# Recovery of Sequential and Non Sequential Memories with a Neural Mass Model

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Abstract:     A neural model for the recovery of learnt patterns is presented. The model simulates the theta-gamma activity associated to memory recall. Two versions of the model are described: the first can learn generic patterns without a given order, while the second learns patterns in a specific sequence. The latter has been implemented to overcome the limited recovery capacity of the former. The network is trained using Hebbian and anti-Hebbian paradigms, and exploits excitatory and inhibitory mutual synapses. The results show that autoassociative memories for storage and recovery of multiple patterns can be built using biologically inspired models which simulate brain rhythms, and that the model which learns sequences can recover much more patterns.

## 1 INTRODUCTION

The execution of complex tasks requires the integration of many different pieces of information spread across the whole brain. A widely used concept to describe the large-scale integration in the brain is the Neural Assembly (NA), which is defined as the ensemble of neurons that take part in the realization of a single cognitive task.

A popular hypothesis, named "binding by synchronization" (Varela et al., 2001); (Singer, 1999), suggests that the formation of NAs is obtained through the phase synchronization of neural groups in the gamma band (>25Hz). According to this hypothesis, a NA can be formed rapidly and can rapidly be disrupted under the influence of external or internal events to make place for alternative NAs. Furthermore, the recent observation that gamma rhythms are modulated by slower theta rhythms (4-7Hz) suggests the idea that the construction and degradation of gamma rhythms is under control of theta oscillations (Canolty et al., 2006); (Doesburg et al., 2009).

Several mathematical models in past years described the formation and segmentation of NAs, simulating how the brain can assign a neural group to the correct assembly, while maintaining it separated from neural groups which belong to other assemblies. However, these models used very simple oscillating neural units, such as Wilson Cowan or relaxations oscillators (Wang et al., 1990); (von der Malsburg and Buhmann, 1992); (Wang and Terman, 1997); (Ursino et al., 2003); (Ursino et al., 2009), which are unable to simulate realistic brain rhythms. A recent work addresses the same issue using a more sophisticate model, able to mimic the electrical activity in cortical regions (Cona et al., 2012). Using a mathematical model for the simulation of brain activity, known as neural mass model (NMM), the authors proposed a neural architecture that can learn different NAs and evoke them separated in time. However, they were able to evoke just three gamma oscillations within each theta cycle, while many works suggest that this number can be between 4 and 7 (Cowan, 2000); (Miller, 1956).

The model itself suggested how to overcome this limitation: by visual inspection of the simulated waveforms, one can observe that most of the time between two consecutive gamma oscillations is 'wasted' to determine which NA should follow. So we inferred that, if patterns are learnt in a specific sequence, the model should be able to evoke much more NAs within a single theta cycle. This corresponds to psychophysical observations.

In the following, we will show the architecture of the NMM used to memorize non-sequential NAs and the changes adopted to learn a sequence of NAs. The different performance of the two models will be outlined.

## 2 METHOD

### 2.1 The Cortical Column

The fundamental unit of our network is the model of a cortical column. It consists of four interconnected neural populations (Figure 1), which are pyramidal cells, excitatory interneurons and inhibitory interneurons with slow and fast kinetics (respectively referred to as $GABA_{A,slow}$ and $GABA_{A,fast}$). The interaction of these populations allows the cortical column to generate different oscillatory activities, while the output of the model (the simulated EEG signal) is given by the membrane potential of the pyramidal cells. See Ursino et al., (2010) for more details on the model.
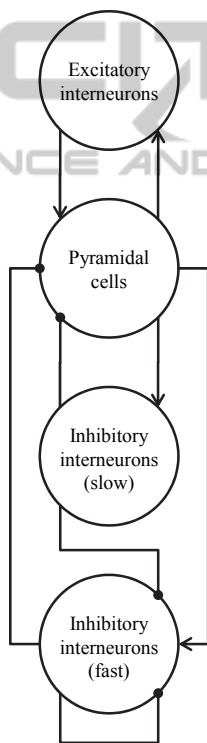


Figure 1: Connectivity between neural populations in the model of a cortical region.

### 2.2 Recall of Non-sequential Patterns

The neural network consists of 2 layers ($L_1$ and $L_2$) of $M \times N$ cortical columns each, (Figure 2). The first receives the external input and sends its output to the second layer. The second generates the cortical output. The network can learn patterns (NAs) of features. Each feature corresponds to a position in both layers and therefore is associated with two oscillatory columns, one in $L_1$ and one in $L_2$.

