

WORKSHOP ON DYNAMICAL PROCESSES ON COMPLEX NETWORKS



May 13 – 17, 2024

at Instituto de Física Teórica - UNESP, Brazil

CONFIRMED SPEAKERS

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Complex systems are characterized by a large number of units, such as particles, individuals or neurons, that interact typically with a few neighbors but lead to the emergence of large-scale collective behavior. Networks provide a natural representation of these systems, where nodes play the role of the units, and links between nodes indicate pairwise interactions. The distribution of links among the nodes is a key property of networks, defining how the units of the system interact. Links may follow simple rules, such as regular lattices or random connections, or may be highly heterogeneous, displaying power law distributions. More recently, the concepts of multilayer and higher-order networks have emerged to describe interconnected sets of networks and many-body interactions, where single-layer networks are generalized to simplicial complexes or hypergraphs.

Two of these processes have become particularly important and will be the focus of this workshop in terms of applications. The first is the spreading of infectious diseases and the dissemination of information. As disease and information propagation depend critically on the network of contacts between people, understanding how the topology of these networks affects the spreading and how that, in turn, modifies the network via quarantine, vaccination, use of masks, or death, has become a major topic of research. A second topic to be discussed in the workshop is the synchronization of coupled oscillators. Understanding how independent oscillators synchronize their motion when coupled together has become an important area of research, both in terms of applications to neuronal dynamics and swarms, and to basic science, characterizing its phase transitions, hysteresis, and dynamical properties.

There is no registration fee and limited funds are available for travel and local expenses.

Registration deadline:
March 24, 2024

Online registration and more information:
<https://www.ictp-saifr.org/wdpcn2024/>



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Plasticity: An incredible brain capacity

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São Paulo
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Brain

The central organ of the human nervous system.

It controls most of the activities of the body, processing, integrating, and coordinating the information it receives from the sense organs, and making decisions as to the instructions sent to the rest of the body.



Malleable

Adapt to needs.



Stable

Do not harm the functions



Brain Plasticity

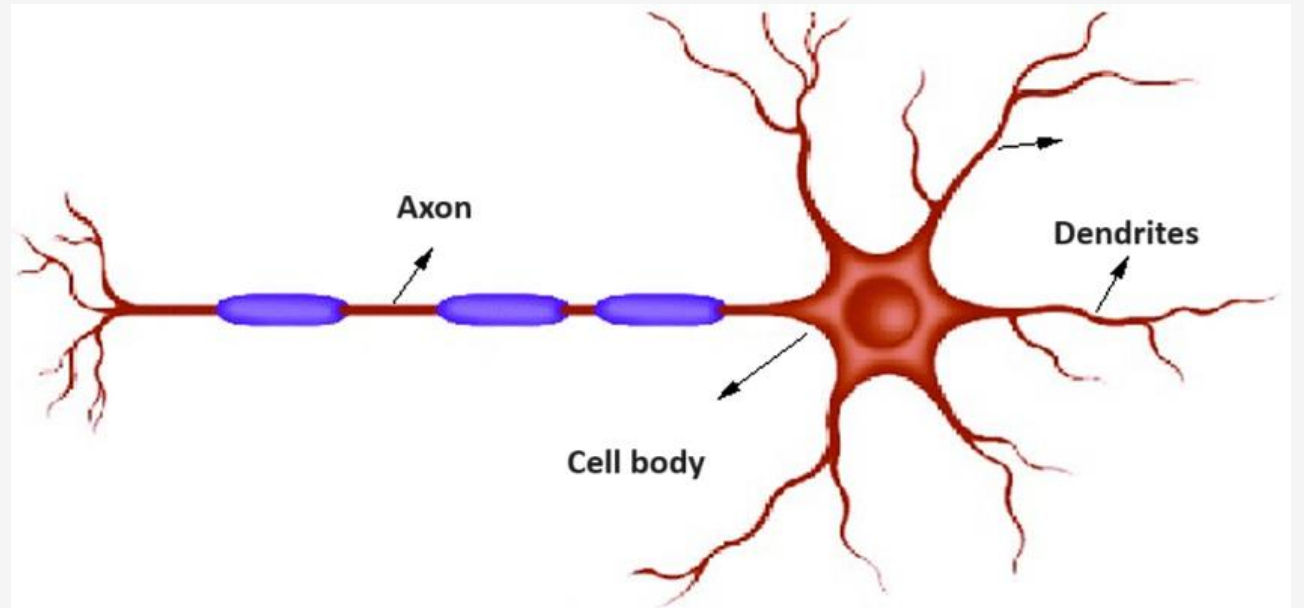


Neuroplasticity or brain plasticity is defined as the ability of the nervous system to change its activity in response to intrinsic or extrinsic stimuli by reorganizing its structure, functions, or connections. A fundamental property of neurons is their ability to modify the strength and efficacy of synaptic transmission through a diverse number of activity-dependent mechanisms, typically referred to as synaptic plasticity.

Neuron

Dendrites: receive or transmit information from/to other neuronal cells.

Axon: transmission of nerve impulses.

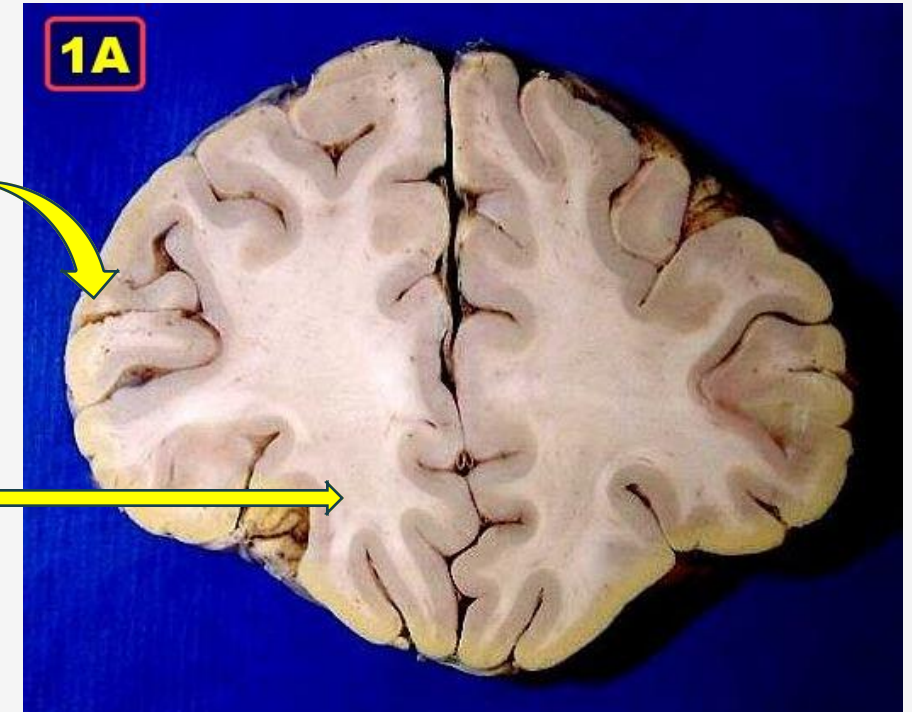
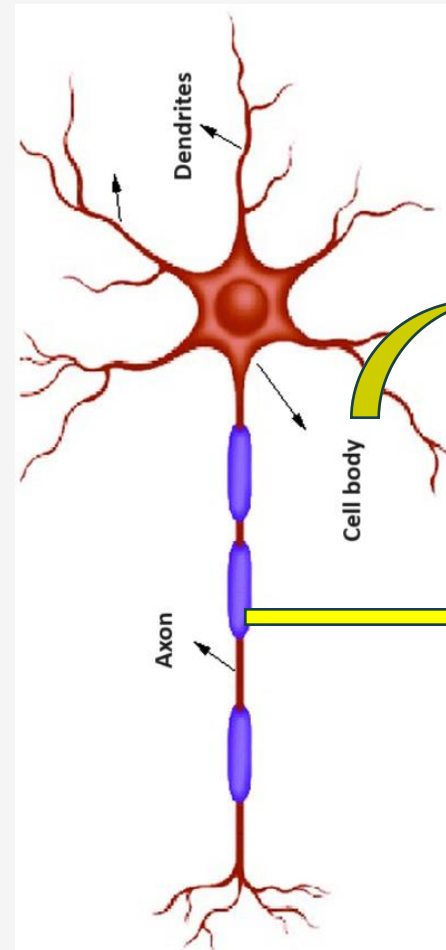


Within a nervous system, a neuron or nerve cell is an electrically excitable cell that fires electric signals called action potentials across a neural network. Neurons communicate with other cells via synapses, which are specialized connections that commonly use minute amounts of chemical neurotransmitters to pass the electric signal from the presynaptic neuron to the target cell through the synaptic gap.

Neuron

Cell Body - Cortex

Axon – white matter



<https://anatpat.unicamp.br/bineucerebrocoronalindice.html>

<https://doi.org/10.1590/S1806-11173721787>
Rev. Bras. Ens. Fis. 37
(2) • Jun 2015

Neuron

Dendrites: receive or transmit information from/to other neuronal cells.

Axon: transmission of nerve impulses.

