Acedo Rodríguez, L.; Moraño Fernández, JA. (2013). Brain oscillations in a random neural network. Mathematical and Computer Modelling. 57(7-8):1768-1772. doi:10.1016/j.mcm.2011.11.028.

Brain oscillations in a random neural network

Article *in* Mathematical and Computer Modelling · April 2013

DOI: 10.1016/j.mcm.2011.11.028

2 authors:

Luis Acedo
Universitat Politècnica de València
82 PUBLICATIONS **925** CITATIONS
SEE PROFILE

José Antonio Moraño
Universitat Politècnica de València
52 PUBLICATIONS **125** CITATIONS
SEE PROFILE

Brain Oscillations in a Random Neural Network

L. Acedo*, J.-A. Moraño
Instituto de Matemática Multidisciplinar,
Universitat Politècnica de València
Building 8G, 2º Floor, 46022 Valencia, Spain

Abstract

It is well-known that rhythmic patterns of neural activity appear both in the normal and abnormal function of the brain. Apart from the standard bands of electric oscillations found in the electroencephalogram (EEG): from alpha (8-12 Hz) to delta waves (1-4 Hz), synchronized firing of neural populations characterize some complex cognitive functions such as memory, attention and consciousness. In the case of electrocardiograms (ECG) it is usually recognized that oscillations can be understood as the limit cycle of an underlying non-linear process in heart dynamics. However, the situation is not so clear for EEG and the origin and purpose of neural oscillations are still the subject of a heated debate.

Our model is a version of the standard SIRS model from epidemiology in which susceptible, infected and recovered sites represent quiescent, firing and refractory neurons, respectively. Here we show that, in a SIRS random network epidemic model for neural activity, self-sustained oscillations appear in a restricted parameter region of the transition probabilities. This could explain the role of synchronized oscillations as a discriminant process for internal or external stimuli in brain dynamics.

Keywords: Neural oscillations, Stochastic neural models, Random networks

*e-mail: luiacrod@imm.upv.es

1 Introduction

The history of electroencephalography (EEG) begins with Hans Berger who discovered in 1929 that between electrodes attached to the human scalp a potential difference in the mV range could be detected by means of a precision galvanometer [1]. He observed that a rhythmic pattern with a frequency of 8-12 Hz was recorded from subjects with their eyes closed (Alpha rhythm or Berger's wave). After opening their eyes the frequency increased to 12-30 Hz (Beta rhythm). Alpha power was larger with eyes closed than with eyes open, and it was associated with a relaxed brain.

In his work, Berger was inspired by the findings of the surgeon Richard Caton who in 1875 measured electrical potentials on the cortex of laboratory animals. The discovery of intracranial measures of electrical activity preceded by half a century the epoch-making Berger's discovery. The importance of Berger's method is that, being an extracraneal and non-invasive technique, could develop into a very useful monitoring and diagnosing tool for neurologists and psychiatrists.

EEG discovery was ignored for almost a decade. Most researchers thought that these small currents were artifacts of the experimental apparatus and the human body. The work of Berger was eventually recognized and EEG developed into a field with vast applications in Neurological Diagnosis, Experimental Psychology and Psychiatry [2]. In 1937, A. Lee Loomis and his collaborators classified the different stages of sleep in relation with the EEG signals [3]. At that time, it was clear that during sleep the EEG pattern suffered changes that could be recognized by counting the number of zero crossings (as a qualitative measure of frequency) and its amplitude. These transitions are:

- Stage 1: As observed by Berger during awareness the brain produces the alpha wave with a mixture of frequencies in the range 8-12 Hz. This is a fast rhythm with low amplitude. When the individual becomes drowsy the pattern changes to the theta wave with lower frequencies in the range 4-7 Hz.

- Stage 2: In this stage the basal EEG is sporadically interrupted by K-complexes (high-voltage peaks around 100 μV occurring with a periodicity of 1.0-1.7 minutes) and the so-called sleep spindles or sigma waves with a frequency 13-17 Hz appearing every 0.5-1.5 seconds.

