Associative Recall in Non-Randomly Diluted Neuronal Networks

Luciano da Fontoura Costa
Cybernetic Vision Research Group
Instituto de Física de São Carlos
University of São Paulo
13560-970 São Carlos, SP, Brazil
luciano@if.sc.usp.br

Dietrich Stauffer
Institute for Theoretical Physics
Cologne University
D-50923 Köln, Euroland
stauffer@thp.uni-koeln.de

February 2, 2008

Abstract

The potential for associative recall of diluted neuronal networks is investigated with respect to several biologically relevant configurations, more specifically the position of the cells along the input space and the spatial distribution of their connections. First we put the asymmetric Hopfield model onto a scale-free Barabási-Albert network. Then, a geometrical diluted architecture, which maps from $L$-bit input patterns into $N$-neurons networks, with $R = N/L < 1$ (we adopt $R=0.1, 0.2$ and $0.3$), is considered. The distribution of the connections between cells along the one-dimensional input space follows a normal distribution centered at each cell, in the sense that cells that are closer to each other have increased probability to interconnect. The models also explicitly consider the placement of the neuronal cells along the input space in such a way that denser regions of that space tend to become denser, therefore implementing a special case of the Barabási-Albert connecting scheme. The obtained results indicate that, for the case of the considered stimuli and noise, the network performance increases with the spatial uniformity of cell distribution.

1 Introduction

A number of mathematical concepts and tools have been extensively applied in order to construct models and simulations of neuronal behavior that could shine some light into the intricate workings of the brain. In physics, particular importance and interest have been placed on Hopfield
models because of their potential for associative recall and close relationship with statistical mechanics [1]. However, while most such approaches have considered fully connected structures, experimental evidences indicate that biological neuronal networks involve a combination of intense local interconnections (responsible, among other things, for lateral inhibition [2]) coexisting with long range mappings which often exhibit topographical structure, in the sense that the connections preserve the spatial adjacency of the signal. Studies addressing partially connected Hopfield networks on lattices have appeared sporadically in the literature [3]. More recently [4], the Barabási-Albert [5] connecting scheme, where the “rich gets richer”, was used to define Hopfield architectures with symmetric couplings whose potential for associative recall was assessed with respect to the number of connections between the constituent cells. The obtained results indicated that, despite the relatively sparse and heterogeneous connections, for suitable parameters such architectures preserved a good deal of the associative recall when compared to fully connected counterparts, bridging the gap to further investigations.

The present work reports the extension of the models described in [6, 4] in three aspects that are specially relevant from the biological point of view, namely the asymmetry that neuron \( i \) may influence neuron \( k \) even if neuron \( k \) has no influence on neuron \( i \), the spatial distribution of cells along the input space (assumed to be a one-dimensional vector), and the spatial distribution of the connections between those cells. More specifically, as the network is constructed, each added cell is placed preferentially at regions of the input space that are denser in cells, in such a way that dense regions tend to become denser. Being diluted, not every bit of the input pattern will be connected to a neuron. Also, the connections between a cell \( i \) and the rest of the network are established in such a way that cells that are closer to each other have higher probability to interconnect, which is achieved through Monte Carlo simulation assuming normal weighting centered at cell \( i \). The potential of the networks for associative recall is experimentally assessed with respect to several parameter configurations, with special attention given to the effect of the uniformity of the cell spatial distribution over the obtained performance.

The article starts by revising some key aspects of biological neuronal structures and proceeds by presenting the obtained results and respective discussion, with special attention placed on the biological interpretation and implication of the observed behavior in terms of the parameter configurations.

2 Spatial Organization in Biological and Computational Networks

First, in what we call the asymmetric Hopfield-Barabási-Albert model, we put the Hopfield model onto the scale-free Barabási-Albert [5] network. In contrast to spin glasses and many physics systems, the interactions between biological neurons are not symmetric: Neuron \( k \) may influence neuron \( i \) even if neuron \( i \) has no influence on neuron \( k \). In other words, the synaptic strengths \( J(i, k) \) are not symmetric, i.e. \( J(i, k) \) is not equal to \( J(k, i) \). We thus construct our model in such a way that every neuron \( i \) selects itself plus exactly \( m \) other neurons (repeated connections are allowed) as neighbours. This fixed number \( m + 1 \) of influencing neighbours greatly simplifies the algorithm.