Chemical synapses

Neurotransmitters

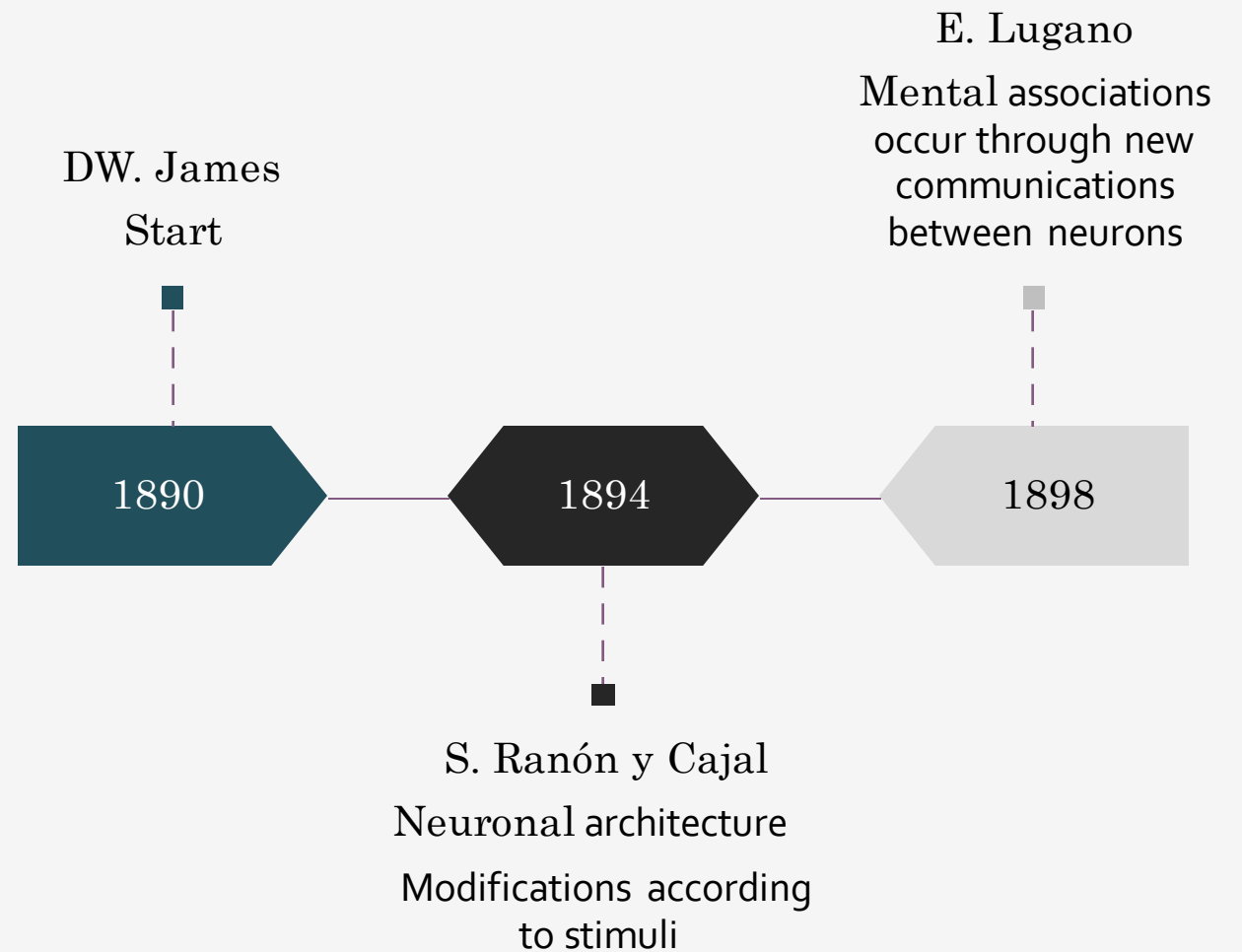
Electrical synapses

Electrical impulses and communication junctions



Time line

Plasticity



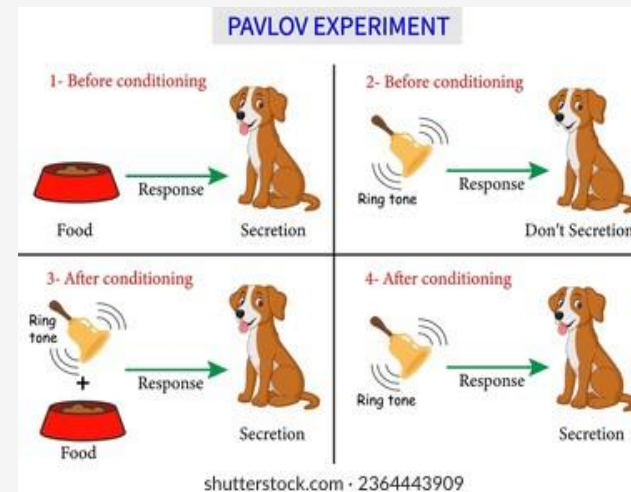
Time line

Plasticity

J. Konorski

Conditioned reflex occurs from changes in neuronal synapses.

1948



Pavlov Museum, Russia.

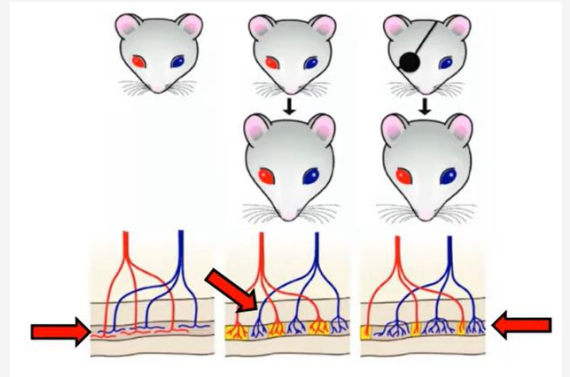
Time line

Plasticity

D. Hebb
Synapses used more frequently
are strengthened as a result of
the physiological adaptation of
the neurons involved.

1949

1983

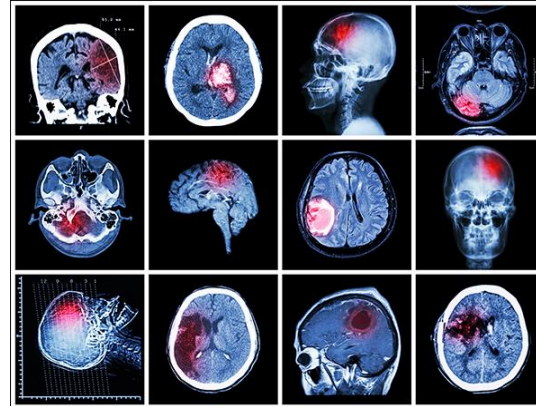


M. Merzenich
Adults plasticity

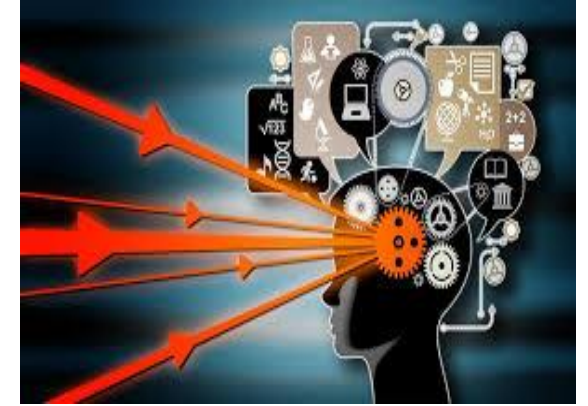


Plasticity

Brain plasticity happens all the time



Recovery from injuries and microinjuries

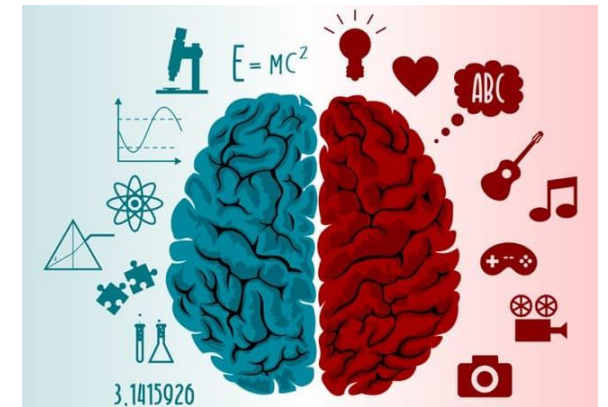


Acquisition of new skills

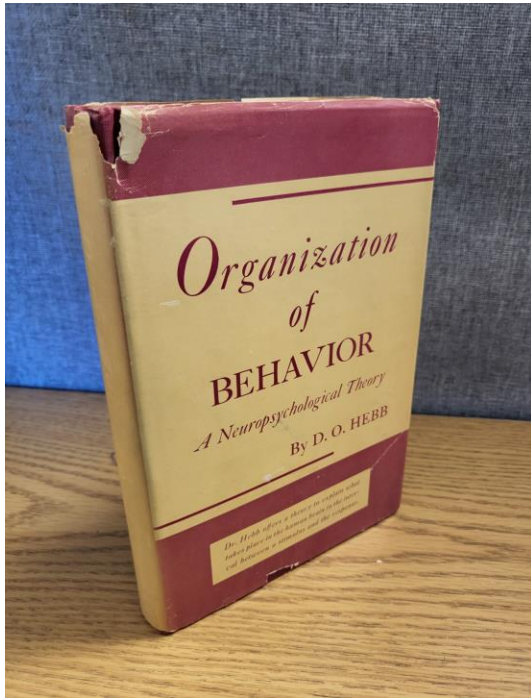


Sensory deprivation

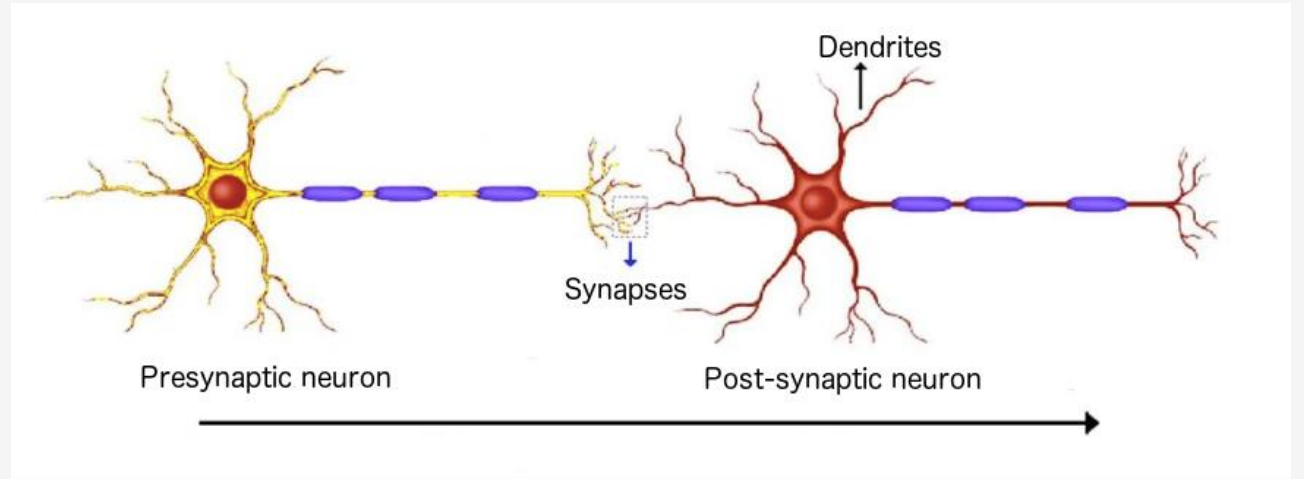
Suedfeld, P, Turner, J.W.Jr., Fine, T.H. (Eds) (1990) *Restricted Environmental Stimulation: Theoretical and Empirical Developments in Flotation REST* Springer. Springer-Verlag [ISBN 0-387-97348-6](https://doi.org/10.1007/978-3-642-81488-6)



Response to external stimuli



Hebbian Theory



Rev. Bras. Ens. Fis. 37 (2) • Jun 2015 (<https://doi.org/10.1590/S1806-11173721787>)