- Stage 3: This is the most deep stage of sleep. It is characterized by delta waves ranging from 0.5 to 2 Hz and an amplitude from the negative to the positive peak around 75 μV. The small frequency of these waves has suggested the name "slow-wave" sleep for this stage.

Frequencies of EEG patterns are commonly associated with a subcortical pacemaker located at the thalamus. This has been critized recently as a fallacy in EEG research [4]. An explanation of EEG must resort to models of brain dynamics.

Mathematical models of the brain as a set of units with connections that determine their evolution has been already proposed. The so-called cellular automata have been studied as a model of cortical physiology in a rough model by Hoffmann [5] and also as a way to disclose spatio-temporal patterns of activity in the hippocampal network [6]. Models of neural populations motivated by the confluence of the theory of cellular automata defined upon networks have developed as a field onto itself known as Neuropercolation [7].

Recently, one of us proposed a stochastic cellular automata model defined upon a complete graph as a simple model of brain structure and studied the fluctuations in global activity in comparison with EEG [8, 9]. The complete graph is, apparently, a very simple structure for a brain but a complete set of connections between compartments in the brain has also been proposed as a reasonable model predicting some invariances observed between different species [10].

Nevertheless, the number of synapses that a typical neuron in the human cortex projects towards their neighbours is on the range of 7000–12000 [11]. Consequently, a more realistic cellular automata model of the brain should be defined upon a more sparse network: random network [12] or a Watts–Strogatz network [13]. The computational effort to simulate a cellular automata model in a significatively large system incorporating, at least, one million nodes is vast and will require the implementation of a distributed computing solution.

In this paper, we follow this natural path towards complexity on mathematical models of the brain by defining a stochastic cellular automata model on a random network. The states of the sites in the network should correspond to the basic behavior of neurons: resting neurons in which the interior of the cell have a negative potential (about -70 mVolts) which respect to the exterior of their membrane, firing neurons in which a visual, auditory or mechanical stimulus (sensory neurons) or the opening of ligand-gated sodium

channels by the action of neurotransmitters induces the depolarization of the interior of the cell and the development of an action potential that propagates along the axons, and refractory neurons which remain inactive after repolarizations for a short time of several milliseconds. These states are parallel to the classic susceptible-infected-recovered-susceptible (SIRS) model of mathematical epidemiology. Following this line of thought we will propose a SIRS model upon a random network as an improved cellular automata model for brain dynamics.

Taking into account the complexity of these networks and that a number $N = 10^6$ nodes was considered, it is obvious that a single computer approach to this problem is out of the question. On the other hand, we have developed a distributed computing solution by means of a client-server TCP program that allows the server to send tasks and retrieve results. This first solution was initially used in the University Intranets. Later on, the standard BOINC protocol for distributed computing throughout the World Wide Web was also applied. A previous implementation of this system for the propagation of the Respiratory Syncytial Virus epidemic has already been studied [14].

The computing power of the distributed solutions allowed us to explore the parameter space of the random network for more than 20 years of computing time in a single computer. In a confined region of the parameter space we have obtained endogenous oscillations in the activity of the network. Brain oscillations have been observed in the human brain and it is thought that they are associated with memory, attention and even consciousness [15]. But also in the mini-brains of insects these oscillations have been studied in connection with odour-encoding in locusts [16], bees [17] and Drosophila flies [18].

Regular behavior of physiological signals is usually mathematically explained within the paradigm of limit cycles and attractors. For example, Clifford and McSharry have recently proposed a model to generate electrocardiogram, blood pressure and respiration signals based upon a system of three non-linear differential equations [19]. However, the role of brain oscillations as an encoding internal language of brain requires a very different origin.

The paper is organized as follows: The random neural network model is defined in Section 2. In Section 3 we discuss the different behavior obtained with a network of $N = 10^6$ nodes and a degree of connectivity that ranges from $k = 10$ to $k = 500$. In Section 4 the results are discussed and some remarks for future work are given.