Each unit in $L_1$ and $L_2$ is represented through a cortical column, according to the diagram in Figure 1. Moreover, units in the same layer communicate through lateral synapses, according to the schematic representation in Figure 3. These synapses were learnt via Hebbian or anti-Hebbian mechanisms, during a training (storage phase) in which seven complete patterns (without any lacking feature) were given separately to the network. After training, the network was tested using incomplete patterns as inputs (recovery phase).
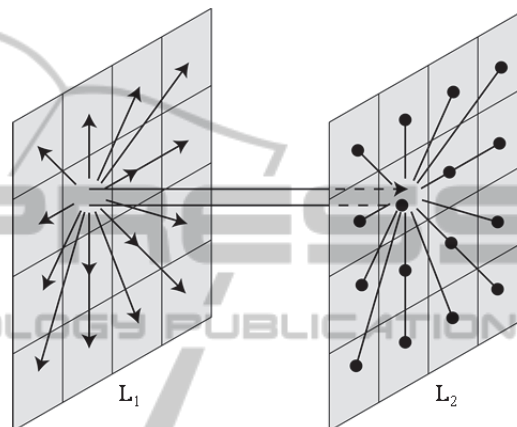


Figure 2: Architecture of the two layers: arrows indicate connections toward pyramidal cells, while circles indicate connections toward $GABA_{A,fast}$ interneurons.

During the recovery phase $L_1$ works as an autoassociative memory, i.e., it serves the main function of recovering the lacking information when the patterns are not totally stimulated. Hence, just a few features are sufficient to evoke the whole NA they belong to. The units in $L_1$ exhibit an oscillatory activity in the theta band which drives $L_2$. This behaviour is obtained by training the excitatory lateral connections in $L_1$ with the Hebb rule, i.e., reinforcing excitation among features which belong to the same pattern and so are simultaneously active. The excitation between features belonging to the same NA ensures the complete recovery of lacking information. However, excitation would lead neurons to the upper saturation (i.e., to a maximal excitation). The theta oscillation emerges thanks to the presence of inhibitory populations with slow synaptic kinetics ($GABA_{A,slow}$ interneurons) which progressively inhibit units through synapses with a time constant of 34 ms.

$L_2$ plays two roles: it makes the cortical columns oscillate synchronously in the gamma band when they encode for the same NA (binding), and out-of-synch if they belong to different NAs

(segmentation). The synchronicity is obtained through lateral inhibitory synapses, trained with the Hebb rule which link pyramidal neurons in the pre-synaptic units to $GABA_{A,fast}$ interneurons in all units which encode for the same NA(Figure 3). As demonstrated in previous works (Bartos et al., 2002); (Bartos et al., 2007); (Ursino et al., 2010), reciprocal fast inhibition is a powerful mechanism to ensure synchronization of gamma rhythms. As a consequence of this mechanisms, all units in the same pattern become part of a bigger gamma oscillator. The segmentation is obtained by training another set of synapses from pyramidal to $GABA_{A,fast}$ interneurons with an anti-Hebbian rule (i.e., a synapse is reinforced when the pre-synaptic unit is active, but the post-synaptic unit is silent). The latter synapses, however, have a much faster dynamics compared with the previous ones (rising time less than 1 ms). These can simulate ultra-fast AMPA synapses. This mechanism induces a very fast inhibition between columns in different patterns of $L_2$, so that different features in different patterns tend to desynchronise (Cona et al., 2012).
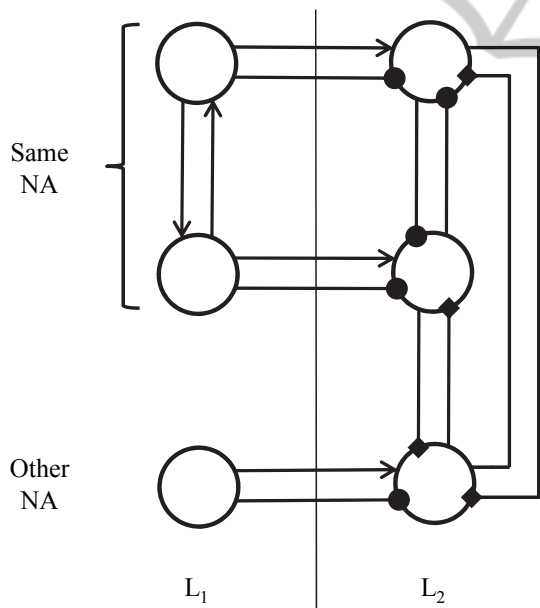


Figure 3: Connections between 6 regions, 3 in $L_1$ and 3 in $L_2$. The 4 upper regions encode for features belonging to the same NA, while the lower 2 encode for a feature belonging to another NA. Arrows, circles and squares represent respectively pyramidal-pyramidal connections, pyramidal-$GABA_{A,fast}$ connections and fast AMPA-mediated pyramidal-$GABA_{A,fast}$ connections.