A NEUROPHYSIOLOGICAL POSTULATE

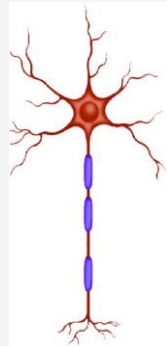
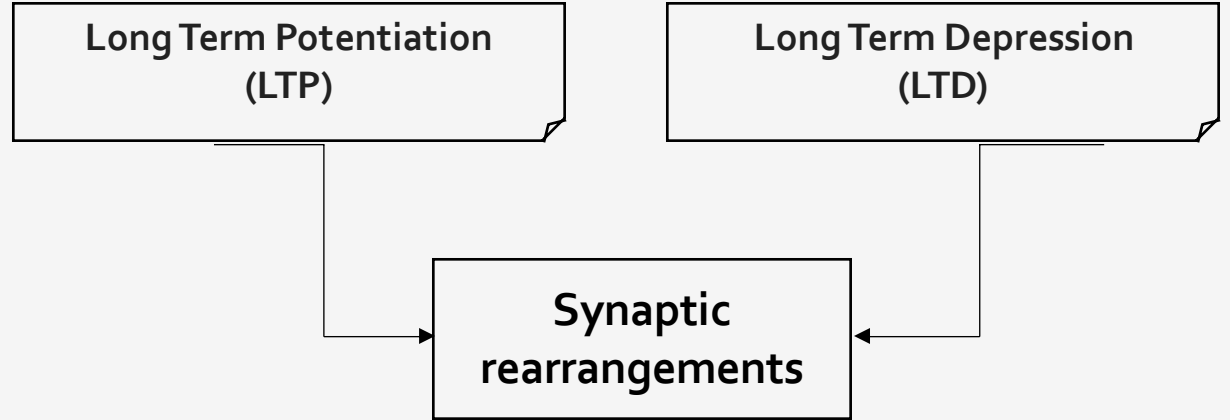
Let us assume then that the persistence or repetition of a reverberatory activity (or "trace") tends to induce lasting cellular changes that add to its stability. The assumption * can be precisely stated as follows: *When an axon of cell A is near enough to excite a cell B and repeatedly or persistently takes part in firing it, some growth process or metabolic change takes place in one or both cells such that A's efficiency, as one of the cells firing B, is increased.*

Hebb, D. O. (1949). *The organization of behavior*. New York: Wiley (pag 62)

https://pure.mpg.de/rest/items/item_2346268_3/component/file_2346267/content

Plasticity

Synapses



Receptors and neurotransmitters: number modified

Reduction of receptors - synaptic weakening

Increase in receptors - synaptic strengthening

Excitatory (eSTDP) and inhibitory synapses (iSTDP)

Structure modification

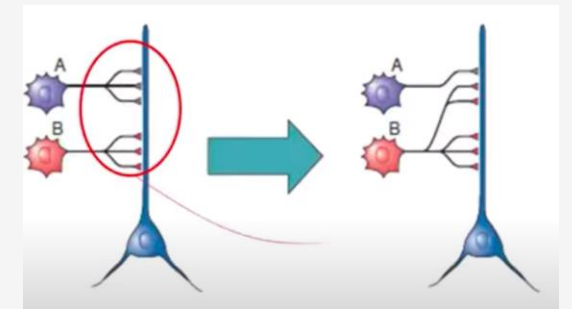
The same number of connections

Rearrangement in synapses

Synapse/dendrites sprouting

Synapse/dendrites elimination

Migration and neurogenesis



Evolutionary Brain Dynamic Behavior

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The influence of connectivity on the firing rate in a neuronal network with electrical and chemical synapses

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ARTICLE INFO	ABSTRACT
Article history: Received 21 February 2015 Received in revised form 14 March 2015 Accepted 4 June 2015 Available online 17 June 2015	Abstract We study the firing rate properties of a cellular automaton model for a neuronal network with electrical synapses. We propose a simple mechanism by which the electrical connections are isolated through electrical and chemical synapses. In the latter case, we introduce a time delay which produces self-referenced activity. Neural connections, or shunters, are randomly introduced according to a specified connection probability. There is a critical connection probability for which random firing ceases to exist. In a critical region, the corresponding networks may be treated from a self-organized criticality point of view [1]. However, if we regard the network as comprising a three-dimensional network (the brain), where they are connected by axons and dendrites, it is convenient to use a network embedded in an Euclidean space [4]. Due to this fact, it is often necessary to use models that exhibit spatiotemporal behavior.
Keywords: Neuronal networks Electrical synapses Cellular automata	© 2015 Elsevier B.V. All rights reserved.

1. Introduction

The human brain is a complex network that contains approximately 10^{11} neurons, each of them being connected with $\sim 10^4$ other neurons, on average. For example, one neuron in the vertebrate brain is connected to more than ten thousand other neurons [1]. These properties make the brain a paradigmatic example of complex networks. Moreover, each neuron is a unit consisting of about a hundred specialized modules with different functions, each of them being a complex network itself, where each module receives excitatory inputs from a few thousands of other neurons [2]. In theoretical and computational studies where the facilitation distance between neurons does not play a significant role, the corresponding networks may be treated from a self-organized criticality point of view [1]. However, if we regard the network as comprising a three-dimensional network (the brain), where they are connected by axons and dendrites, it is convenient to use a network embedded in an Euclidean space [4]. Due to this fact, it is often necessary to use models that exhibit spatiotemporal behavior.

Accordingly, neural network models are used to study biological neurons that are spatially distributed and are connected by both excitatory and inhibitory synaptic interactions, of both electrical and chemical nature [5]. For example, there are theoretical studies of the dynamics of networks with sparsely connected excitatory and inhibitory integrate-and-fire neurons [6]. It is possible to insert inhibitory connections in mathematical models through synapses by considering the dynamical units as being non-responsive during resting states until at least a many excitatory or inhibitory pulses are received [7].

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Dynamic range in a neuron network with electrical and chemical synapses

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ARTICLE INFO	ABSTRACT
Article history: Received 21 February 2015 Received in revised form 14 March 2015 Accepted 4 June 2015 Available online 17 June 2015	Abstract The dynamic range is the logarithmic difference between maximum and minimum levels of responsive produced by neural stimuli. In the human sensory system the dynamic range is typically larger than for single neurons, the amplification being essentially a collective effect of the neural network. We investigated the dynamic range obtained by a cellular automaton network with electrical and chemical synapses, when the stimuli are received by a Heaviside process of superposition and a superposed unit amplitude and the neuron response is its average firing rate.
Keywords: Dynamic range Cellular automata Neural network	© 2015 Elsevier B.V. All rights reserved.

1. Introduction

The quantitative characterization of the sensation we assign to a given stimulus was one of the key problems in Psychophysics [1]. Weber and Fechner proposed, in the 19th century, that the relationship between stimulus and response was logarithmic, rather than linear; the magnitude of the sensation related to a given stimulus depends on the magnitude of the stimulus itself by $P \sim I^{1/4}$ [2].

In the mid-1930s, Stevens has proposed a more general stimulus-response theory based on a power law $P \sim I^n$. This is an empirical mathematical statement obtained by electrophysiological experiments [3]. The exponent n has been found to be 0.67 for loudness, 0.23 to 1.0 for brightness, 0.8 to 1.4 for taste, and 0.7 to 1.6 for weights, just to give some examples [1].

Due to anatomical and physiological limitations of our senses, any quantitative relationship between stimulus and sensation must have upper and lower bounds corresponding, respectively, to the largest and the smallest values of the response, measured by some characteristic quantity. The dynamic range is the logarithm of the difference between the largest and the smallest value of this quantity, and is usually expressed in decibels (dB). For example, the human senses of sight and hearing use relatively large values of the dynamic range of 90 dB and 100 dB, respectively [4]. In terms of a general stimulus-response relation the dynamic range is the interval over which a power law (Stevens law) can be fitted.

Experimental evidence points out that the dynamic range of a single neuron is substantially smaller than the dynamic range observed at the macroscopic level [5]. As an example, in the case of mouse olfactory system, isolated receptor neurons respond to odour chemicals have a dynamic range of the order of 10 dB [6]. On the other hand, the corresponding

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Dynamic range in small-world networks of Hodgkin-Huxley neurons with chemical synapses

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ARTICLE INFO	ABSTRACT
Article history: Received 21 May 2014 Available online 1 June 2014	Abstract According to Stevens' law the relationship between stimulus and response is a power law within an interval called the dynamic range. The dynamic range of sensory organs is found to be larger than that of a single neuron, suggesting that the neural network structure plays a key role in the behavior of both the scaling exponent and the dynamic range of neuron assemblies. In order to verify computationally the relationship between stimulus and response for scaling processes, we investigated small-world networks of neurons described by the Hodgkin-Huxley equations connected by chemical synapses. We found that the dynamic range increased with the average link, suggesting that the enhancement of the dynamic range observed in sensory organs, with respect to single neurons, is an emergent property of complex network dynamics.
Keywords: Small-world networks Hodgkin-Huxley neurons Dynamic range	© 2014 Elsevier B.V. All rights reserved.

1. Introduction

The quantitative relation between stimulus and response is perhaps the oldest problem in psychophysics [1]. Our experience suggests that this relation should be nonlinear; a linear relation would lead to an unbounded response, so overexciting the limited capabilities of our neuronal organs. Hence some form of response saturation should exist, limiting the response amplitude to external stimuli. Weber and Fechner proposed, in the 19th century, that the relationship is logarithmic; the magnitude of the response related to a given stimulus of magnitude I given by $P \sim I^{1/4}$ [2].

In 1937 Stevens proposed a more general stimulus-response power law relationship: $P \sim I^n$, where n is a positive exponent that can vary across cases. In fact n is 0.67, for example, for a sound pressure of 3 kHz tone (intensity) the loudness response obeys Stevens' law with $n = 0.67$. However, the value of n may vary according the type of stimulus; the response to a vibrating plate, as captured by the neuronal organs at the finger, leads to different exponents of $n = 0.95$

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Complementary action of chemical and electrical synapses to perception

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ARTICLE INFO	ABSTRACT
Article history: Received 21 February 2015 Available online 17 June 2015	Abstract We study the dynamic range of a cellular automaton model for a neuronal network with electrical and chemical synapses. The neural network is separated into two layers, where one layer corresponds to inhibitory, and the other corresponds to excitatory neurons, that randomly distribute electrical synapses in the network, in order to analyze the effect on the dynamic range. We verify that electrical synapses have a complementary effect on the enhancement of the dynamic range. The enhancement depends on the proportion of electrical synapses as compared to the chemical ones, and also on the layer they appear.
Keywords: Dynamic range Cellular automata Neuron	© 2015 Elsevier B.V. All rights reserved.