2 The Random Neural Network Model

The emergent science of networks provides several standard alternatives to implement the network substrate. The most traditional is based upon the pioneer work of Erdös and Rényi [12], the so-called random graphs where connections among the pairs of subjects are created with the same probability. Alternative models are the scale-free networks [20] or the small-world networks of Watts and Strogatz [13]. The small-world phenomenon, i. e., every pair of nodes are connected through a path which crosses a small amount of neighbours, is found in many social networks linked by friendship, collaboration or other social links.

The cytoarchitecture of the human cortex is charaterized, in general, by stratified layers of neurons. This basic structure is already present at birth but dendritic arbors develop and grow during the first two years. It is known that the details of this fine substructure develops throughout many years [21, 22]. In our approach, we are more interested in the topological properties of these networks and we should ignore the stratified architecture. We should choose the Erdös-Rényi random network model charaterized by a Poisson distribution of contacts among nodes with a mean value k.

Random networks are characterized by the number of sites or nodes ($N = 10^6$ in our simulations) and the average number of contacts of every individual, k (called the degree of this node). Consequently, the number of links in the network is given by $Nk/2$. These links are randomly assigned to pairs of nodes with the obvious rule that, at most, only a link can connect two nodes.

By following this algorithm we have checked that a Poisson distribution for the degree of the nodes (with $N = 10^6$ and $k = 10$) is obtained as shown in Figure 1.

Once the random network is generated we apply an evolution algorithm in order to analyze the number of active neurons as a function of time. As the initial state we consider only a small fraction of firing neurons. All quiescent neurons are then checked iteratively and they start to fire at time step $t + 1$ with probability α for every contact with a firing neuron at time t. The average time a neuron remains in the firing state is $1/\nu$ whereas the average refractory time is $1/\gamma$. These firing and refractory times are intrinsic to the neuron structure and we assume that they follow a Poisson distribution with the aforementioned averages $1/\nu$ and $1/\gamma$.

In our stochastic model we are simulating the transmission of action po-

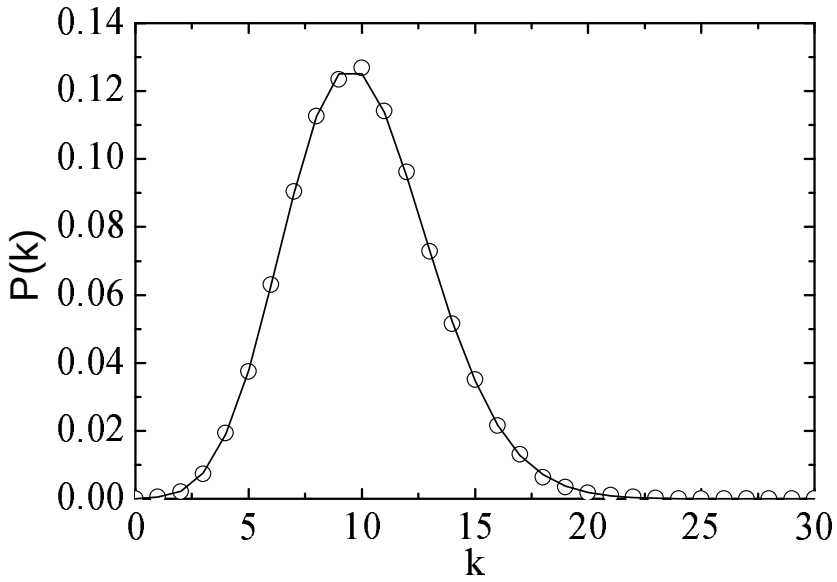

Figure 1: Fraction of nodes with degree k, $P(k)$, vs the degree k in a network with $N = 10^6$ nodes and average value $\langle k \rangle = 10$. The solid line is the exact Poisson distribution and the circles are the simulation results. An average over 50 realizations of the network building was performed.

tentials from neuron to neuron as a stochastic process. In practice, neurons integrate the signals received from the axon projections of their neighbors, this is the basic idea of the so-called integrate-and-fire models. If a certain threshold is reached the quiescent neuron start to fire. In our model this collective effect is captured by the independent stochastic interations of a quiescent neuron with their firing networks in its neighborhood. If we have $F(t)$ firing neurons in contact with a given quiescent neuron at time t the probability for this neuron to start firing at $t+1$ is $F(t)\alpha$ and, consequently, increase linearly with the number of firing neurons in their topological vicinity.

