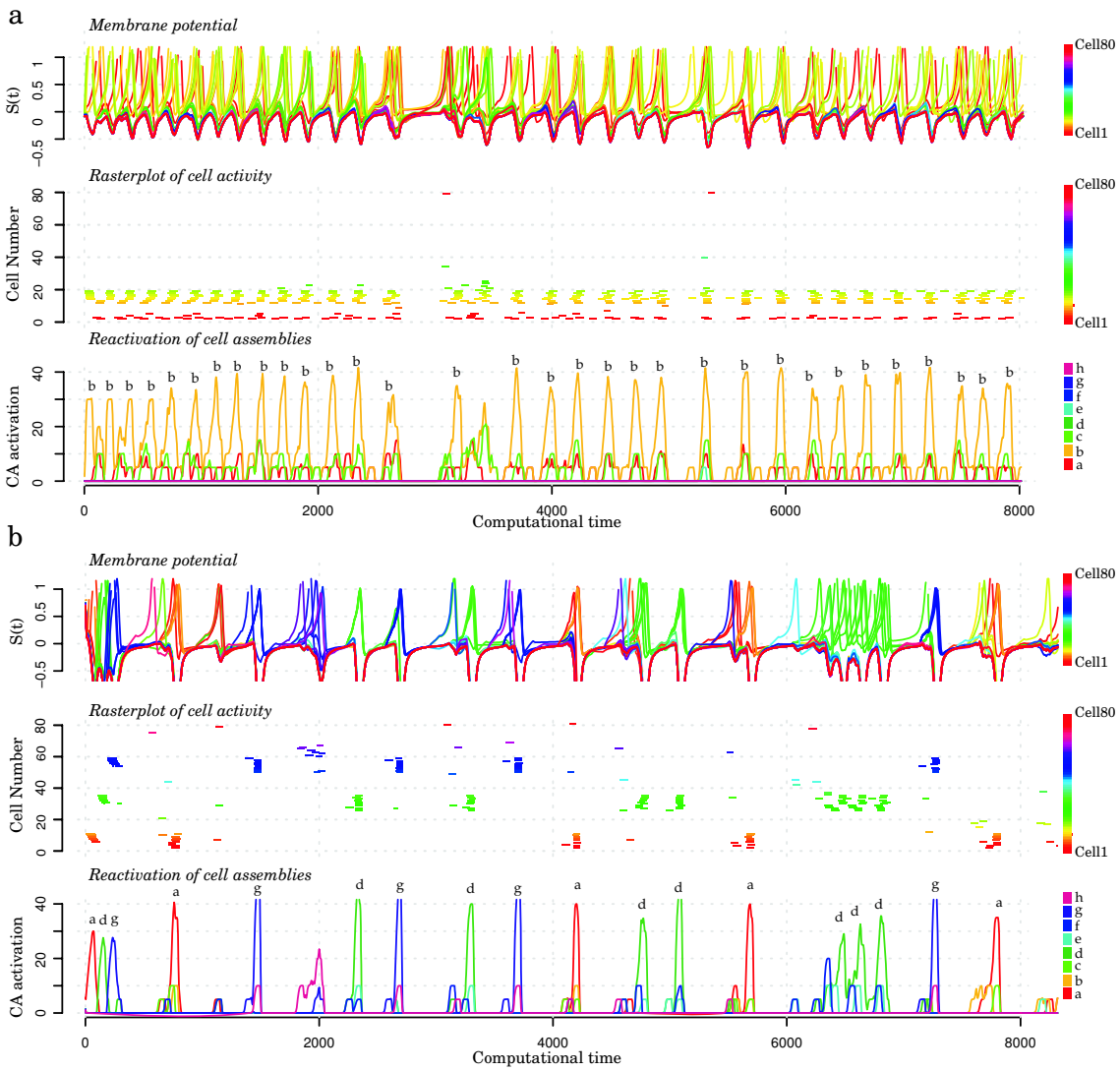

Fig. 3. Working memory in a 80 units network containing 8 overlapped cell assemblies of 10 cells each. In both figures a and b, the upper figures shows the membrane potential of each individual cell (80 cells, each cell one color). Middle figures show the rasterplots of individual cells activity (a cell is said to be in an active state if its spike density is larger than 0.5 ($R(S_i) > 0.5$) . The lower figures show the reactivation of the different cell assemblies (each assembly has its own color and letter). (a) An external stimulus was impinging part (40%) of one cell assembly during a short transient (10 computational time steps). As a result, this cell assembly is continuously activated as a short term memory. (b) External stimuli are successively applied to part (40%) of 3 cell assemblies (each CA is stimulated during 10 computational time steps). After stimulation, we observe that these 3 cell assemblies have sustained activity.

dynamics. Second, the attracting orbits force the network to go back to the quasi-stable state where it is receptive to second order dynamics of the internal state and to external stimuli. During that "receptive" or "attention-like" state, a different information can be reactivated, which prevents the network to be "occulted" by one information. As a result chaotic dynamics is observed, in agreement with numerous observations reporting that the brain is better characterized by complex dynamics than by fixed point attractors [30], [31], and with computational studies reporting that chaotic dynamics plays important role in improving the formation of memories [32], [28] and is necessary to itinerate through them during spontaneous activity [4], [33].

Second simulations tested the capacity of the network to transiently hold specific memories triggered by external stimuli. To reliably and robustly enforce the activity of the triggered cell assemblies, a short period of synaptic plasticity was simulated, reproducing attention-like processes [19], [20]. Results indicate first that the stimulation of part of the cell assembly leads to its entire activation, as predicted by the

theory of associative memory. Second, we confirmed that the activity of one or multiple cell assemblies can selectively be maintained in agreement with the working memory feature observed in biological networks (Figure 3).