In conclusion, ultrafast anti-Hebbian synapses from pyramidal to $GABA_{A,fast}$ neurons ensures a very rapid desynchronization of units, while slower

Hebbian synapses from pyramidal to $GABA_{A,fast}$ neurons ensure synchronization in the gamma range.

## 2.3 Recall of a Sequence of Patterns

To recall patterns in a specified sequence we added a third layer ($L_3$) whose cortical columns are connected in the same way as columns in $L_2$. Units in $L_2$ target units in $L_3$ which belong to the same NA. On the other hand, units in $L_3$ target units in $L_2$ which belong to the next NA in the sequence (Figure 4). The connections between $L_2$ and $L_3$ are mediated by the same synapses involved in the generation of the gamma rhythm. These are learnt with the Hebb rule during a training period, in which the different patterns in a sequence are given to the network in the correct order, delayed by 25 ms each. Subsequently, the network is tested by providing just the first pattern of the sequence as an input to $L_1$. Thanks to the trained synapses, when a NA pops out in $L_2$ it pops out also in $L_3$ with a half gamma cycle delay. In turn, a NA in $L_3$ evokes the next one in $L_2$ with the same delay. As a consequence, if the first NA of the sequence is evoked in $L_2$, all the other NAs will follow with a gamma period delay each.
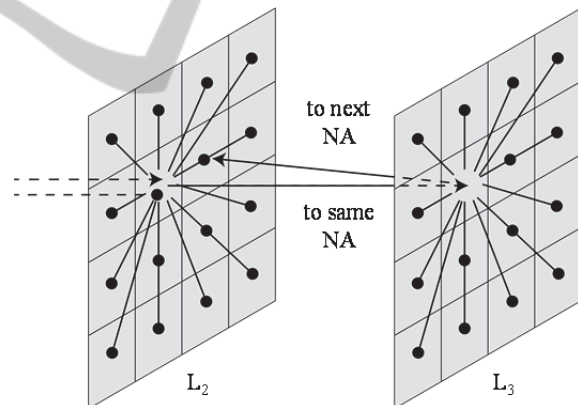


Figure 4: Architecture of the third layer and its connections with $L_2$: as in figure 2, arrows indicate connections toward pyramidal cells, while circles indicate connections toward $GABA_{A,fast}$ interneurons.

## 3 RESULTS

Figure 5 shows the behaviour of a NMM that has learnt 7 simultaneous non sequential patterns. Note that in $L_1$ all the NAs are activated and oscillate in phase in the theta frequency range (4Hz in this case). In $L_2$ the NAs are activated one at a time, but in each theta cycle just a few (2 or 3) gamma oscillations take place. Panel c shows that, when a NA

completes its gamma oscillation (and thus stops inhibiting other NAs) all the other ones compete to start the new cycle. However, due to their mutual inhibition, none of them prevails immediately and it takes almost 50 ms for a new pattern to win the competition. This delay prevents the system from evoking more than 2-3 oscillations within a theta cycle. All seven patterns can be recovered, but in different theta cycles.

This observation suggests that, if at the end of a gamma impulse one of the patterns is privileged with respect to the other ones, the delays between the oscillations in $L_2$ can be avoided, thus allowing for the appearance of a greater number of gamma impulses within a theta cycle.
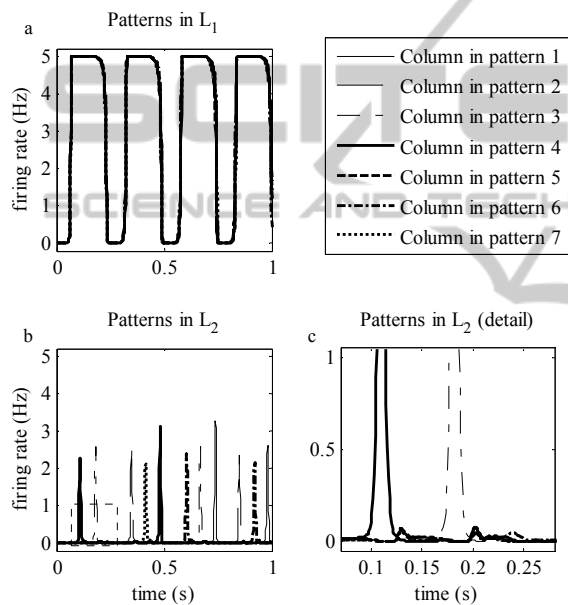


Figure 5: Activity of 7 non-sequential patterns. Panels a and b show the activity of 7 cortical columns (one for each learnt pattern) in $L_1$ and $L_2$ respectively. Panel c shows a zoom of the area within the dotted boundary in panel b.

