ISTANBUL TECHNICAL UNIVERSITY ★ GRADUATE SCHOOL OF SCIENCE ENGINEERING AND TECHNOLOGY

MODELLING THE NEOCORTICAL PYRAMIDAL NEURONS AND THEIR GROUP BEHAVIOUR

M.Sc. THESIS

Sadeem Nabeel Saleem KBAH

Department of Electronic And Communication Engineering

Biomedical Engineering Master Programme

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İSTANBUL TEKNİK ÜNİVERSİTESİ ★ FEN BİLİMLERİ ENSTİTÜSÜ

NEOKORTİKAL PİRAMİD NÖRONLARIN MODELLEMESİ VE GRUP DAVRANIŞLARI

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Sadeem Nabeel Saleem KBAH, a M.Sc. student of ITU Graduate School of Science Engineering and Technology 504101409 successfully defended the thesis entitled **"MODELLING THE NEOCORTICAL PYRAMIDAL NEURONS AND THEIR GROUP BEHAVIOUR"**, which he/she prepared after fulfilling the requirements specified in the associated legislations, before the jury whose signatures are below.

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To my parents, Nabeel Kubba and Alaa Alshalchi, who endlessly taught us all what we know and did their best to present us a comfortable life,

FOREWORD

First, I would like to dedicate special thanks to Assoc. Prof.Dr. Neslihan Serap Sengor, supervisor of this project, without her directions, genuine interest, endless patient and dedication, this work would not be done.

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ABBREVIATIONS

NN	**:** Neural Network
AP	**:** Action Potential
HH	**:** Hodgkin Huxley
WHO	**:** World Health Organization
IAF	**:** Integrate and fire
RAF	**:** Resonate and fire
DLA	**:** Dynamic Link Architecture
Burden	**:** Brain diseases (neurological, neurosurgical and psychiatric diseases together)
GBD	**:** The Global Burden of Disease

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MODELLING THE NEOCORTICAL PYRAMIDAL NEURONS AND THEIR GROUP BEHAVIOUR

SUMMARY

The cerebral cortex is the outer covering of grey matter over the hemispheres. It is composed of two hemispheres and considered as the most advanced part of the brain as it is responsible of many functions including sensory functions as interpreting sensory incomes, cognitive functions as thinking, analyzing and understanding language. Understanding language is considered as the most complex and difficult job along cognitive functions. By studying cortex we can learn more about brain dysfunctions during mental disorders and neurological diseases. These kind of studies can be done only by modeling and simulation.

Brain simulation is necessary for many reasons, one of them is that real system does not exist and it is costly or impossible to build and experiment with prototypes.

The other advantages of brain modeling is to trace the connections between individual neurons in animal models. Such improvements can be applied and further studied by classifying into two groups:

1-To do a hard-wired connection schemes and

2-To create a structure-learning algorithms inspired by animal learning.

In this thesis we made a simple model for the cerebral cortex of human and rat. We did this by taking in our considerations the number and type of the composing cells avialable in the cerebral cortices of both human and rat. We also considered the spiking properties of the neurons in the cortex. So, our work can be classified under the neurocomputational theories, which states that in the produced model, both the behavior and biological restrictions must be emphasized.

In order to be sure that expected accuracy of simulation is consistent with needed requirements and get the right results we used Izhikevich model as the neuron model. We preferred it over the other available models, since it seems to be more suitable as it is, and

• Easer to be programmed and computed.

• More realistic since it can create more spiking types similar to that presented by biological cells.

Basing on this we made the model of the single Pyramidal Neuron and then study the behavior of group of neurons when connected to each other in a way similar to the real connections in the original cortical tissue.

In our modeling we used the new coded BRIAN simulator which had been coded over PYTHON by group of postdoctoral researchers. We preferred it since it saves the time of processors, easy to learn and use, highly flexible and easily extensible. It is also specialized in simulating neural network spiking compared to other normal programs

like MATLAB, and can simulate both single neuron and large connected network of neurons which is also a property compared to other neural simulating programs like NEURON.

We also studied the synchronization and the factors affecting the existence of synchronization between two group of neurons. The importance of measuring the synchronization is that it indicates the degree of coherence between neuron groups that are interactive with each other. Synchronization have a role in the well-timing of coordination and communication between different cortical neurons while cortical tasks are realized, specially in determining the amount of neurons being engaged in cognitive processes.

Finally, we can conclude that increasing the weight of connection will increase the connection between neurons, the synchronization also increases to some extend, in a nonlinear relationship.

Increasing the sparseness also increase the connection which in turn increase the synchronization between the neuron groups

The synchronization affected by other factors like type of spiking, during simulation we noticed RS and FS types of spiking shows higher values of synchronization compared to other types of spiking with the same values of weight and sparseness. Obviously in large scale networks, the synchrony needs a high number of connections between neurons and to overtake some threshold value, which is controlled by the changing of the weights of connection and the sparseness.

NEOKORTİKAL PİRAMİD NÖRONLARIN MODELLEMESİ VE GRUP DAVRANIŞLARI

ÖZET

Serebral korteks bir diğer deyişle beyin kabuğu, canlılar içinde primatlarda en gelişmiş bölgedir. Gri madde olarak da adlandırılır ve beynin her iki yarım küresinde bir örtü şeklindedir. Düşünme, algı, dil gibi üst düzey bilişsel süreçlerden sorumludur. Duyusal verileri algılama ve anlamlandırma, karar verme, öğrenme gibi bilişsel süreçlerin oluşmasında korteksin farklı kısımları rol alır.

Korteksin yapısı ve sorumlu olduğu işlevlerin oluşmasını inceleyerek nörolojik düzensizlikler ve rahatsızlıklar hakkında daha fazla bilgi sahibi olabiliriz. Bilişsel süreçlerin oluşmasında etkili olan korteksin davranışlarına ilişkin yapılan çalışmalar, özellikle beyin dinamiğini anlamaya yönelik olan çalışmalar, genellikle süreçlerin oluşumu sırasında gözlemlenen işaretlerin toplanması ve sınıflandırılmasına yönelik olmuştur. Süreçlerin nasıl ve neden oluştuğuna ilişkin öneriler, yaklaşımlar ancak son yıllarda, özellikle beynin süreçler esnasında görüntülenmesine ilişkin geliştirilen araçlar sayesinde mümkün olmaya başlanmıştır. Özellikle, EEG işaretlerine ilişkin çok sayıda veri toplanmasına rağmen, hala daha bu işaretlerin oluşmasında yer alan mekanizmalar bilinmemektedir. Bu mekanizmalara ilişkin önerilen yaklaşımları analiz etmek ve irdelemek için hesaplamalı modeller önemli bir araçtır. Önerilen hesaplamalı modeller, farklı seviyelerdeki oluşumları içermektedir. Tek hücre davranışından, hücrelerin oluşturduğu grupların davranışlarına kadar değişen bu seviyelerdeki incelemeler sinirbiliminin ilgi alanı içindedir.