Second, in what we call the geometrical model, we try to incorporate the geometry instead of only the topology of neural nets. Another characteristic feature exhibited by the mammalian cortex is the spatial structuring permeating both the input signal and neuronal architectures. For instance, the visual field is mapped onto the retina space, which innervates, through the lateral
geniculate nucleus, into the primary visual cortex in such a way that the spatial adjacency of the input patterns is maintained. The importance of such topographical mappings is corroborated by the fact that they are found ubiquitously along the cortex, and not only in its primary sensory regions. It should be observed that such mappings are not isometric. One important neuronal phenomenon closely related to topographical mappings is known as lateral inhibition, where the activity at a given cell is inhibited by its spatial neighbours along the input space. In spite of its key role in cortical organization, relatively few neuronal networks have explicitly taken spatial organization into account, Kohonen’s SOM model [7] being one of the pioneering exceptions. At the same time, much of the efforts devoted to Hopfield models have systematically overlooked the spatial structure of input and topographical mappings. The situation is similar in the biological community, where only more recently (e.g. [3]) attention was focused on this important organizational principle. The fact that neuronal cells that are closer together tend to have increased chance to interconnect (e.g. [2]) has also been often overlooked. Indeed, cortical organization seems to combine local, intensive connections with long distance, sparser, mappings between distinct neuronal modules. Such local/global organization may also underly other spatial scales structures in the brain, possibly leading to scale-free characteristics over limited spatial scale intervals. In the current work, we propose a novel variation of the Hopfield model for associative recall that explicitly takes into account the spatial organization of the neuronal cells and the localized and global spatial distribution of their connections.

The input space is assumed to be the one-dimensional vector $I(i), i = 1, 2, \ldots, L$, to which a total of $N$ neuronal cells are attached. The number of connections made by each cell is fixed and equal to $m$. The network is diluted, in the sense that $R = N/L < 1$. Figure 1 illustrates a typical realization of the assumed neuronal architecture for $N = 7$, $m = 3$ and $L = 15$, with the respective asymmetric weight matrix shown in Equation (1). Each line $i$ shows the connections of neuron $i$ to other neurons by a ’1’. The construction of the network involves the two following steps: (a) neurons are placed at spatial positions $p \in 0, 1, \ldots, L$ according to a probability density function $h(p)$ (cells can only be added at empty sites); and (b) $m$ not necessarily distinct synaptical connections are established between the just added cell and its neighbours according to a normal (Gaussian) density function $g_\sigma(d)$, where $d$ is the distance between the position $p$ of the reference cell and each of the other cells in the network and $\sigma$ is the standard deviation of the normal distribution. It is assumed throughout this article that $L = 1000$.

![Figure 1: Example of the networks considered in this article assuming $N = 7$, $m = 3$ and $L = 15$. The black squares stand for the position of the neurons.](image)
are calculated taking into account only the neurons attached to the input pattern.

50 roughly a symmetric case. The distribution of the number of neurons, influencing
the input space smaller or equal to 1.

Fig. 4 shows that the scaling law:

\[ \Psi = f(m/P) \]

is presented to the network with ten percent of the sites flipped (overlap = 0.8), and after a few
iterations the dynamics comes to a fixed point for which the overlap

\[ \Psi = 0.8 \]

is determined. Fig. 3 shows

\[ \Psi = \sum_i S_i \xi_i \]

if, without loss of generality, the first pattern is supposed to be recovered.

3 Results

A non-stationary (along time) density function \( h(p) \) is obtained at each interaction during
the network construction by adding the function \( f(i) = 1 \) if \( |i - p| \leq a \) and 0 otherwise to the previous
instance of \( h(p) \), where \( p \) is the position of the most recently added cell, and renormalizing \( h(p) \).