Figure 6 shows that the modified network, which learns a sequence of patterns in a well-established order, can recover all 7 NAs within a theta cycle, in response to the first pattern given as input. This is due to the fact that at each gamma period one NA in $L_2$ receives an additional excitation from the previous NA in $L_3$, thus greatly unbalancing the competition between the different NAs to emerge.

# 4 CONCLUSIONS

We can draw some considerations on the results

obtained. The main point is that this modelling study suggests a possible neural basis for an everyday phenomenon: it is much easier to keep in mind objects or concepts assigned in a given order, than totally uncorrelated ones. Furthermore, the two models can afford two alternative problems: i) to store and recover different patterns provided simultaneously; ii) to store and recover a sequence of patterns, so that the first can evoke all the others in the assigned order.
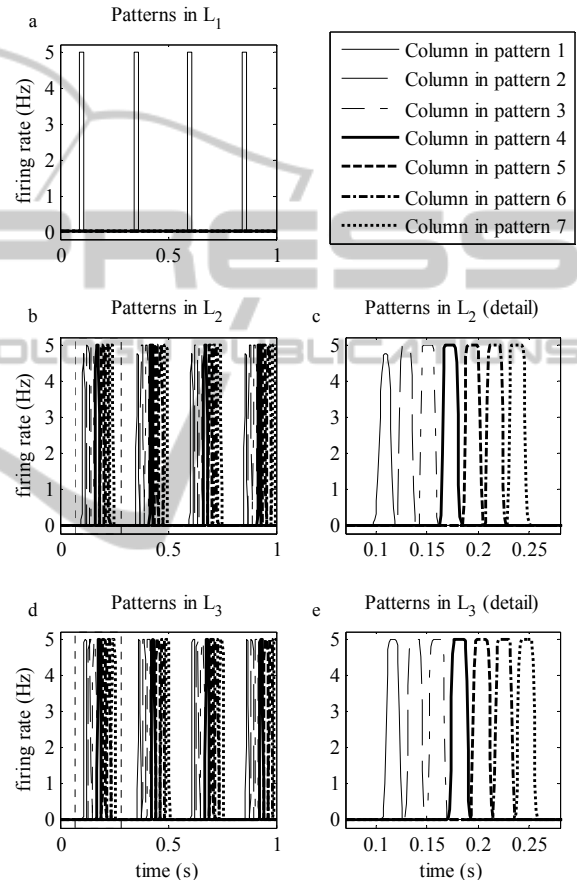


Figure 6: Activity of 7 patterns in a sequence. Panels a, b and d show the activity of 7 cortical columns (one for each learnt pattern) in $L_1$, $L_2$ and $L_3$ respectively. Panels c and e show zooms of the areas within the dotted boundaries in panels b and d respectively.

Furthermore, the present model emphasizes the strict relationship existing between theta and gamma rhythms in memory storage and recall, a behaviour which has been observed in many memory tasks, such as during phase precession (Lisman, 2005) and the formation of memories (Bikbaev and Manahan-Vaughan, 2008) in the hippocampus.

A point which requires a brief discussion is the learning capacity of the network. In this work we

trained the network with just 7 NAs, but there is no theoretical limit to the number of NAs that can be learnt, as long as there are enough cortical columns to represent all features and the NAs are orthogonal. The analysis of the network performance with non-orthogonal NAs (i.e., with shared features) and the comparison with other neural models for data storage and recovery will be the subject of future works.

A problem related to learning a longer sequence is that the recall of the whole sequence in $L_2$ would last more than one theta cycle; hence the following theta cycle would begin in the midst of the previous sequence. In this case a reset mechanism is required: this should be realized to inhibit columns in $L_2$ and stop the sequence when the theta cycle ends. It is possible that dopamine plays a role in this process.

This study underlines the great value of mathematical models as hypothesis generators, since they allow exploration of all the mechanisms involved, even those that are practically inaccessible with a purely experimental approach.

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