Modelleme ve benzetim aracılığıyla da sinirbilimdeki çalışmalara katkı sağlanmasına özellikle son yıllarda önem verilmektedir. Dinamik sistemlerin analizine ilişkin geliştirilen matematiksel yöntemler benzetim araçlarının geliştirilmesinde etkili olmuştur. Benzetim içinde farklı seviyelerde etkili çeşitli araçlar geliştirilmiştir. Tek hücre modellemesinde etkili olan NEURON, hücre gruplarının davranışlarını incelemekte çokca kullanılan NEST ve BRIAN, dinamik sistem açısından detaylı çalışmalar yapılmasına yardımcı olan XPPAUT bu araçlardan ilk akla gelenlerdir. Bu tez çalışmasında da bu araçlardan Phyton tabanlı bir yazılım olan BRIAN'dan yararlanılmıştır.

Tez çalışmasında, korteksdeki farklı sinir hücresi tiplerinden en yoğun olarak bulunan piramid ve stellate yapısındaki hücreler ele alınmıştır. Öncelikle bu hücrelerin, davranışları sinirbilim literatüründe mevcut çok sayıdaki kaynaktan yararlanılarak incelenmiş ve bu davranışlar, Izhikevich tarzı sinir hücresi modeli ile BRIAN ortamında yeniden elde edilmiştir. Izhikevich hücre modelini ile piramid ve stellate tipi hücrelerin farklı davranışlarını elde etmek için modele ilişkin parametreler değiştirilmiştir. Böylece iki diferansiyel denklem ve bir yenileme (reset) koşulu ile ifade edilen Izhikevich hücre modeli ile, normal vuru, patlama tarzı vuru, hızlı ateşleme gibi sinir hücresi dinamiğine ilişkin değişik davranışlar elde edilmiştir.

Tez çalışmasında piramid ve stellate tarzı hücrelerin davranışları elde edildikten sonra, bu hücrelerin birbirleri ile bağlantıları ele alınmış, ve hücre gruplarının davranışları özellikle senkronizasyon gözönüne alınarak incelenmiştir. Hücre grupları oluşturulurken uyarıcı ve baskılayıcı hücre grupları farklı davranışlar gösteren hücreler ile modellenmiş ve bu iki tür hücre grubundaki hücre sayısı oransal olarak insan ve fare beynindeki oranlar gözetilerek belirlenmiştir. Biyolojik gerçekliğe uygun, böylece oluşturulan farklı modeller ile uyarıcı ve baskılayıcı hücre gruplarının bağlantı yoğunluğu ve ağırlıklarının senkronizasyona etkisi ele alınan senkronizasyon ölçütü çercevesinde incelenmiştir. Bu incelemeler için BRIAN ortamında hazırlanan yazılımlar kullanılmıştır. Tezin ilk bölümünde tezde ele alınan yaklaşım genel olarak anlatılmış, ikinci bölümünde korteks hakkında hem yapısal hemde işlevsel anlamda detaylı bilgi verilmitir. Bu bölümün sonunda korteksde çok sayıda bulunan piramid hücre yapısının farklı davranışları Izhikevich Hücre modeli ile elde edilmiştir. Böylece bir sonraki bölünde oluşturulacak gruplarda kullanılacak hücre davranışlarına ilişkin model parametreleri belirlenmiştir. Üçüncü bölümde Izhikevich hücre modellerinden yararlanarak hücre grupları oluşturulmuş bu hücre gruplarının BRIAN ortamında nasıl oluşturulduğuna dair detaylı bilgi verilmiştir. Elde edilen modellerden yararlanarak senkronizasyonu etkilediği düşünülen bağlantı ağırlıkları ve bağlantı yoğunluğunun etkisi incelenmiştir. Son bölümde elde edilen benztim sonuçları tartışılmıştır.

Beyindeki bölgelerde gözlemlenen senkronizasyon özellikle EEG işaretleri bağlamında çokca ele alınmıştır. Senkronizasyon, kimi zaman bir ödevin yerine getirilmesinde farklı bölgeler arasındaki iletişimi belirtse de kimi zaman da Parkinson, Alzheimer hastalıklarında olduğu gibi nörolojik defermasyonun bir ölçütü olmaktadır. Sinir hücre gruplarının birlikte davrandığının bir ölçütü olan senkronizasyona ilişkin olarak literatürde çoğunlukla deneysel sonuçlar verilmektedir. Ancak senkronizasyonun arkasındaki nedenleri anlayabilmek için son yıllarda hesaplamalı modellerden de yararlanılmaya başlanmıştır.

Bu tez çalışması da bu çerçevede değerlendirilebilinir. BRIAN ortamında hazırlanan yazılımlar ile elde edilen sonuçlarda hücreler arasındaki ağırlığın artması ile senkronizasyonun da artığı, bağlantı yoğunluğunun da benzer bir özellik gösterdiği gözlemlenmiştir. Bu sonuçlar hem raster diyagramları hem de senkronizasyon ölçütünün bağlantı ağırlıkları ve yoğunlukları ile değişimini gösteren diyagramlar ile verilmiştir. Raster diyagramları zaman içinde senkronizasyondaki değişimi gözönüne koyarken, senkronizasyon ölçütündeki değişimleri gösteren diyagramlar daha genel ve bütünlükçü olarak elde edilen sonuçları özetlemektedir. Uyarıcı ve bastırıcı gruplardaki sonuçlar diyagramlarda ayrı ayrı verildiğinden ağırlıkların ve bağlantı yoğunluklarının bu iki grup üzerindeki etkisi farklı hücre yapıları ve oranlar içinverilen grafiklerden gözlenebilir. Benzetim sonuçları elde edilirken, hem her iki grup arasında hem de gruplar içindeki bağlantı topolojisi sadece her hücre her hücre ile bağlanır yapısı çerçevesinde ele alınmıştır. Bu ağ yapısının tercih edilmesi, literatürde bu konuda fazla çalışma olmadığından en olası ağ yapısı olarak bu yapı akla yakın geldiğindendir. Hücreler arasındaki bağlantı dinamiği ise BRIAN ortamında kullanılan en basit dinamik model olarak ele alınmış, özel bir yapı denenmemiştir.