Standard Monte Carlo sampling is then performed over the respective distribution function so as to
select the position \( p \) for a new cell. The probability function \( h(p) \) is initially set as uniform. Monte
Carlo simulation is also employed to select \( m \) cells for symmetric connections with the current
cell. The connections are performed as follows: (i) a vector \( v(i) \) is built such that \( v(i) = 1 \) at the
positions \( i \) where neurons exist and 0 otherwise; (ii) this vector is weighted according to a normal
density function centered at \( p \), the position of the current cell; and (iii) Monte Carlo is used to
select \( m \) sites of \( v \). It is observed that all cells are always self-connected. Figure 2 illustrates
typically obtained connection matrices. The reinforced diagonal structure of such matrices favors
conditioning and reflects the locality of the synapses at specific spatial scales defined by \( \sigma \).

For both the Hopfield-Barabási-Albert and the geometrical model, the neurons \( i, \ i = 1, 2, \ldots N \),
in our models are either firing \( (S_i = 1) \) or silent \( (S_i = -1) \) and are updated according to

\[ S_i \rightarrow \text{sign}(\sum_k J_{ik} S_k) \]

with synaptic strengths \( J_{ik} = \sum_{\mu} \xi_{i}^{\mu} \xi_{k}^{\mu} \) (Hebb rule) if \( i \) and \( k \) are connected. Here \( \xi_{i}^{\mu} = \pm 1, \mu = 1, 2, \ldots P \), are \( P \) random bit-strings called patterns, and one of them is supposed to be recalled by
this updating rule if it is presented to it in a perturbed fashion: Associative memory. The quality
of recall is measured by the overlap \( \Psi = \sum_i S_i \xi_i \) if, without loss of generality, the first pattern is
supposed to be recovered.

For the asymmetric Hopfield-Barabási-Albert model, \( N \) binary neurons are added to the initial
core of \( m + 1 \) neurons, and \( P \) binary patterns are stored. Initially, the first of these patterns
is presented to the network with ten percent of the sites flipped (overlap = 0.8), and after a few
iterations the dynamics comes to a fixed point for which the overlap \( \Psi \) is determined. Fig. 3 shows
as in the symmetric case that for \( P \ll m \ll N \) the desired pattern is fully restored. However,
Fig. 4 shows that the scaling law: \( \Psi = f(m/P) \) for infinite \( N \) is not fulfilled well, in contrast to
the symmetric case. The distribution of the number of neurons, influencing \( q \) sites each, follows
roughly a \( 1/q^3 \) law for small \( q \) but not for large \( q \) (not shown).

For the geometrical model, simulations were performed considering combinations of \( m = 25 \)
and 50 with \( a = 10 \) and 100. Parallel updating is adopted for Hopfield recovery and the number of
trained patterns is \( P = 0.05 N \). Relative overlaps are used, i.e. \( R = \Psi/N \). The relative overlaps
are calculated taking into account only the neurons attached to the input pattern.

Figure 5 shows the correlation integral \( C(r) \) [9], namely the number of neurons with distance
along the input space smaller or equal to \( r \) divided by \( N^2 \). Figures 5 to 7 show examples of
Figure 2: Example of connection matrices, shown as gray-color images, for $m = 25$ and $a = 10$ (a) and 100 (c), and for $m = 50$ and $a = 10$ (b) and 100 (d). Black means unconnected neurons, white means connected neurons.

typically obtained density and respective distribution functions obtained for $a = 10$ (a) and 100 (b) and considering 10 different realizations of the spatial distribution of cells for each parameter settings.