1. Introduction

The cerebral cortex contains neurons and their fibres [1]. These neurons are grouped together into functional or morphological units, called cortical areas [2], each of them playing a well-defined role in the processing of information in the brain [3]. Hence the theoretical understanding of the principles of organization and functioning of the cerebral cortex can shed light on the knowledge of many distinct and important subjects in neuroscience [4]. One relevant subject in psychophysics, that analyzes the perception due to external stimuli [5].

Studies about the relation between sensation and stimulus by measuring the quantity of both factors were realized by Weber and Fechner [6]. They proposed that the relation was logarithmic [7]. However, Stevens proposed a theory based on a power law relation between stimulus and response, where the exponent depends on the type of stimulation [8].

The capacity of a biological system to discriminate the intensity of an external stimulus is characterized by the dynamic range [DR] [9]. DR is a range of intensities for which responses can be distinguished. It is the logarithm of the difference between the smallest and the largest stimulus value for which the responses are not too weak to be distinguished or too close to saturation, respectively. The lower and upper bounds are arbitrarily chosen due to the fact that the scaling region is well fit by a power law. In other words, small changes do not affect our results. In visual and the auditory perception have high dynamic range. The human sense of sight can perceive about ten orders of magnitude of luminosity, and the hearing covers twelve decades in a range of intensities of sound pressures [1]. The DR of the human vision is important in the design of high dynamic range display devices [10]. Whereas the DR of the hearing is used for cochlear implants [11].

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Sincronização de disparos em redes neuronais com plasticidade sináptica
(Spikes synchronization in neural networks with synaptic plasticity)

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 Brasília em 03/2015, Arxiv em 03/2015, Publicado em 03/2015

Este artigo investiga a sincronização de disparos neuronais em uma rede neuronal com plasticidade sináptica e perturbação externa. Na simulação, a dinâmica neuronal é descrita pelo modelo de Hodgkin-Huxley, considerando sinapses químicas (excitatórias) entre neurônios. De acordo com a sincronização do disparo é esperado que uma perturbação entre regiões não sincronizadas. No entanto, as trajetórias estacionárias mostram que a combinação de plasticidade sináptica e perturbação externa pode gerar regiões sincronizadas. Este artigo descreve o efeito da plasticidade sináptica na sincronização, onde consideramos a perturbação descrita por uma distribuição binomial. Este estudo é relevante para pesquisas de controle de disfunções neuronais.

Palavras-chave: sincronização, distribuição, sinapses químicas.

In this paper, we investigated the synchronization in a neural network with synaptic plasticity and external perturbation. In the simulation the neural dynamics is described by the Hodgkin-Huxley model considering chemical synapses (excitatory) among neurons. According to neural spikes synchronization is expected that a perturbation produce non-synchronized regions. However, in the literature there are works showing that the combination of synaptic plasticity and external perturbation may generate synchronized regions. This article describes the effect of synaptic plasticity on the synchronization, where we consider a perturbation with a binomial distribution. This study is relevant to researches of neural disorders control.

Keywords: neuron, plasticity, binomial distribution.

1. Introdução

O controle automático de diagnósticos de perturbações neurológicas é uma preocupação de nível mundial, tanto em termos do bem estar humano como no impacto na economia. Tais perturbações incluem além de transmissões cerebrais, inflexões neurológicas, esclerose múltipla, a doença de Parkinson¹, epilepsias e Mal de Alzheimer. Além de alterações primárias de todos os países, independentemente da idade, sexo, nível de educação ou residência, tais doenças também afetam o Póndulo Lateral Basal (PLB) das células, através da falta de substância cinzenta que decorre do desenvolvimento de áreas dorsais mediais, o que nos questiona: “O que seria o sintoma?”

¹ *http://www.who.int/pt-br/news/2014/05/04/*

Desde o progresso do movimento de diagnóstico de doenças que controlam a atenção e a atenção, os sintomas cerebrais devido do controle cerebral para os níveis de corpo humano. Alguns do tratamento neurológico é baseando em medicamentos por atividade excessiva e normal das células neuronais do cérebro.

² *http://www.who.int/pt-br/news/2014/05/04/*

Doenças neurodegenerativas que possuem declínio de funções intelectuais, além a capacidade de trabalhar, redução sensor e ínterferem com a qualidade de vida.

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Sincronização de disparos em redes neuronais com plasticidade sináptica
(Spikes synchronization in neural networks with synaptic plasticity)

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Este artigo investiga a sincronização de disparos neuronais em uma rede neuronal com plasticidade sináptica e perturbação externa. Na simulação, a dinâmica neuronal é descrita pelo modelo de Hodgkin-Huxley, considerando sinapses químicas (excitatórias) entre neurônios. De acordo com a sincronização do disparo é esperado que uma perturbação entre regiões não sincronizadas. No entanto, as trajetórias estacionárias mostram que a combinação de plasticidade sináptica e perturbação externa pode gerar regiões sincronizadas. Este artigo descreve o efeito da plasticidade sináptica na sincronização, onde consideramos a perturbação descrita por uma distribuição binomial. Este estudo é relevante para pesquisas de controle de disfunções neuronais.

Palavras-chave: sincronização, distribuição, sinapses químicas.

In this paper, we investigated the synchronization in a neural network with synaptic plasticity and external perturbation. In the simulation the neural dynamics is described by the Hodgkin-Huxley model considering chemical synapses (excitatory) among neurons. According to neural spikes synchronization is expected that a perturbation produce non-synchronized regions. However, in the literature there are works showing that the combination of synaptic plasticity and external perturbation may generate synchronized regions. This article describes the effect of synaptic plasticity on the synchronization, where we consider a perturbation with a binomial distribution. This study is relevant to researches of neural disorders control.

Keywords: neuron, plasticity, binomial distribution.

1. Introdução

O controle automático de diagnósticos de perturbações neurológicas é uma preocupação de nível mundial, tanto em termos do bem estar humano como no impacto na economia. Tais perturbações incluem além de transmissões cerebrais, inflexões neurológicas, esclerose múltipla, a doença de Parkinson¹, epilepsias e Mal de Alzheimer. Além de alterações primárias de todos os países, independentemente da idade, sexo, nível de educação ou residência, tais doenças também afetam o Póndulo Lateral Basal (PLB) das células, através da falta de substância cinzenta que decorre do desenvolvimento de áreas dorsais mediais, o que nos questiona: “O que seria o sintoma?”

¹ *http://www.who.int/pt-br/news/2014/05/04/*

Desde o progresso do movimento de diagnóstico de doenças que controlam a atenção e a atenção, os sintomas cerebrais devido do controle cerebral para os níveis de corpo humano. Alguns do tratamento neurológico é baseando em medicamentos por atividade excessiva e normal das células neuronais do cérebro.

² *http://www.who.int/pt-br/news/2014/05/04/*

Doenças neurodegenerativas que possuem declínio de funções intelectuais, além a capacidade de trabalhar, redução sensor e ínterferem com a qualidade de vida.

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Evolution work

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Chimeric-like states in a neuronal network model of the cat brain
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ABSTRACT
Neuronal systems have been modeled by complex networks in different descriptions. Recently, it has been realized that the network can simultaneously exhibit an ordered and also an irregular behavior. In this work, we study the existence of chimeric-like states in a network considering the connectivity matrix based on the cat cerebral cortex. The cerebral cortex of the cat can be separated in 10 critical areas separated into four cognitive regions: visual, auditory, somatosensory-motor and frontal cortex. We consider a network where the total dynamics is given by the Hindmarsh-Rose model. The Hindmarsh-Rose equations are a well known model of the neuronal activity that has been considered to simulate the membrane potential of neurons. Then, an order order which exhibits chimeric-like states are present, as well as the effect induced by stimulus of coupling on them. We identify two different kinds of chimeric-like states: spiking chimeric-like state with synchronized spiking and bursting chimeric-like state with desynchronized burst. Moreover, we find that chimeric-like states with desynchronized bursts are more robust to external noise than with desynchronized spiking.
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Brain Plasticity
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Synaptic Plasticity and Spike Synchronization in Neuronal Networks
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ABSTRACT
Brain plasticity, also known as neuroplasticity, is a fundamental mechanism of neuronal adaptation in response to changes in the environment or due to brain injury. In this review, we show our results about the effects of synaptic plasticity on neuronal networks composed by Hodgkin-Huxley neurons. We show that the final topology of the evolved network depends critically on the ratio between the strength of the inhibitory and excitatory synapses. Excitation of the same order of magnitude evokes an evolved network that presents the rich-club phenomenon, well known to exist in the brain. For initial networks with considerably larger inhibitory strengths, we observe the emergence of a complex evolved topology, where neurons sparsely connect to other neurons, also a typical topology of the brain. The presence of noise enhances the strength of both types of synapses, but if the initial network has synaptic weights of both neurons with similar strengths. Finally, we show how the synchronous behavior of the evolved network will reflect the evolved topology.
Keywords: Neuronal network, Plasticity, Synchronization

1. Introduction
The brain¹ is the most complex organ in the human body. It contains approximately 10¹¹ billion neurons and 10¹⁵ synaptic connections, where each neuron can be connected to up to 10⁴ other neurons [1]. The neuron is the basic working unit of the brain and it is responsible for carrying out the communication and the processing of information within the brain [2]. Those tasks are achieved through neuronal firing spikes temporal patterns that are dependent on the neuron own dynamics and the way they are networked.
Towards the goal to understand the brain, over the past several years, mathematical models have been introduced to capture neuronal firing patterns. A simple model that has been considered to describe neuronal spiking is based on the cellular automata [3, 4]. This model uses discrete state variables, coordinates and time [5]. Another proposed firing behavior model is a simplification of the neuron model described by differential equations, where the

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We focus on the existence of chimeric-like states in a neuronal network model based on the cat cerebral cortex. The chimeric states are spatiotemporal patterns in which coherent and incoherent behaviors coexist in the network. With this model we analyze the cat brain, that it is composed by groups of areas. There are many models about chimeric-like states. With Constantinou et al. observed the chaotic motion of the chimeric pattern along an axis of non-trivially broken symmetry [16]. Chimeric and phase states in populations of coupled electrical neurons were studied by Tsodyks et al. [19]. Considering coupled Hindmarsh-Rose neurons, we studied chimeric-like states in approximately equal numbers with spiking sites. There are also experimental investigations about chimeric states e.g., in [20].

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Synchronised firing patterns in a random network of adaptive exponential integrate-and-fire neuron model
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ABSTRACT
We have studied neuronal synchronization in a network network of adaptive exponential integrate-and-fire neurons. We study how spiking or bursting synchronous behavior appears as a function of the coupling strength and the probability of connection by constructing connectivity matrices that identify these stochastic behaviors from measurements of the integrate-and-fire interval and the calculation of the order parameter. Moreover, we verify the existence of synchronization by applying an external perturbation to each neuron. The synchronization state that bursting synchronization is more robust than spiking synchronization.