Tezde elde edilen sonuçlar daha farklı bağlantı dinamikleri, ve farklı ağ yapıları gözönüne alınarak geliştirilebilir. Özellikle korteksin farklı bölgeleri daha dikkatle modellenerek, ölçüm sonuçlarının derlendiği süreçlere ilişkin hesaplamalı modeller geliştirlebilir. Bu tez çalışması ile BRIAN ortamında böylesi çalışmaların yapılmasının

mümkün olabileceği görülmüştür. Izhikevich sinir hücresi modelinden farklı modellerde sınanabilir, ancak tezde de geniş şekilde açıklandığı gibi büyük boyutlu bir modelleme için fizyolojik açıdan daha gerçekçi olan Hodgkin-Huxley hücre modelini kullanmak uygun değildir. Izhikevich sinir hücresi gibi daha basit ancak farklı dinamikleri gösteren hücre modelleri kullanılabilir.

Fazla sayıda hücrenin bir araya gelerek oluşturduğu grupların davranışlarını incelemek için uygun bir ortam yaratan, farklı hücre modelleri ve bağlantı dinamiklerinin incelenmesini sağlayan BRIAN ortamı, bilişsel süreçlerin oluşmasındaki mekanizmaları açıklamaya yönelik çalışmaların yapılmasına olanak sağlamaktadır. Bu tür yazılımlardan yararlanarak, bir deney ortamı oluşturmak ve bilişsel süreçler sırasında beyinde oluşan mekanizmalara ilişkin farklı varsayımları sınamak mümkündür. Bu çalışmada sadece korkeks ile ilgilenilmiş ve özellikle gözlemlenen EEG işaretlerinin açıklamasına ilişkin bir model geliştirilip geliştirilemiyeceğine ilişkin basit sınamalar yapılmıştır. Elbette sadece korteks değil beyinde bilişsel süreçlere katkıda bulunan diğer yapılarda ele alınarak modeller geliştirilebilinir. Böylece süreçlere ilişkin daha kapsamlı modeller elde edilmiş olduğu gibi bu modeller aracılığı ile yaşlanan dünyamızda hem ekonomik hem de insani nedenlerden dolayı bir sorun olan Parkinson Alzheimer hastalıkları gibi nörodejeneratif hastalıkların erken teşhisi ve tedavisi içinde yöntemler geliştirilebilinir.

1. INTRODUCTION

The human brain is composed of approximately 10 to the power of 11 of specialized cells called neurons. These cells can be electrically activated when being simulated by an external source. These cells are capable of producing special type of proteins that work as connecting channels called ionic channels which are inserted in the cell membrane. These channels connect selectively the internal and external of the neuron cells. These neurons are connected to each other by movable ions, which are electrically charged molecules, and there are thousands of specialized parts in the cell membrane called synapses. Neuronal groups which composed of many neurons connected to each other more closely, can realize some functions and accomplish more sophisticated jobs. Such kinds of groups are called columns since during doing some job they make connections in between different layers of the cortex, and this gives them morphological aggregation like if they were columns.

Normally there are about 10000 columns in the neocortex of the human brain, which is the most outer layer of the cerebral cortex [1] as shown in the Figure 1.1 [2] which describes the structure of the brain down to the ion channels in details showing the neocortex columns.

Modeling of the human brain has been a dream for a lot of scientist since long time ago. However, our current knowledge is more concentrated on classification of the brain to parts up to the level of neuron cells. This knowledge allows us to make graphics and plots that help us to understand how certain parts of brain are working. So, to improve our understanding about how brain is working we have to investigate further in how these neurons are connected to each other by studying the role of connection types, connection weights and the role of cognitive processes.

The advantages of brain modelling is: Trace the connections between individual neurons in animal models; Learn more about brain dysfunction in mental disorders and neurological disease. Such improvements can be applied and further studied by classifying into two groups [3] :

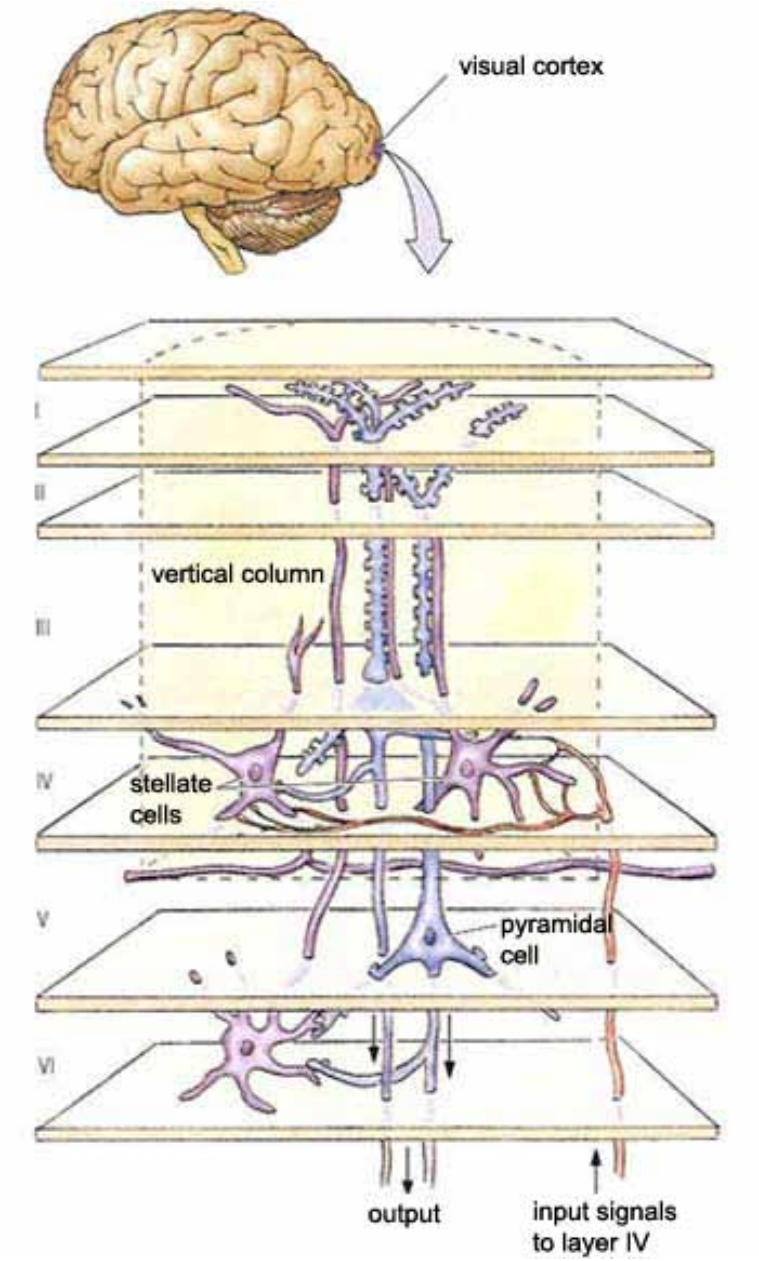

Figure 1.1: Detailed structure of brain[2].

1-To do a hard-wired connection schemes and

2-To create a structure-learning algorithms inspired by animal learning.