Following previous studies [34], [35], information in our model is stored in the recurrent connections of the network and is reactivated through spatio-temporal patterns of activity. In our model, cells from a same cell assembly are synchronously reactivated while different cell assemblies are reactivated at different phases. This is a requisite of information processing for decoding information and for solving the famous binding problem. For example, let us imagine that we have to remember two objects, a red triangle and a green square. If all cells representing these two objects were reactivated simultaneously, the components and features would be mixed up and it would become impossible to know if we had a red triangle, a red square or a complex image represented by all these cells (see also [36] for a formalization of the binding problem) . Accordingly, our model solves the binding problem by reactivating the different cell assemblies at different phases.

Dynamics in our model is tightly related to the presence of the different cell assemblies. To optimize the network, it is important to understand how these cell assemblies are created in brains. As suggested by numerous biological reports (e.g. [27]), we believe that the formation of these cell assemblies results from the transfer of hippocampal information. In that view, during behavioral and/or attentional tasks, information is first acquired and stored as cell assemblies in the CA3 recurrent connections (the CA3 is part of the hippocampus and is characterized by massive recurrent connections). Then, during sharp waves events occurring during sleep or immobility, these cell assemblies are reactivated and are pushed to the prefrontal cortex for long term storage. Future work should link our hippocampal computational model already described in a previous paper [28] to the cortical model proposed here.

To summarize, we are proposing a compact, effective, and powerful dynamical system of working memory which may be a great platform for further conceptual extensions and abstractions of information processing. The main features of our computational model are the following. First, we are simulating networks of interconnected cells, where cells representing specific "objects", memories or "chunks of information" are bound together using synaptic plasticity and where global inhibition prevents the saturation of the network. Second, the dynamics is characterized by a Milnor attractor which enforces chaotic itinerancy between the different remembered representations. As a result, our network can act as a working memory, and the information conveyed during spontaneous activity and working memory tasks appears similar. As the famous Colombian neurophysiologist Rodolfo Llinas uses to say: "A persons waking life is a dream modulated by the senses"[37].

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