It is difficult to ascribe a definite value to α according to our present understanding of brain physiology. However, some recent physiological studies have revealed that the levels of glutamic acid, the main excitatory neurotransmitter in the cerebral cortex, increase after sleep deprivation in rats [23, 24, 25, 26]. The measurements of cerebral glucose utilization by means of the positron emission tomography technique also reveal a decline in several areas of the brain during sleep [27]. These observations are consistent with a scenario in which the homeostatic equilibrium of neurotransmitters is different between sleep states and the awaken state and could be mimicked by adjusting the values of α.

In the case of external stimuli that promote the opening of sodium channels in sensory neurons the role of α is clearer as a probability that measures the increase of firing probability of the stimulated neurons. Furthermore, the mean firing time, $1/\nu$, and the mean refractory time, $1/\gamma$, can, in principle, be obtained from the physiology of a single neuron. Values of the refractory period around 0.8-1.1 ms were reported early in the literature [28]. The firing phase should be comprised between the polarization and depolarization stages where the spike train is generated and should be much shorter.

In our study we should consider the values $1/\nu = 10$ and $1/\gamma = 200$. Similar results are obtained for other proportions between ν and γ. In the next Section we will analyze the behavior found with special interest in the periodic oscillations of the brain activity.

3 Neural Oscillations in the Random Network

We have obtained a phase diagram of behavior in the α–k plane we have tested 60,000 combinations of k (in the range 10-500) and the transmission probability, α, of firing activity through the axons ($0 \leq \alpha \leq 0.005$ with 0.00001 jumps). The values of the firing and refractory periods were taken constant: $1/\nu = 10$ and $1/\gamma = 200$. The number of neurons is $N = 10^6$. Although this value is small even for a small region of the human brain it is larger than the average number of neurons in the honey-bee ($N \simeq$960,000) which is the insect with the largest brain in relative size [29]. In the brain of the insects the number of axonal projections per neuron is also smaller [30] and a value of k around 500 should be reasonable.

The upper bound of our range $k = 124$ exceeds the capacity of most computers and can only be simulated in the present-day standard 4GB RAM computers. For this reason the case $k = 500$ was analyzed in a dedicated 12GB server.

The results are as follows: In the larger part of the explored α–k region the system settles in a stationary state with very low or null activity. This occurs after a transient of high activity (when the α probability is set to excessively large values) or a fast decay for low values of α. For large values of α almost all neurons become excited and, afterwards, they return to the refractory state. Therefore, during the refractory time there are neither active neurons nor quiescent neurons and the activity fades out rapidly. In a small region of the α–k plane we find damped oscillatory or regular oscillatory behavior. This region is confined within the two curves shown in Fig. 2 These curves can be fitted numerically in term of exponential series as follows:

$$k_{\rm up}(\alpha) = 22.56 + 626.40e^{-\alpha/0.00036} + 180.64e^{-\alpha/0.00174} \quad (1)$$

$$k_{\rm down}(\alpha) = -2.54 + 740.27e^{-\alpha/0.00041} + 100.46e^{-\alpha/0.00387}\,, \quad (2)$$

and the oscillations (damped or regular) are find only in the interval $k_{\rm down}(\alpha) \leq k \leq k_{\rm up}(\alpha)$. In Fig. 3 we have plotted the oscillations found for $k = 500$ and two close values of the excitation probability: $\alpha = 2.6 \times 10^{-4}$ and $\alpha = 2.9 \times 10^{-4}$. We notice that the amplitude is different despite the variation of α is only 3×10^{-5}. Consequently, we have deduced that the neural random network with high connectivity is a very sensitive device to discriminate between different values of the excitation probability.