However, when we have a better understanding on the other parts of the brain like the neocortex this will improve our ability to make computational models and hard-wired models. Also in the future an advanced learning model can be created by mixing our knowledge from biology with machine learning and by making the comparison with our obtained graphs and plots.

Generally, classical modelling theories fall into three approaches [4] :

1- Theories that are dealing with the morphological aspects of neurons, these are also called compartment modelling, which used differential equations to describe the propagation of Action Potential (AP) in each of the compartment separately [5]. Normally the simulation programs work step by step to solve differential equation of each compartment and collect the results of all the compartments according to the original location in the cell. The models produced from this type of modelling can be very accurate from the biological point of view, but on the other hand, can be complex specially if the model contains so many compartments, some models contain up to hundreds or even thousands of compartments. Also these models normally care about modelling the neuron cells morphology without considering the dynamic behaviour.

2- Theories that are dealing with the behaviour of neurons and neural system as in NN (Neural Network). These kind of theories belongs to older times when there were no complete knowledge about biological aspects. As a result, these theories are almost talking about behaviour and nothing about neuroscience. Modern NN theories however take steps towards the neuroscience by taking some consideration of biological properties, although these steps are still considered as advantageous and are rarely being used. If we take the NN as an example of our approach, we can notice the following differences between NN and human brain:

a- There is no identification between units of NN and regions of the brain.

b- Modelling the inputs and outputs of the model is not accurate with respect to the brain.

c- The building units of NNs are not similar or simulate the equivalent ones in the regions of the brain.

d- Methods and algorithms used in the learning processes in NN do not include biological plasticity and normal learning concepts [5].

3- There are also the modern theories which lie in the middle between the first two theories called computational neuroscience. In the computational neuroscience theories, assumptions of the model are taken as similar as possible to the biological concepts so that the model can emphasize no violation to the biological restrictions and give a resemblance of biological behaviour. Based on this, it gives us a different approach to learning which differs from the traditional NN [5].

In addition to that, in computational neuroscience there are two fields or two work levels:

a- Neuron level which focus on modelling the structure and activity of the single neuron.

b- System level which focus on modelling the structure and behaviour of tissues of the brain, by connecting multiple single neuron models together and observe their behavior [6].

Computational neuroscience is a branch of neuroscience, which is becoming more and more an interdisciplinary subject. The importance of neuroscience in general comes from the fact that, according to the World Health Organization (WHO). One billion people are affected by neurological diseases and disorders which make them 11 percent of all the recorded diseases all over the world, without counting for the other mental illnesses. Also, its very high cost of treatment reach up to one trillion dollar in Europe alone according to the European Brain Council [3, 7].

By the use of neuroscience and with the implication of our knowledge in brain anatomy, physiology and brain imaging field, we can model the brain and simulate many particular tasks of it, which can improve our understanding of properties of the brain disorders and help us to develop proper solutions for them [6]. More than 100 years ago, the Italian scientist Camillo Golgi discovered a technique to stain individual brain cells. This technique had been used by other scientists to observe and study the brain cells and tissues.

In 1958, Frédéric Bremer made a study about synchrony and functional significance of brains waves.

Recently, on 2004 Eugene M. Izhikevich developed a large scale model of the brain, also simulation models for the cortical neuron spikes. His models include the implication of the most of the well-known previous works like Hodgkin–Huxley (HH), integrate and fire (IAF), and resonate and fire (RAF) [8].

There are projects as Blue Brain, where the ultimate task is to have one to one model of human cortex, by using super computers and modelling each neuron in detail [9]. So, in neuroscience the models range from detailed single neuron models to models of cortex where membrane potentials are modelled with simple models as IAF.

Based on this, we started to make the model of the single Pyramidal Neuron and then study the behaviour of groups of these neurons when connected to each other in a way similar to the real connections in the original cortical tissue.

In this thesis we made a simple model for the cerebral cortex of human and rat, we did this, considering the number and type of the composing cells available in the cerebral cortices of both human and rat, and the produced spikes also. So, our work can be classified under the neurocomputational theories, which states that in the produced model, both the behaviour and biological restrictions must be emphasized.

Reaching this point, we started to study cortex behaviour starting from the single spiking neuron for different spike types and considered the behaviour of a group of spiking neurons. Finally, we calculated the amount of synchronization occurring when multiple neuron groups are interactive with each other.

The importance of measuring the synchronization is that it indicates the degree of coherence between neuron groups synchronization interactive with each other. Synchronization have a role in the well-timing coordination and communication between different cortical neurons while cortical tasks are realized, specially in determining the amount of neurons being engaged in cognitive processes [12].

In this thesis, we are going to use Izhikevich's neuron model, since it is simple but still can model different spiking behaviour and form a network to model cortex behaviour. While focusing on cortex, we are going to model different behaviour of Pyramidal neurons and when forming the network we are going to consider the role of

inter-neurons, too. With the model we are going to investigate the role of connection, weights and spareness of connection on synchronization as it is considered to be important for forming association and it is believed to be the reason of EEG signals observed [10].

Where, the weight of connection is the factor being multiplied by the inputs being entered to neurons. While, spareness is the probability of neurons to be connected to each other.

In the second part of this thesis, a short summary on cortex is given, in third part, will be an explanation of our work, in the fourth part, a conclusion about our work will be given.

2. CORTEX

In this section, first a brief review on the cortex will be given to give an idea about the role and structure of cortex. Then different firing patterns observed in cortex will be introduced. In the sequel, these firing patterns will be obtained with a simple neuron model (Izhikevich's neuron model) used in simulations with different parameters and while constructing the network these firing patterns will be considered and two of these patterns will be simulated to investigate the synchronization in the network.

2.1 The Structure of Cortex

The mammalian brain consist of gray and white matter as in the Figure 2.1 [11].

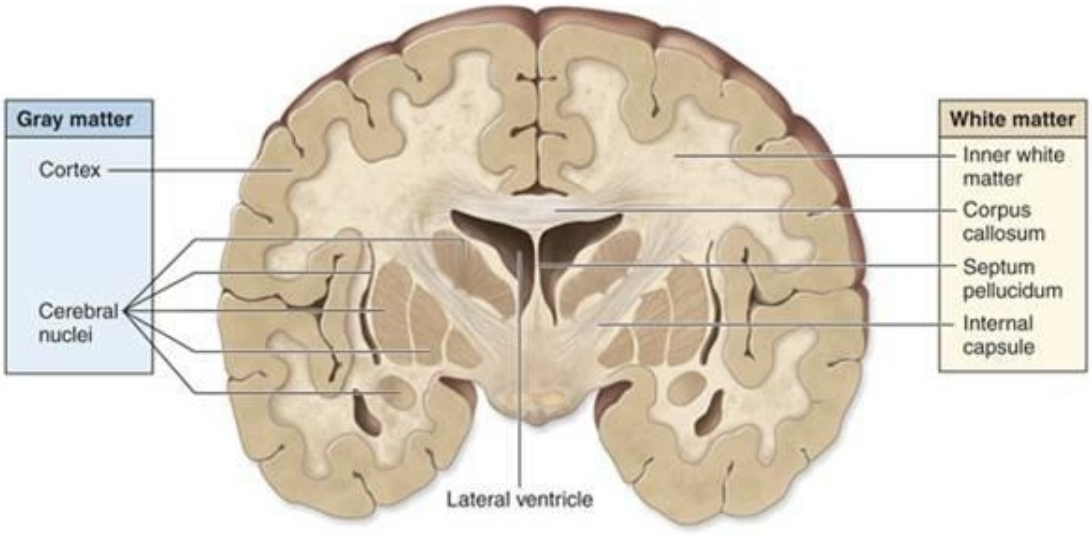

Figure 2.1: A coronal section of the cerebral cortex with White and Grey matters [11].