1. Introduction
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Neural Networks
Spike timing-dependent plasticity induces non-trivial topology in the brain
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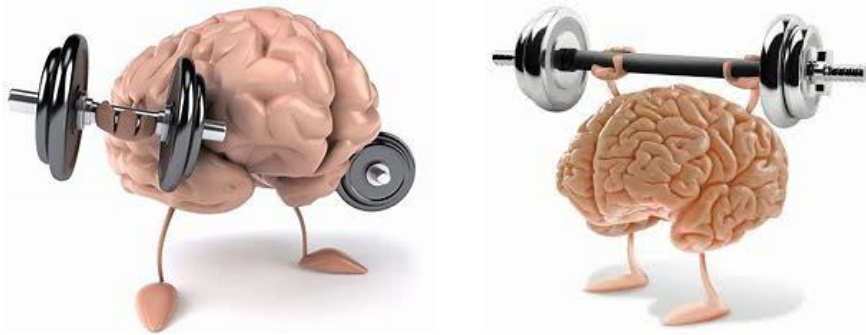
ABSTRACT
Spike timing-dependent plasticity (STDP) is a function of their synchronous behavior. We consider STDP of arbitrary and inhibitory synapses formed by Hebbian rules. We show that the final state of network evolved by a STDP depends on the initial network configuration. Specifically, an initial all-to-all coupling evolves to a complex spiking. Moreover, external perturbations can induce the existence of chimeric-like states where neurons are synchronous and where neurons are desynchronized. This work reveals that STDP based on Hebbian rules leads to a change in the direction of the synaptic between high and low frequency synapses, and therefore, Hebbian learning can be explained in terms of professional attachment between these two diverse communities of neurons, those with low-frequency spiking neurons, and those with higher-frequency spiking neurons.
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1. Introduction
Neuroplasticity, also known as brain plasticity or brain remodeling [1], is the ability of the brain to change its structure and function in response to environmental stimuli. [2]. This process is essential for learning, development, sensory stimulation, or damage [3]. [4]. [5]. [6]. [7]. [8]. [9]. [10]. [11]. [12]. [13]. [14]. [15]. [16]. [17]. [18]. [19]. [20]. [21]. [22]. [23]. [24]. [25]. [26]. [27]. [28]. [29]. [30]. [31]. [32]. [33]. [34]. [35]. [36]. [37]. [38]. [39]. [40]. [41]. [42]. [43]. [44]. [45]. [46]. [47]. [48]. [49]. [50]. [51]. [52]. [53]. [54]. [55]. [56]. [57]. [58]. [59]. [60]. [61]. [62]. [63]. [64]. [65]. [66]. [67]. [68]. [69]. [70]. [71]. [72]. [73]. [74]. [75]. [76]. [77]. [78]. [79]. [80]. [81]. [82]. [83]. [84]. [85]. [86]. [87]. [88]. [89]. [90]. [91]. [92]. [93]. [94]. [95]. [96]. [97]. [98]. [99]. [100]. [101]. [102]. [103]. [104]. [105]. [106]. [107]. [108]. [109]. [110]. [111]. [112]. [113]. [114]. [115]. [116]. [117]. [118]. [119]. [120]. [121]. [122]. [123]. [124]. [125]. [126]. [127]. [128]. [129]. [130]. [131]. [132]. [133]. [134]. [135]. [136]. [137]. [138]. [139]. [140]. [141]. [142]. [143]. [144]. [145]. [146]. [147]. [148]. [149]. [150]. [151]. [152]. [153]. [154]. [155]. [156]. [157]. [158]. [159]. [160]. [161]. [162]. [163]. [164]. [165]. [166]. [167]. [168]. [169]. [170]. [171]. [172]. [173]. [174]. [175]. [176]. [177]. [178]. [179]. [180]. [181]. [182]. [183]. [184]. [185]. [186]. [187]. [188]. [189]. [190]. [191]. [192]. [193]. [194]. [195]. [196]. [197]. [198]. [199]. [200]. [201]. [202]. [203]. [204]. [205]. [206]. [207]. [208]. [209]. [210]. [211]. [212]. [213]. [214]. [215]. [216]. [217]. [218]. [219]. [220]. [221]. [222]. [223]. [224]. [225]. [226]. [227]. [228]. [229]. [230]. [231]. [232]. [233]. [234]. [235]. [236]. [237]. [238]. [239]. [240]. [241]. [242]. [243]. [244]. [245]. [246]. [247]. [248]. [249]. [250]. [251]. [252]. [253]. [254]. [255]. [256]. [257]. [258]. [259]. [260]. [261]. [262]. [263]. [264]. [265]. [266]. [267]. [268]. [269]. [270]. [271]. [272]. [273]. [274]. [275]. [276]. [277]. [278]. [279]. [280]. [281]. [282]. [283]. [284]. [285]. [286]. [287]. [288]. [289]. [290]. [291]. [292]. [293]. [294]. [295]. [296]. [297]. [298]. [299]. [300]. [301]. [302]. [303]. [304]. [305]. [306]. [307]. [308]. [309]. [310]. [311]. [312]. [313]. [314]. [315]. [316]. [317]. [318]. [319]. [320]. [321]. [322]. [323]. [324]. [325]. [326]. [327]. [328]. [329]. [330]. [331]. [332]. [333]. [334]. [335]. [336]. [337]. [338]. [339]. [340]. [341]. [342]. [343]. [344]. [345]. [346]. [347]. [348]. [349]. [350]. [351]. [352]. [353]. [354]. [355]. [356]. [357]. [358]. [359]. [360]. [361]. [362]. [363]. [364]. [365]. [366]. [367]. [368]. [369]. [370]. [371]. [372]. [373]. [374]. [375]. [376]. [377]. [378]. [379]. [380]. [381]. [382]. [383]. [384]. [385]. [386]. [387]. [388]. [389]. [390]. [391]. [392]. [393]. [394]. [395]. [396]. [397]. [398]. [399]. [400]. [401]. [402]. [403]. [404]. [405]. 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[690]. [691]. [692]. [693]. [694]. [695]. [696]. [697]. [698]. [699]. [700]. [701]. [702]. [703]. [704]. [705]. [706]. [707]. [708]. [709]. [710]. [711]. [712]. [713]. [714]. [715]. [716]. [717]. [718]. [719]. [720]. [721]. [722]. [723]. [724]. [725]. [726]. [727]. [728]. [729]. [730]. [731]. [732]. [733]. [734]. [735]. [736]. [737]. [738]. [739]. [740]. [741]. [742]. [743]. [744]. [745]. [746]. [747]. [748]. [749]. [750]. [751]. [752]. [753]. [754]. [755]. [756]. [757]. [758]. [759]. [760]. [761]. [762]. [763]. [764]. [765]. [766]. [767]. [768]. [769]. [770]. [771]. [772]. [773]. [774]. [775]. [776]. [777]. [778]. [779]. [780]. [781]. [782]. [783]. [784]. [785]. [786]. [787]. [788]. [789]. [790]. [791]. [792]. [793]. [794]. [795]. [796]. [797]. [798]. [799]. [800]. [801]. [802]. [803]. [804]. [805]. [806]. [807]. [808]. [809]. [810]. [811]. [812]. [813]. [814]. [815]. [816]. [817]. [818]. [819]. [820]. [821]. [822]. [823]. [824]. [825]. [826]. [827]. [828]. [829]. [830]. [831]. 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Spike timing-dependent plasticity induces non-trivial topology in the brain

Neural Networks

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Our main goal is to show that spike timing-dependent plasticity of excitatory and inhibitory synapses induces non trivial topologies in the plastic brain.

Initial networks of neurons fully connected, evolve to a non trivial complex network.

Consequently, this non-trivial topology alters the synchronous behavior

We have considered: Initial network with a global coupling, with chemical synapses where the connections are unidirectional, and the local dynamics is described by the Hodgkin–Huxley model

Model Hodgkin-Huxley

$$\begin{aligned}C\dot{V}_i &= I_i - g_K n_i^4 (V_i - E_K) - g_{Na} m_i^3 h_i (V_i - E_{Na}) \\ &\quad - g_L (V_i - E_L) + \frac{(V_r^{\text{Exc}} - V_i)}{\omega_{\text{Exc}}} \sum_{j=1}^{N_{\text{Exc}}} \varepsilon_{ij} S_j \\ &\quad + \frac{(V_r^{\text{Inhib}} - V_i)}{\omega_{\text{Inhib}}} \sum_{j=1}^{N_{\text{Inhib}}} \sigma_{ij} S_j + \Gamma_i, \\ \dot{n}_i &= \alpha_{n_i}(V_i)(1 - n_i) - \beta_{n_i}(V_i)n_i, \\ \dot{m}_i &= \alpha_{m_i}(V_i)(1 - m_i) - \beta_{m_i}(V_i)m_i, \\ \dot{h}_i &= \alpha_{h_i}(V_i)(1 - h_i) - \beta_{h_i}(V_i)h_i,\end{aligned}$$

The excitatory eSTDP is given by

$$\Delta \varepsilon_{ij} = \begin{cases} A_1 \exp(-\Delta t_{ij}/\tau_1), & \Delta t_{ij} \geq 0 \\ -A_2 \exp(\Delta t_{ij}/\tau_2), & \Delta t_{ij} < 0, \end{cases}$$