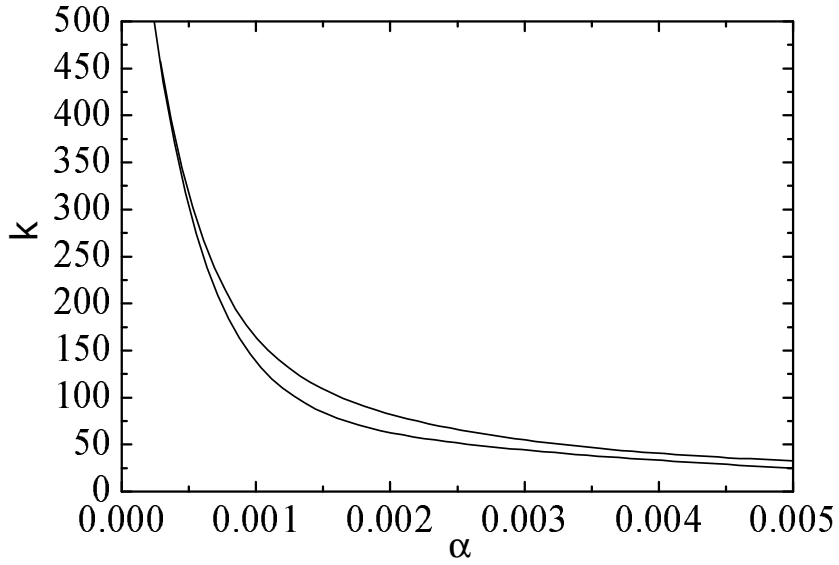

Figure 2: Upper and lower curves delimiting the region where regular oscillatory or damped oscillatory behavior of brain activiry is found. The number of neurons is $N = 10^6$. Firing and refractory times were $1/\nu = 10$ and $1/\gamma = 200$, respectively.

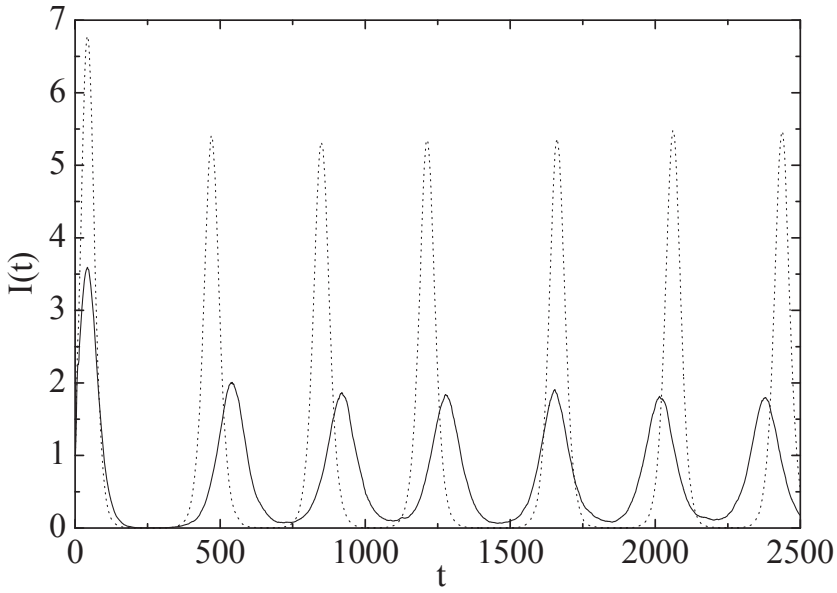

Figure 3: Number of firing neurons in the random network, $I(t)$, measured in units of 10,000 for an average degree of each node $k = 500$ and $\alpha = 2.6 \times 10^{-4}$ (solid line), $\alpha = 2.9 \times 10^{-4}$ (dotted line). Oscillatory behaviour is observed in these two cases.