Where the grey matter is consists of the body of the neuron cells (soma), the white matter consists mainly of the axons and dendrites of the neuron cell, as in the Figure 2.2 [12].

The cerebral cortex is the outer covering of grey matter over the hemispheres. Its a sheet of neural tissue of about 2-3 mm of thick covering the gyri and sulci [13]. It covers the cerebrum and cerebellum, and is divided into left and right

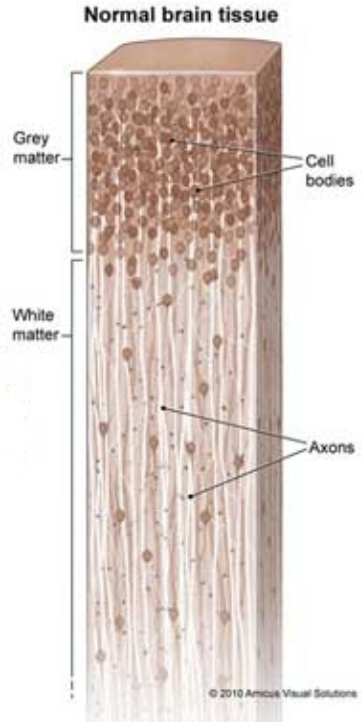

Figure 2.2: An enlarged section of the cerebral cortex shows the white and grey matter [12].

hemispheres [13]. Generally the cortex can be subdivided according to anatomically and functionally point of views. Starting from the anatomical point of view, the cortex can be subdivided to neocortex and allocortex as in Figure 2.3.

The allocortex parts of the cortex are more primitive compared to the neocortex parts, and are located in the medial temporal lob of the brain. Allocortex it self has two components, the Paleocortex and the Archicortex. The Archicortex consists of the hippocampus, which is a three-layered cortex dealing with encoding declarative memory and spatial functions. On the other hand, the neocortex consist the great majority of the cortex [13].

The importance of the cerebral cortex is that it plays a key role in our life, since it is the most developed part of the brain. And it does a lot of functions in interpretion of sensing, such as hearing, seeing and touching. Also it is responsible of cognitive functions like coding and decoding the information going and coming from the memory, attention, perceptual awareness, thought, understanding languages, and consciousness [14]. The human cortex consists of up to six horizontal layers

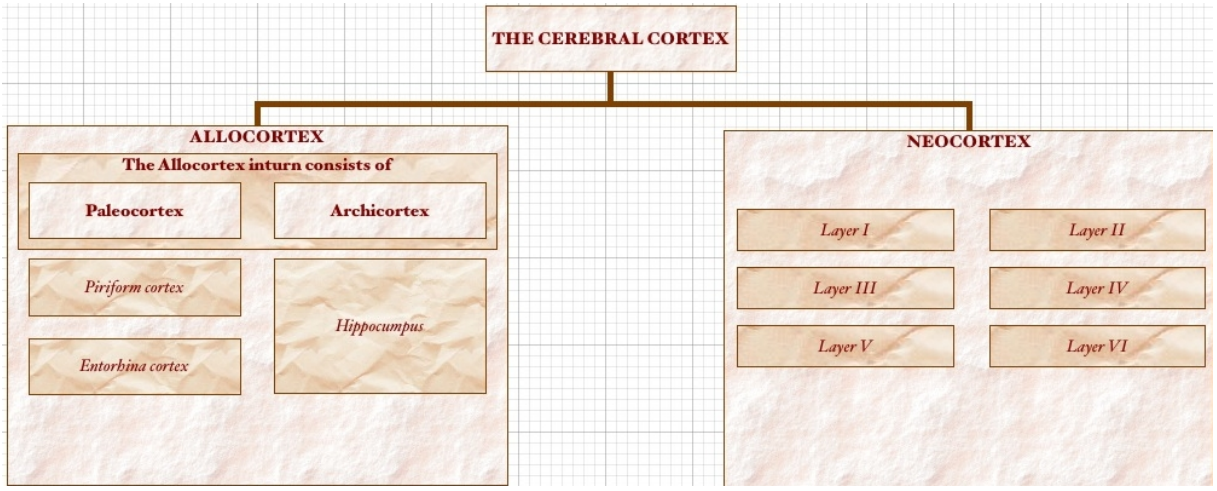

Figure 2.3: A tree diagram showing the anatomical structure of the cerebral cortex in the human brain.

as in Figure 2.4 [2], each with a different composition in terms of neurons and connectivity. Neurons in various layers connect vertically to form small microcircuits, called columns. Different neocortical architectonic fields are distinguished upon variations in the thickness of these layers, their predominant cell type and other factors such as electrochemical markers [14]. The neurons of the cerebral cortex are grouped into six main layers, from outside (pial surface) to inside (white matter):

Layer I, the molecular layer, contains few scattered neurons and consists mainly of extensions of apical dendritic tufts of Pyramidal neurons and horizontally oriented axons, as well as glial cells. Some Cajal- Retzius and spiny Stellate cells can be found here. Inputs to the apical tufts are thought to be crucial for the "feedback" interactions in the cerebral cortex involved in associative learning and attention. While it was once thought that the input to layer I came from the cortex itself, it is now realized that layer I across the cerebral cortex mantle receives substantial input from "matrix" or M-type thalamus cells (in contrast to "core" or C-type that connect to layer IV).

Layer II, the external granular layer, contains small Pyramidal neurons and numerous Stellate neurons.