$$\Delta t_{ij} = t_i - t_j = t_{\text{post}} - t_{\text{pre}}$$

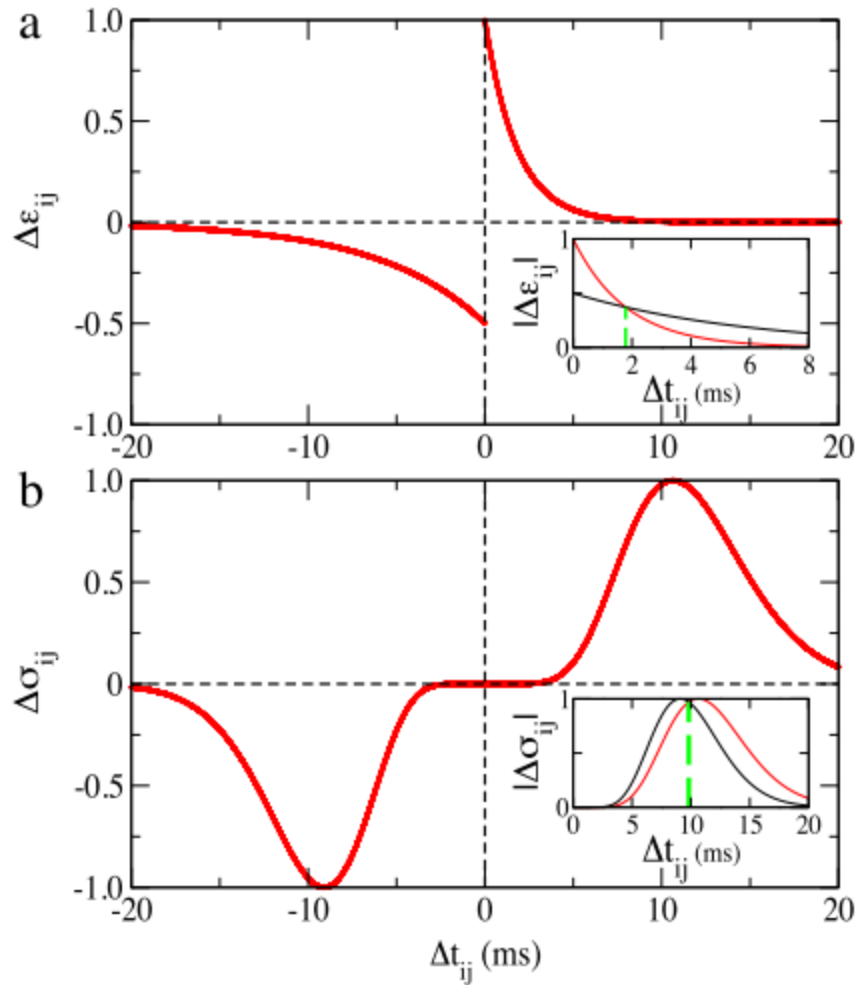


Fig. 1(a): result obtained for $A_1 = 1.0$, $A_2 = 0.5$, $\tau_1 = 1.8$ ms, and $\tau_2 = 6.0$ ms.

The green dashed line: denotes the Δt_{ij} value at which the curves of potentiation and depression intersect.

The inset in Fig. 1(a) shows that for $|\Delta t_{ij}| < 1.8$ ms the potentiation of ε_{ij} is bigger than the depression.

iSTDP (inset in Fig. 1(b)) the potentiation of σ_{ij} is bigger than the depression for $|\Delta t_{ij}| > 9.8$ ms.

$$\Delta \sigma_{ij} = \frac{g_0}{g_{\text{norm}}} \alpha^\beta |\Delta t_{ij}| \Delta t_{ij}^{\beta-1} \exp(-\alpha |\Delta t_{ij}|),$$

g_0 - scaling factor accounting for the amount of change in inhibitory conductance induced by the synaptic

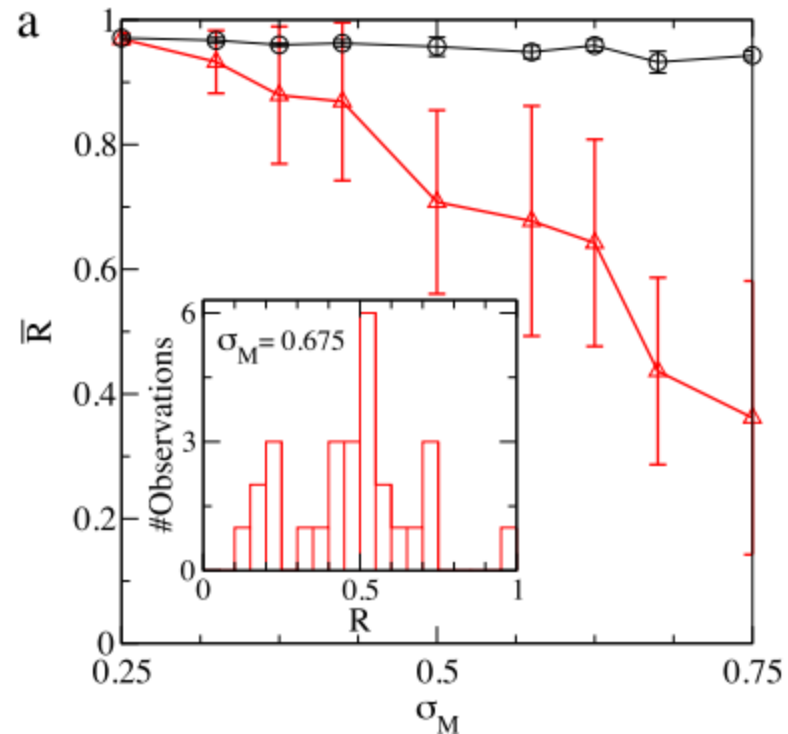
$g_{\text{norm}} = \beta \exp(-\beta)$ is the normalizing constant.

Fig. 1(b) exhibits the result obtained from Eq. (14)

As a consequence, $\Delta \sigma_{ij} > 0$ for $\Delta t_{ij} > 0$, and $\Delta \sigma_{ij} < 0$ for $\Delta t_{ij} < 0$. The initial inhibitory synaptic weights σ_{ij} are normally distributed with mean and standard deviation equal to σ_M .

Then, the coupling strengths are updated according to Eq. (14), where $\sigma_{ij} \rightarrow \sigma_{ij} + \Delta \sigma_{ij}$. The updates for ε_{ij} and σ_{ij} are applied for the last postsynaptic spike.

Fig.2(a) shows the mean order parameter (R) that is calculated for different initial conditions.



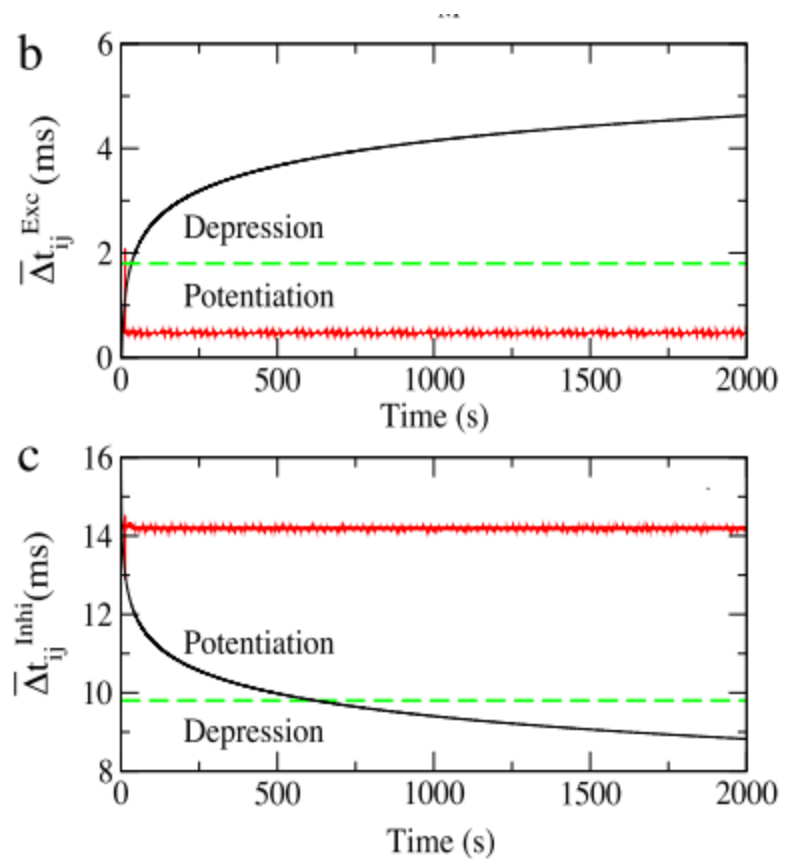
Function of the inhibitory coupling strength σ_M for a neural network with excitatory and inhibitory synapses. Case without STDP (black circles) and STDP (red triangles).

For ϵ_M equal to 0.25 and varying σ_M , we do not observe a significant alteration of the R value without STDP - initially the network has an all-to-all topology.

Increase of σ_M and present a large standard deviation. This standard deviation occurs due to the existence of different synchronization states.

The upper border of the inhibitory coupling 2σ and the different initial conditions are important to change the dynamics of the network with STDP and without external perturbation.

This is verified by means of the decay of the R values and the large standard deviation bar.



In Fig. 2(b) and (c): $\sigma_M = 0.675$, for different configurations of the initial networks and $\tau = 100$ ms.

The black line shows the case in which the network goes to a desynchronized state ($R \approx 0.1$), whereas the red line exhibits the case of a network that presents synchronous behavior ($R \approx 1$).

In both cases, we consider the same parameters, except the seed to generate the random distribution of the constant current density I_i .

Through Fig. 2(b) and (c) it is possible to verify why and when the coupling matrix suffer substantial changes. The transition occurs when the black or red curves cross the green line.

At this time, depreciation induces weak strength in the coupling matrix, and potentiation induces strong strength.