4 Discussion

For the Hopfield-Barabási-Albert model, the asymmetric version gave results similar to the symmetric version \[4\], provided self-interaction in taken into account in both cases. For the geometrical model, as indicated by Fig. 5, the considered scheme for positioning the neurons along the input space led to similar power law behavior for $a = 10$ and 100 for the considered correlation lags. At the same time, as is clear from Fig. 6, the value of $a$ strongly influences the characteristic spatial distribution of neurons, in the sense that shorter clusters of cells, indicated by the clumps in Fig. 6, are obtained for smaller values of $a$. As expected, higher values of $m$ tend to produce more widespread distribution of synaptic connections, leading to the wider dispersions around the main diagonal of the matrices in Fig. 2(b) and (d) and substantially superior potential.
for associative recall, shown in Fig. 7. This is a consequence of the fact that a larger number of connections has direct impact over the amount of memory in the network. At the same time, the network performance tends to improve as $a$ is increased (see Fig. 7). A more uniform spatial distribution of the neurons is obtained for larger values of $a$, with the limit case $a \to \infty$ tending to the uniform distribution. It is clear that, at least for the considered kind of stimuli and noise (uniform distribution of signal changes), the effect of irregular distribution of neurons along the input pattern space tends to deteriorate associative recall. The important biological implication is that the development (ontogeny) of real neuronal systems coping with uniformly distributed stimuli and noise has to incorporate mechanisms for ensuring uniform distribution of neuronal placement. In addition to topographical maps, other possible biological mechanisms that could be involved in such spatial tuning may involve controlled apoptosis (i.e. programmed neuronal cell death) and chemotatic migration modulated by density. It is important to bear in mind that different results could be obtained for distinct input stimuli and noise models. For instance, stimuli characterized by specific correlation lengths could be better processed by networks with neurons and connections presenting congruent spatial characteristics.

All in all, the present work has considered extensions of the classical Hopfield network which are characterized by enhanced biological plausibility as far as asymmetric connections and spatial distribution of cell bodies and synapses are concerned. Such models were analysed according to the shape-function paradigm, i.e. by assessing how the model parameters influenced the associative recall performance, and a series of perspectives has been opened for further investigation. A particularly interesting issue being currently addressed regards the characterization of the effect of the individual morphology of real neuronal cells over the network behavior, as well as the influence of stimuli and noise spatial features over the respective network performance.
Normalized overlap from 10 samples, \( N+m = 10000 \) neurons, \( P = 10 \) (+) and 100 (x); \( m=2 \) to 2000

Figure 4: \( \Psi/10000 \) for \( m \) neurons in the core and \( N = 10000 - m \) neurons surrounding them, for 100 and 1000 patterns. If scaling would be valid the two data sets would overlap in this plot.

Acknowledgments  Luciano da F. Costa is grateful to the Human Frontiers Science Program, FAPESP (99/12765-2) and CNPq (468413/00-6 and 301422/92-3) for financial help. D. Stauffer thanks H. Sompolinsky for suggesting asymmetric networks and GIF for travel support.

References

[1] J.J. Hopfield, Proc. Natl. Acad. Sci. USA 79, 2554 (1982).
[2] E. R. Kandel, J. H. Schwartz and T. M. Jessel, Principles of Neural Science, Appleton and Lange, 1991.
[3] K.E. Kürten, J. Physique 51, 1585 (1990); B.M. Forrest, J. Physique 50, 2003 (1989); D. Ji, B. Hu and T. Chen, Physica A 229, 147 (1996). See also: O. Shefi et al., Phys, Rev. E 66, 021905 (2002); S. Morita et al., Physica A 298, 553 (2001); J. Karbowski, Phys. Rev. Lett. 86, 3674 (2001).
[4] D. Stauffer, A. Aharony, L. da F. Costa and J. Adler, preprint cond-mat/0212601.
[5] A.L. Barabási and R. Albert, Science 286, 509 (1999); R. Albert and A.L. Barabási, Rev. Mod. Phys. 74, 47 (2002); S.N. Dorogovtsev and J.F.F. Mendes, Adv. Phys. 51, 1079 (2002).
[6] L. da F. Costa and E. T. M. Manoel, Neuroinformatics 1, 66 (2002).
[7] T. Kohonen, Self-Organizing Maps, Springer-Verlag (2001).
[8] L. da F. Costa, E. T. M. Manoel, F. Faucereau, J. Chelly, J. van Pelt and G. Ramakers, Network 13, 283 (2002); G. Ascoli, Computational Neuroanatomy: Principles and Methods, Humana Press (2002).
[9] M. Schroeder, Fractal, Chaos, Poser Laws, W. H. Freeman (1991).
Figure 5: The loglog graph of the correlation integral $C(r)$ obtained for $a = 10$ (a) and $100$ (b).

Figure 6: Typically obtained density (up) and respective distribution functions (down) obtained for $a = 10$ and $100$ (b).

Figure 7: Relative overlaps for $m = 25$ and $a = 10$ and $100$ (upper pair of curves) and $m = 50$ and $a = 10$ and $100$ (lower pair).