Layer III, the external Pyramidal layer, contains predominantly small and medium-size Pyramidal neurons, as well as non-Pyramidal neurons with vertically oriented intra-cortical axons; layers I through III are the main target of inter-hemispheric

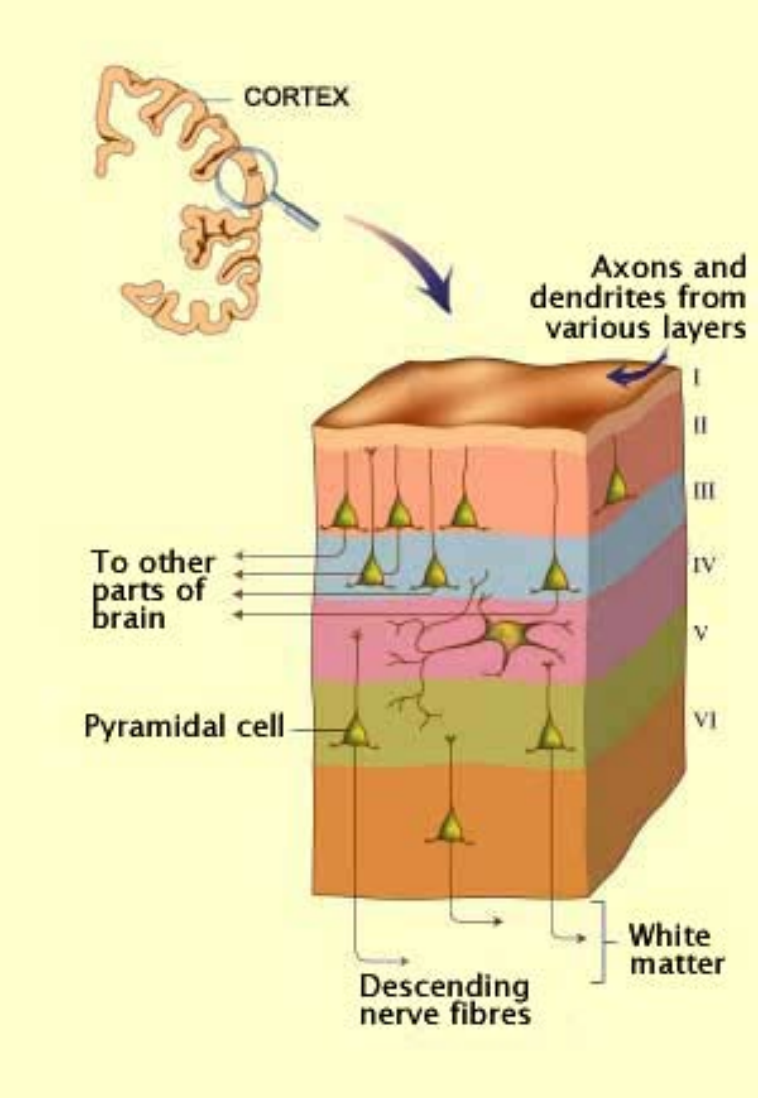

Figure 2.4: The horizontal layers of the cerebral cortex [2].

cortico- cortical afferents, and layer III is the principal source of cortico-cortical afferents.

Layer IV, the internal granular layer, contains different types of Stellate and Pyramidal neurons, and is the main target of thalamo-cortical afferents from thalamus type C neurons, as well as intra-hemispheric cortico-cortical afferents.

Layer V, the internal Pyramidal layer, contains large Pyramidal neurons (such as the Betz cells in the primary motor cortex). It is the principal source of sub cortical afferents, as such, there are large Pyramidal cells which give rise to axons leaving the cortex and running down through the basal ganglia, the brain stem and the spinal cord.

Layer VI, the Polymorphic or Multiform layer, contains few large Pyramidal neurons and many small spindle-like Pyramidal and multiform neurons; layer VI sends efferent fibers to the thalamus, establishing a very precise reciprocal interconnection between the cortex and the thalamus. These connections are both excitatory and inhibitory [13].

On the other hand, from the functional point of view, the cortex can be subdivided into primary cortices which are the regions in which the simple functions are done like receiving sensory inputs (vision, hearing, somatic sensory) for the sensory cortex, controlling the eye and limbs movements for the motor cortex as in the Figure 2.5 [15]. The other parts are the associated cortices which can do more complex functions. Regions of association cortex are adjacent to the primary cortices and include much of the rostral part of the frontal lobes and also regions encompassing areas of the posterior parietal lobe, the temporal lobe and the anterior part of the occipital lobes. These areas are important in more complex cortical functions including memory, language, abstraction, creativity, judgement, emotion and attention. They are also involved in the synthesis of movements [13].

Different parts of the cortex have different jobs, for example all the information coming to and out the neocortex to the sub-cortical regions are being transmitted through the thalamus, the neocortex communicates normally with up to 20 different regions of the brain. Other parts like primary motor cortex are responsible for sending signals that controls movements of skeletal and visceral muscles, either directly through the connections to the spinal cord or indirectly by passing through the cerebellum first. There are also visual cortex and auditory cortex and the language area as illustrated in Figure 2.5. It has been experimentally recognized that the neurons in the neocortex are forming some type of vertical columns extend from the outer shield down to the deeper part of the cortex. Neurons in these columns seems more dedicated to do more complicated functions than single neurons. The other facility of the brain is that columns are not fixed in size or in number of lateral connections. And it seems that these connections are being controlled by signals from the outside of the cortex itself. The evidence of this idea come from the study of the visual cortex, when noticed that the number of neurons in these columns are more than the number of the neuron cells in the visual cortex. Which leads us to the idea that the neurons made connections such that they anatomically group, but however its dynamical groups can change and

re-change there connections and this connection forming is important as it implies the response of cortex to input signal. In our model we substitute the change of the strength of connections in terms of different weights and sparseness of connections. This shows that the brain has no specialized psycho-physio jobs, that means the brain has no specific regions for each specific job, rather it has a highly distributed functionally facility. This means the ability to highly distribute a specific function over a wide are of the surface of the cortex. This property of neocortex called Dynamic Link Architecture (DLA) [16]. It has an importance in the improvement of Neural Network in the way of representing object composition as a coordinate correlation of many individual.

These cell groups have a good affect on the performance of the cortex, that means these groups do more sophisticated and dedicated functions which can not be done by single neurons. And to have such a connected activity, these neurons have to work together in synchrony, in our work we calculated synchronization measure to show the role of connection dynamics on synchronization. Synchrony is used to detect the coordinate activity between different parts of of the brain, just like the coordination when someone wants to do something that needs the cooperation of different parts of his body. It is has strongly thought that the cortex plays a role which leads these different parts of the brain to work in coordination by prompting them through excitatory spikes, which will appears eventually in the way of synchrony in their spiking [17].

Recently, with spiking neural networks a new approach which focuses on brain activation has been proposed for computational applications [4]. Also, it has been proposed that the computational mechanisms taking part in the brain especially for working memory, learning and attention emerge from the synchronization of spiking neurons [13]. Synchronization in neuronal activity had drawn attention since the observation of EEG rhythms. Their relation with cognitive processes became more and more active field of research [9]. While synchrony denotes the emergence of attention in association cortex, it is sign of disease in striatum. In this work, the effect of connection weights on synchrony is investigated as recent work on spiking neurons are promising for the design of intelligent systems [4]. It is claimed that the relation between synchrony and the neuronal code aroused from the activation of a group of neurons will inspire new learning techniques [1].