In experiments with insects it has been found that intrinsic oscillations around 20 Hz are induced by different odours. These oscillations are generated as a global pattern involving feedback with the mushroom bodies structures of the insect protocerebrum and they are not observed as local field potentials recordings in the antennal lobes [16]. These oscillations have also been observed in bees and they are regarded as the encoding in the neural assembly of the odour stimulus. These oscillations are different for very similar odours (1-hexanol and 1-octanol), although the discriminant capacity is impaired by the action of antagonists of the γ-aminobutyric acid receptors such as picrotoxin [17]. In humans neural oscillations are related with cognitive processes: memory encoding (theta wave), attention (alpha and gamma) and conscious awareness of meaningful visual patterns (synchronous gamma oscillations through separate brain areas).

4 Conclusions and Final Remarks

In this paper we propose a random network model for collective synchronous behavior in the brain. We find that in a certain window of connectivity, k, and excitation probability, α, regular endogenous oscillations appear. The interesting fact is that in the past two decades increasing evidence has been gathering of the presence of these oscillations even on simple insect protocerebrums [17, 16, 18]. In these cases, oscillations are mainly elicited by odour stimulation. Insects use these oscillations as an internal language that encodes different odour stimuli.

These neural oscillations are also present in humans but, according to a different evolutionary history, they are mainly related to memory, attention and conscious awareness [15].

We have shown that oscillations are an intrinsic feature of networks with a high number of nodes and high connectivity. We have been able to simulate networks with $N = 10^6$ nodes and an average degree of connectivity up to k =500. In order to speed up the calculations a distributed computing solution has been developed under the basis of the BOINC (Berkeley Open Infrastructure for Network Computing) infrastructure [31]. This way we achieved more than 21 years of computing time in only three weeks.

We have shown that oscillations exhibit an extreme sensitivity in the α–k diagram. This sensitivity increases with the average degree of the random network. This fine tuning could explain the role of neural oscillations as a

code to represent some stimuli and distinguish among them.

In the future we plan to study larger networks with a higher degree of connectivity in order to approach the topological architecture of more complex brains. Moreover, stratified layer of neurons could also be considered because large portions of the cortex are organized this way.

Acknowledgements

Supported by grant from the Universidad Politécnica de Valencia PAID-06-09 ref: 2588 and FIS Research Grant PI10/01433 from the Instituto de Salud Carlos III.

References

[1] H. Berger, Über das Elektroenkephalogramm des Menschen, Archiv für Psychiatrie und Nervenkrankheiten, 87 (1929) 527–570.

[2] S. Tong and N. V. Thakor (editors), Quantitative EEG Analysis: Methods and Clinical Applications, Artech House, Norwood, MA, 2009.

[3] A. L. Loomis, E. N. Harvey and G. A. Hobart, Cerebral states during sleep, as studied by human brain potentials, Journal of Experimental Psychology, 21(2) (1937) 127–144.

[4] P. L. Nunez and R. Srinivasan, Electric Fields of the Brain–The Neurophysics of EEG, Oxford University Press, New York, Second Edition, 2006.

[5] M. I. Hoffmann, A cellular automaton model based on cortical physiology, Complex Systems, 1 (1987) 187–202.

[6] M. Tatsuno, Y. Nagai and W. Aizawa, Rule–dynamical approach to hippocampal network, Neurocomputing, 38–40 (2001) 965–971.

[7] R. Kozma, Neuropercolation, Scholarpedia, 2(8) (2007) 1360.

[8] L. Acedo, A second-order phase transition in the complete graph stochastic epidemic model, Physica A, 370 (2006) 613–624.

[9] L. Acedo, A cellular automaton model for collective neural dynamics, Mathematical and Computer Modelling, 50 (2009) 717-725.

[10] V. Braitenberg, Brain size and number of neurons: An exercise in synthetic neuroanatomy, Journal of Computational Neuroscience, 10 (2001) 71–77.

[11] M. Abeles, Corticonics: Neural Circuits of the Cerebral Cortex, Cambridge University Press, Cambridge, 1991.