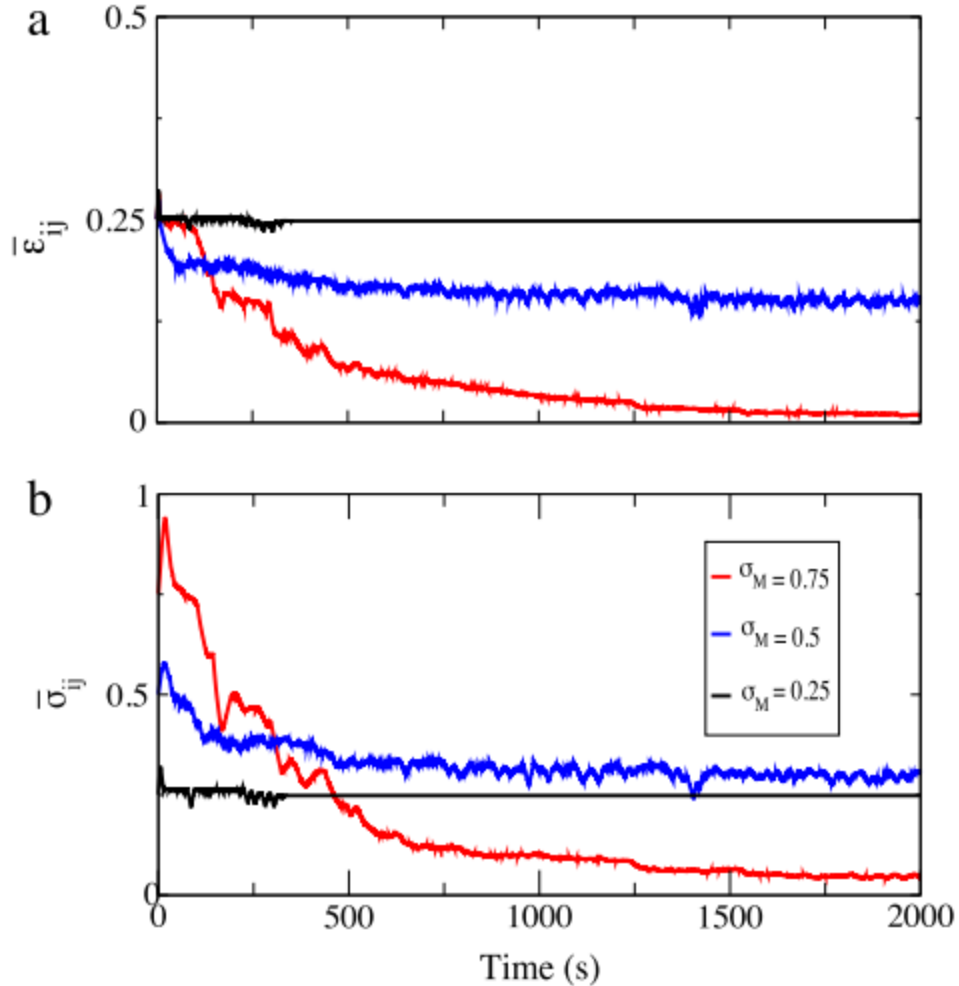
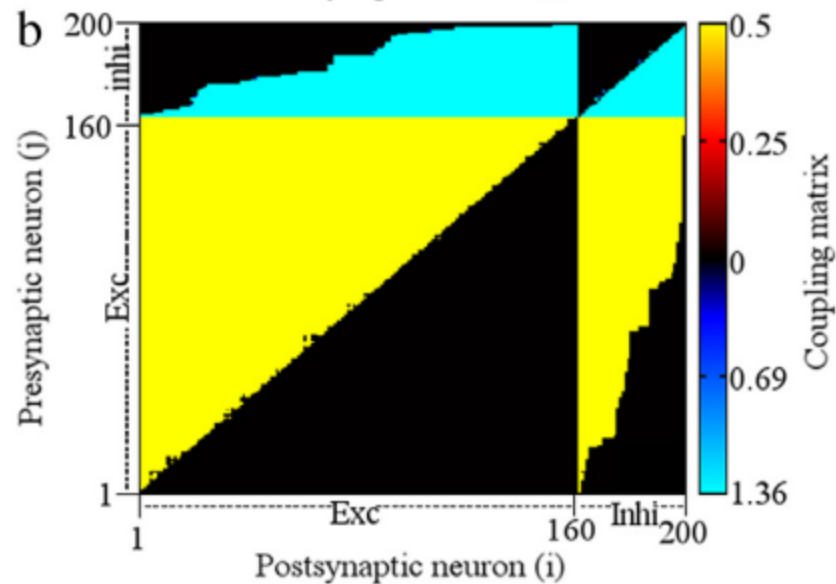
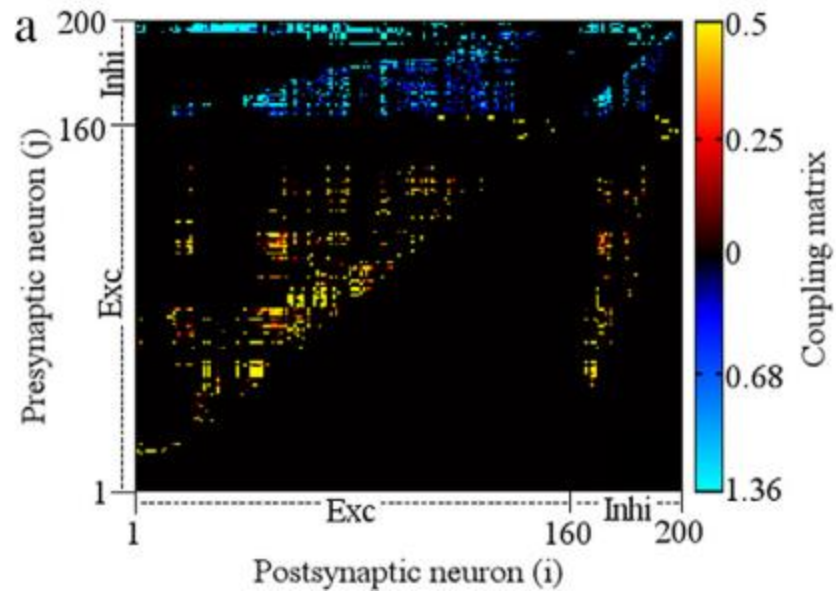


Fig. 3 exhibits the time courses of the mean excitatory (Fig. 3(a)) and inhibitory (Fig. 3(b)) coupling strengths from the multiple coexisting regimes that are shown in Fig. 2(a).

We see that for $\sigma_M = 0.25$ both $\bar{\epsilon}_{ij}$ and $\bar{\sigma}_{ij}$ have constant values for the time approximately greater than 700 s, and the learning produces a triangular-type connecting matrix (as shown in Fig. 4), meaning that the connections among all neurons become preferentially directed.

For $\sigma_M = 0.5$ the $\bar{\epsilon}_{ij}$ values decrease to approximately 0.15, while $\bar{\sigma}_{ij}$ values oscillate about 0.25, and the coupling matrix becomes partitioned, indicating the existence of larger clusters.

Increasing the upper border σ_M to 0.75 both $\bar{\epsilon}_{ij}$ and $\bar{\sigma}_{ij}$ tend to 0, and the coupling matrix becomes sparse.



The synaptic weights are suppressed in the desynchronized regime (Fig. 4(a)), coupling matrix presents a small number of connections. This behavior: black lines in Fig. 2(b) and (c).

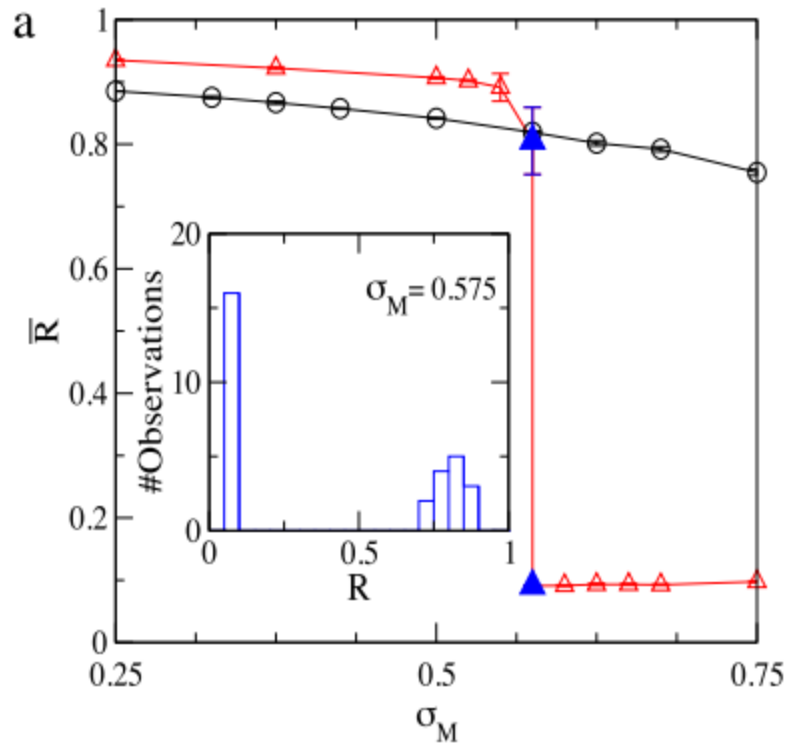
In addition, the synaptic weights are potentiated (red lines in Fig. 2(b) and (c)) in the synchronized regime (Fig. 4(b)), and the coupling matrix exhibits a triangular shape.

The synchronous behavior has a dependence on the direction of synapses. When the presynaptic neurons are excitatory the synapses from the high frequency to the low frequency neurons become stronger.

Presynaptic neurons are inhibitory, the synapses from the low frequency to the high frequency neurons become stronger.

Fig. 4 shows the final topologies for two networks initially set with a global coupling topologies after being evolved by a STDP process.

We see that the STDP induces a non-trivial topology in the network resulting in networks sparsely connected, moderately connected (Fig. 4(a)), or densely connected with strong preferential attachment (Fig. 4(b)).

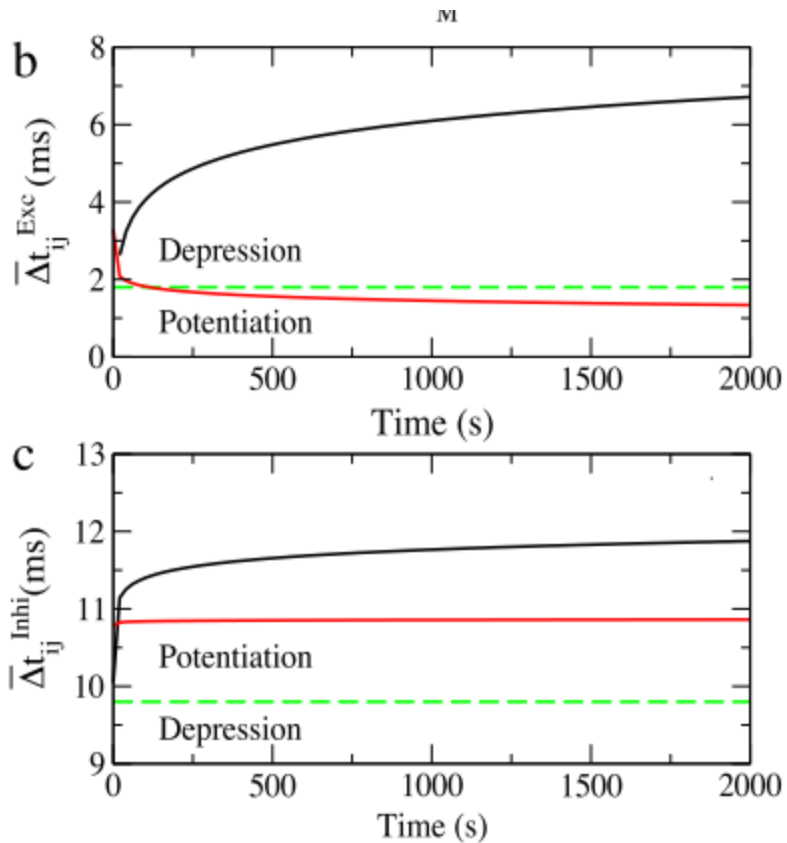


Considering an external perturbation ($\Gamma_i > 0$), we also study the cases without and with plasticity. In the case without STDP, we verify that the mean order-parameter has a small decay when σ_M increases, as shown in Fig. 5(a) with black circles.

The red triangles represent the case with STDP, and unlike the case without perturbation (Fig. 2(a)), there is an abrupt transition (blue triangles), due to a first-order transition in the average order parameter.

The upper border of the inhibitory coupling is relevant to produce alteration in the dynamics, while the different initial conditions are important only at the transition.