Neuronal synchronization can be defined as a correlated appearance in time of two or more events associated with various aspects of neuronal activity. Neuronal synchronization depends on chemical and electrical synaptic interaction [18], with synchronization we can:

1- Specify the area of the brain that are working together

2- The ability of prediction of the long term behavior of the neuron groups.

3- It gives us an indication of the mental disorders and diseases.

4- The strength of synchronization is functionally related to perceptual accuracy and behavioral efficiency [19].

5- Synchronization also help to keep the signals between interconnected neurons from the intrinsic noise (the inter-neuronal noise), which allow a reliable communication and signal transition even in a noisy environment [20].

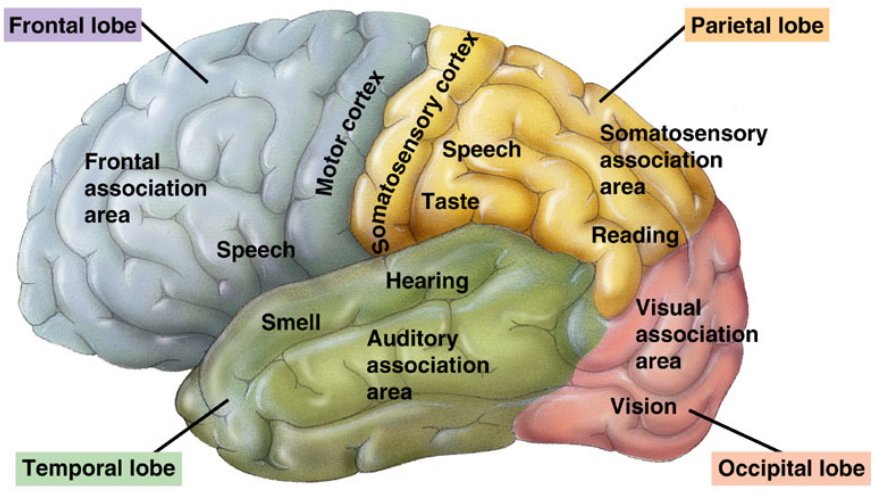

Figure 2.5: Cerebral cortex with the related sensory areas shown in different colors [15].

There are different lobes in the cerebral cortex which are responsible for different things. These are:

• The parietal lobe – this is involved in the reception and processing of sensory information from the body such as pain and touch sensation and visual perception, it is also involved in spatial orientation, speech, cognition, and information processing

• The frontal lobe – this is involved in decision making, problem solving and planning as well as reasoning, judgment and impulse control

• The occipital lobe – this is involved in vision and color recognition

• The temporal lobe – this is involved in memory, emotion, hearing, and language [21].

Basically speaking, the cerebral cortex is vital for sensing and interpreting input from various parts of the body and also for maintaining cognitive function. Sensory functions of the cerebral cortex include hearing, touch, and vision. Cognitive functions include thinking, perceiving, and understanding language. In this way, it can be said that the cerebral cortex determines intelligence. It is also responsible for planning and organization. The cerebral cortex also determines a person's personality [1].

From the constructional point of view, the cortex is constructed from two parts, the newest and biggest part is the neocortex, and the allocortex, which is located in the medial part of the temporal lobe and is responsible for olfaction and survival actions such as visceral and emotional reactions. The neocortex is the newest and biggest part and consist of six layers, functionally the neocotex layers can be devided as following: supergranual, which includes the layers I-III, highly developed and responsible for the intracortical connections, which is either between parts of the cortex of between parts that are on opposite hemisphere. The infragranular layer and layer IV are responsible of the connections with hypothalamus, sub-thalamus and subcortical parts. This is prominent specially in the primary sensory parts.

Motor Cortex is essential for mental functions that are more complex than detecting basic dimensions of sensory stimulation, for which primary sensory areas appear to be necessary. In humans the association areas are by far the most developed part of the cerebral cortex, and the brain in general. These areas are necessary for perceptual activities, like recognizing objects (toasters, horses, trees, words, etc), rather than simple contours, edges or sensory qualities like color or pitch. Each sensory system for example (vision, hearing, etc.)has its own association areas on the cerebral cortex. They have their own primary area on the cortex, which gets the most direct connections from its sense. Each primary sensory area sends information to its own cortical association areas, which are next to their primary areas, as shown in Figure 2.6 [22].

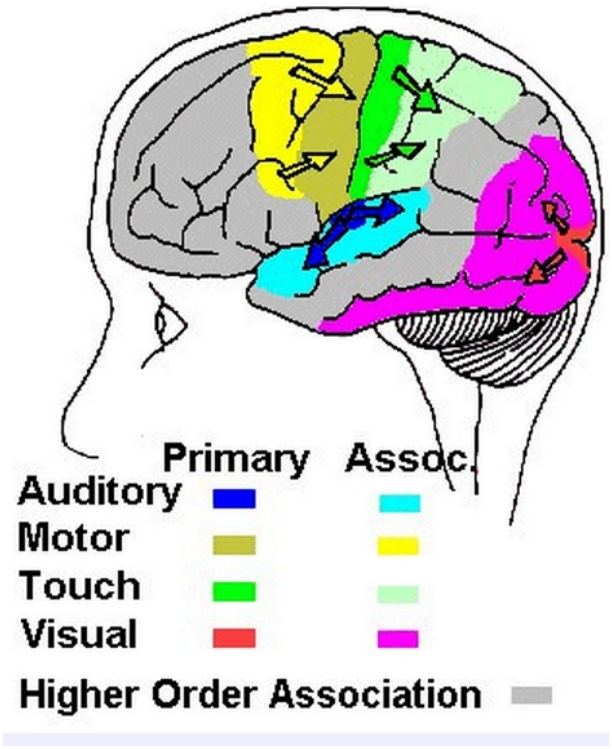

Figure 2.6: Cerebral cortex shows the association of primary areas to their related part in the association cortex [22].

The role of association cortex can be noticed in the sensory associated area during the done of complex mental processes. The sensory system in the brain is composed of two parts, the primary part and the sensory association part.

The primary part area located under its related associated part and is responsible mainly for processing the primary information necessary for perception of objects and events like contours, boundaries, and colors. On the other hand, sensory association areas combine this kind of information to represent complex objects.

The higher order association cortex combine information sent as signals from all the primary information area as shown in the Figure 2.6 [22].

2.2 The Action Potential (AP)

The Action Potential (AP) is a chemo-electro phenomena, occurs as a result of ion transfer between the inside and outside of the neuron cell across the voltage gated channels [23]. AP is consider as the fundamental mean of communication in the nervous system [24]. The AP consist of three parts as shown in the Figure 2.7 [25].