[12] B. Bollobás, Random Graphs, Cambridge University Press, Second Edition, Cambridge, 2001.

[13] D. J. Watts, Small Worlds: The dynamics of networks between order and randomness, Princeton University Press, 1999.

[14] L. Acedo, J. A. Moraño, R. J. Villanueva, J. Villanueva-Oller and J. Díez-Domingo, Using random networks to study the dynamics of respiratory syncytial virus (RSV) in the Spanish region of Valencia, Mathematical and Computer Modelling, 54 (2011) 1650–1654.

[15] L. M. Ward, Synchronous neural oscillations and cognitive processes, TRENDS in Cognitive Sciences 7(12) (2003) 553–559.

[16] G. Laurent and M. Naraghi, Odorant-induced oscillations in the mushroom bodies of the locust, The Journal of Neuroscience 14(5) (1994) 2993-3004.

[17] M. Stopfer, S. Bhagavan, B. H. Smith and G. Lauren, Impaired odour discrimination on desynchronization of odour-encoding neural assemblies, Nature 390 (1997) 70–74.

[18] L. L. Prieto-Godino and G. G. de la Polavieja, Brain Activity at 70 80 Hz Changes during Olfactory Stimulation Protocols in Drosophila, PLoS ONE 5(9) (2010) e12867.

[19] P. E. McSharry, G. D. Clifford, L. Tarassenko and L. Smith, Dynamical model for generating synthetic electrocardiogram signals, IEEE Trans. BioMed. Eng. 50(3) (2003) 289–294.

[20] R. Albert and A. L. Barabási, Emergence of scaling in random networks, Science 286 (1999) 509–512.

[21] J. M. Fuster, The Prefontal Cortex, Elsevier Academic Press, Amsterdam, Fourth Edition, 2008.

[22] M. Marín-Padilla, The Human Brain: Prenatal Development and Structure, Springer-Verlag, Berlin, 2011.

[23] C. M. MCDermott, M. N. Hardy, N. G. Bazan, and J. C. Magee, Sleep deprivation-induced alterations in excitatory synaptic transmission in the CA1 region of the rat hippocampus, J. Physiol. 570(3) (2006) 553–565.

[24] S. Majumdar S and B. N. Mallick, Increased levels of tyrosine hydroxylase and glutamic acid decarboxylase in locus coeruleus neurons after rapid eye movement sleep deprivation in rats, Neurosci. Lett. 338(3) (2003) 193–196.

[25] H. Murck, T. Struttmann, M. Czisch, T. Wetter, A. Steiger, and D. P. Auer, Increase in amino acids in the pons after sleep deprivation: a pilot study using proton magnetic resonance spectroscopy, Neuropsychobiology 45(3) (2002) 120–123.

[26] L. Bettendorff, M. Sallanon-Moulin, M. Touret, P. Wins, I. Margineanu, and E. Schoffeniels, Paradoxical sleep deprivation increases the content of glutamate and glutamine in rat cerebral cortex, Sleep 19(1) (1996) 65–71.

[27] P. Maquet, D. Dive, E. Salmon, B. Sadzot, G. Franco, R. Poirrier and G. Franck, Cerebal glucose utilization during sleep-wake cycle in man determined by positron emission tomography and [18F]-2-fluoro-2 deoxy-D-glucose method, Brain Research 513 (1990) 136–143.

[28] E. T. Rolls, Absolute refractory period of neurons involved in MFB self-stimulation, Physiology & Behavior 7(3) (1971) 311–315.

[29] R. Menzel and M. Giurfa, Cognitive architecture of a mini–brain: the honeybee, TRENDS in Cognitive Sciences 5(2) (2001) 62–71.

[30] S. E. FahrBach, Structure of the MushRoom Bodies of the Insect Brain, Annu. Rev. Entomol. 51 (2006) 209–232.

[31] Berkeley Open Infrastructure for Network Computing, http://boinc.berkeley.edu