Based on the results in the inset (Fig. 5(a)), we verify that the network in the transition can be either in one of the states: (i) high R with potentiation of the average-time difference for excitatory and inhibitory connections (red lines in Fig. 5(b) and (c)), or (ii) low R with excitatory average time-difference in the depression region and inhibitory in the potentiation region (black lines).



The transition from the synchronized to the desynchronized states was reported in studies on how stimulation impact on neurological disorders induced by an abnormal neuronal synchronization (Popovych & Tass, 2012; Tass & Majtanik, 2006).

A first order transition was also observed in Popovych et al. (2013) when the stimulation intensity varies in a neural network with eSTDP.

In our simulations, we observe the transition to desynchronization caused by a variation in the inhibitory coupling in neural networks with both eSTDP and iSTDP.

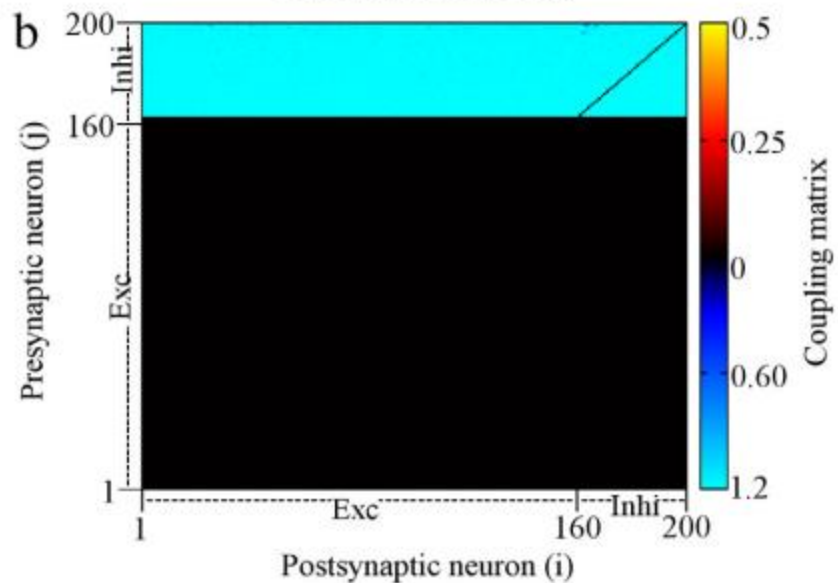
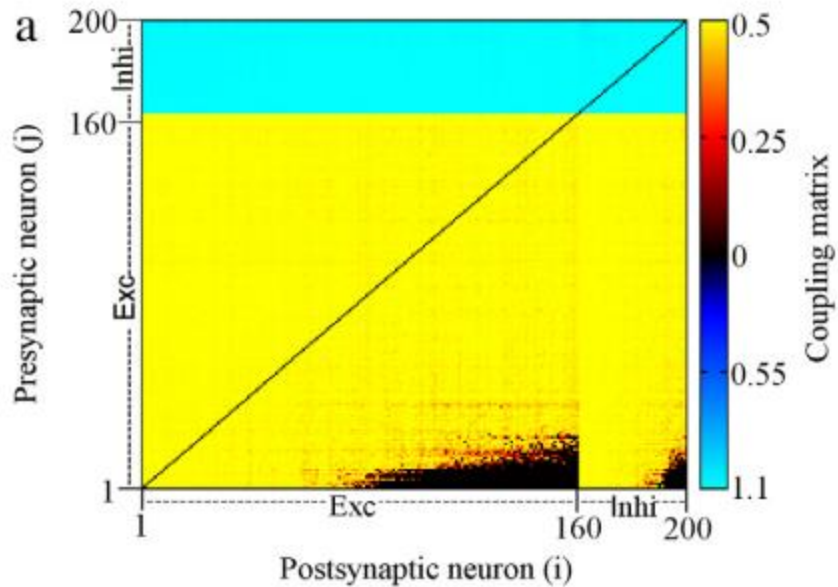


Fig. 6 illustrates the coupling matrix for the two states of the first-order transition.

In Fig. 6(a), we can see the coupling configuration that corresponds to high R.

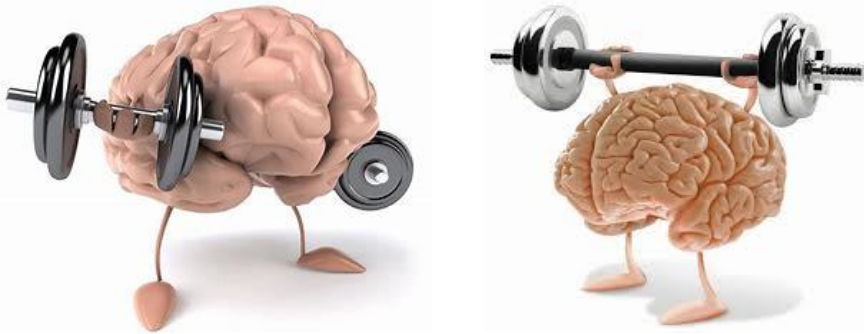
The network presents high connectivity, and for this reason it is possible to observe synchronous behavior.

For the case of low R, we verify that the network has only connections from neurons belonging to the inhibitory population to any other neuron, as shown in Fig. 6(b).

Spike timing-dependent plasticity induces non-trivial topology in the brain

Neural Networks

<http://dx.doi.org/10.1016/j.neunet.2017.01.010>



In our results, we have observed for some parameter conditions not only the improvement of neural spiking synchronization, but also for other parameter conditions that promote desynchronization.

The onset of synchronicity comes along side with desynchronicity in the plastic brain. This balance between different synchronous behaviors is vital to maintain a fundamental property of a brain network.

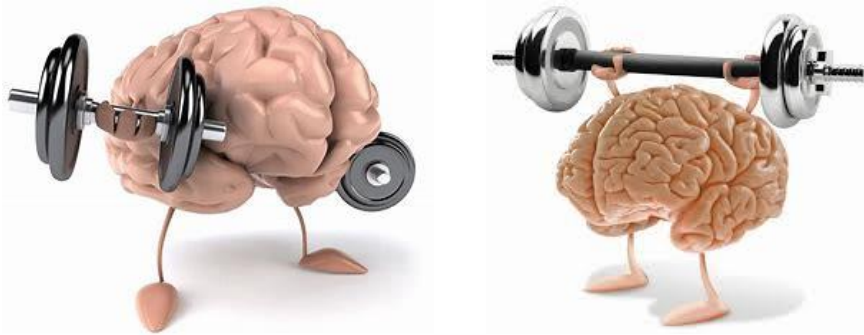
Clusters need to be sufficiently synchronous for information to be efficiently exchanged, but at the same time sufficiently desynchronous to behave independently.

Finally, we show that when there is an external perturbation, the plastic neural network has an abrupt change in behavior characterized by a first-order transition.

Spike timing-dependent plasticity induces non-trivial topology in the brain

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In conclusion, we have studied the effects of spike timing dependent plasticity on the synchronous behavior and the evolved connecting topology of neural networks constructed with Hodgkin–Huxley neurons.

Regarding the evolved topology, our main conclusion is that learning under a STDP results in evolved networks that present complex topology.

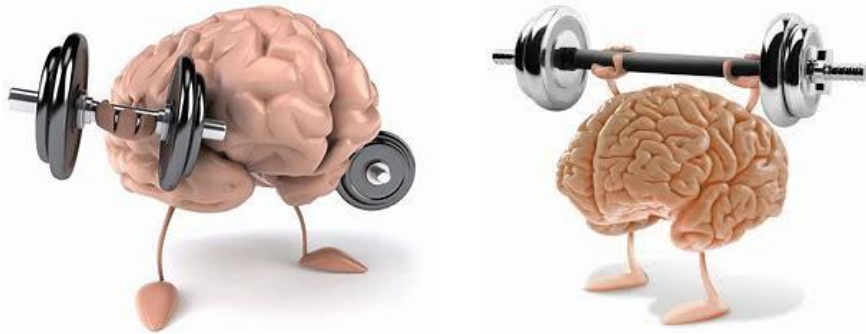
Concerning the dynamic synchronous behavior of the evolved networks, we observe that the studied networks exhibit concurrent synchronous and non synchronous states with characteristics that depend on both the upper border of the inhibitory coupling and the initial conditions.

Specifically, we verify that the main role of the inhibitory connections is to produce a delay in the spiking time of the postsynaptic neurons.

Spike timing-dependent plasticity induces non-trivial topology in the brain

Neural Networks

<http://dx.doi.org/10.1016/j.neunet.2017.01.010>



As a consequence, the increase of the inhibitory coupling strength can suppress synchronous behavior, which contributes to a decrease in the mean order parameter.

Moreover, the transition from low to a high synchronous state is smooth by alterations of the inhibitory synapses.

When a random external perturbation is introduced in the network, this transition becomes discontinuous, i.e., we observe a first-order transition.

Similarly to the non-perturbed network, we also find coexistence of synchronous and non-synchronous neurons in the perturbed networks.

WORKSHOP ON DYNAMICAL PROCESSES ON COMPLEX NETWORKS



May 13 – 17, 2024

at Instituto de Física Teórica - UNESP, Brazil

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Guilherme Ferraz de Arruda (CENTAI I, Italy)

Marcelo Avila (U. Mayor de San Andrés, Bolivia)

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Jean-Gabriel Young (U. of Vermont, USA)

Complex systems are characterized by a large number of units, such as particles, individuals or neurons, that interact typically with a few neighbors but lead to the emergence of large-scale collective behavior. Networks provide a natural representation of these systems, where nodes play the role of the units, and links between nodes indicate pairwise interactions. The distribution of links among the nodes is a key property of networks, defining how the units of the system interact. Links may follow simple rules, such as regular lattices or random connections, or may be highly heterogeneous, displaying power law distributions. More recently, the concepts of multilayer and higher-order networks have emerged to describe interconnected sets of networks and many-body interactions, where single-layer networks are generalized to simplicial complexes or hypergraphs.

Two of these processes have become particularly important and will be the focus of this workshop in terms of applications. The first is the spreading of infectious diseases and the dissemination of information. As disease and information propagation depend critically on the network of contacts between people, understanding how the topology of these networks affects the spreading and how that, in turn, modifies the network via quarantine, vaccination, use of masks, or death, has become a major topic of research. A second topic to be discussed in the workshop is the synchronization of coupled oscillators. Understanding how independent oscillators synchronize their motion when coupled together has become an important area of research, both in terms of applications to neuronal dynamics and swarms, and to basic science, characterizing its phase transitions, hysteresis, and dynamical properties.

There is no registration fee and limited funds are available for travel and local expenses.

Registration deadline:
March 24, 2024

Online registration and more information:
<https://www.ictp-saifr.org/wdpcn2024/>



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