1- Sharp increase in the membrane potential, depolarization.

2- Less sharp decrease towards the resting potential, repolarization.

3- Afterhyperpolarisation phase, here the potential fails below resting potential before it returns eventually to resting potential.

HH used voltage clamp step by step to produce experimental data and use them to produce their model. In the below, there is a summery of steps of how HH did their experiment:

1-They used the voltage clamp in the intracellular space to make records to know how is the current is related to the voltage, and how much current is curried by Na ions and by other ions as well.

2- Then they fitted these results to a mathematical model, the other part is based on the ion selective voltage dependent gates. The remaining of the model were fitting curves. The final model is expressed in terms of the mathematical equations and called HH model.

3- They solved the equations of their model under different conditions and found that all the results were similar to that being measured [26].

However, HH model has some limitations when used for many ion currents but HH formalism is a useful and popular technique for modeling channel types action potential as shown in the following equations [27]:

$$C\dot{V} = I - \bar{g}_K n^4 (V - E_K) - \bar{g}_{Na} m^3 h (V - E_N a) - \bar{g}_L (V - E_L) \tag{2.1}$$

since the membrane is considered as a capacitance.

$$\dot{n} = \alpha_n(V)(1-n) - \beta_n(V)n \tag{2.2}$$

$$\dot{m} = \alpha_m(V)(1-m) - \beta_m(V)m \tag{2.3}$$

$$\dot{h} = \alpha_h(V)(1-h) - \beta_h(V)h \tag{2.4}$$

Where n is the probability of individual channel to be open, $\dot{n}$ is the rate law or first order differential equation determines how does variable n changes with time. The

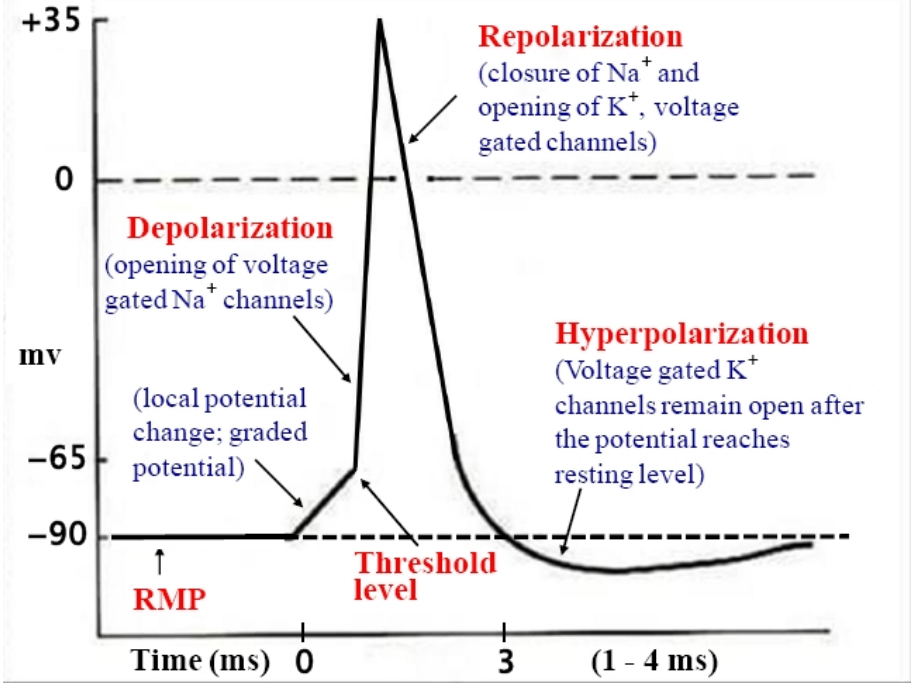

Figure 2.7: A plotted AP pulse, shows the three consisting regions[25].

same thing for variables m and $\dot{m}$, while h represents the level of inactivation [26]. where,

$$\alpha_n(V) = 0.01\frac{10-V}{exp(\frac{10-V}{10})-1}, \tag{2.5}$$

$$\beta_n(V) = 0.125exp(\frac{-V}{80}), \tag{2.6}$$

$$\alpha_m(V) = 0.1\frac{25-V}{exp(\frac{25-V}{10})-1}, \tag{2.7}$$

$$\beta_m(V) = 4exp(\frac{-V}{18}), \tag{2.8}$$

$$\alpha_h(V) = 0.07exp(\frac{-V}{20}), \tag{2.9}$$

$$\beta_h(V) = \frac{1}{exp(\frac{30-V}{10})+1} \tag{2.10}$$

Equation 2.1 consist of these parts of current equations as following:

$$I_K = \bar{g}_K n^4 (V - E_K) \tag{2.11}$$

Table 2.1: Nerst equation values.

E_K mV	E_{Na} mV	E_L mV
-12	120	10.6
$\bar{g}_K\ mS/cm^2$	$\bar{g}_{Na}\ mS/cm^2$	$\bar{g}_L\ mS/cm^2$
36	120	0.3

$$I_{Na} = \bar{g}_{Na} m^3 h (V - E_N a) \tag{2.12}$$

$$I_L = \bar{g}_L (V - E_L) \tag{2.13}$$

where C is the membrane capacitance, I is the total ionic current current, $\bar{g}$ indicates that it is constant, while the others depends on the recent history of the membrane potential (voltage dependent) and is equals to:

$$g_{Na} = \bar{g}_{Na} m^3 h \tag{2.14}$$

$$g_K = \bar{g}_K n^4, \tag{2.15}$$

I_C is the capacitive current which came from the fact that, when the in-ionic current enters and exit from the membrane, it in fact charges its voltage, so the membrane voltage is current dependent as shown in this equation, with I_{Na}, I_k and I_L as the leakage current [26]. The permeability of the ions $\bar{g}$ is voltage dependent, so these gates are active not passive, where α and β, are rate coefficients which depend of membrane potential, they represent the opening and closing reaction rates respectively.

$$C \rightleftharpoons^{\alpha_n}_{\beta_n} O \tag{2.16}$$

where I_C here refers to Close state, and O refers to Open state [26]. In order to reach Nerst equilibrium, which states the rest potential where no ions transfer occur between the inside and outside of the cell membrane, the following values must used in HH equation 2.1 as shown in Table 2.1 [27].

Izhikevich model on the other hand, seems to be more logically and we chose to use this model in our simulation since it is,

• Easer to be programmed and computed.

• More realistic since it can creates more spiking types similar to that presented by biological cells.

$$v' = 0.04v^2 + 5v + 140 - u + I \tag{2.17}$$

$$u' = a(bv - u) \tag{2.18}$$

With reset conditions set as follows:

$$if\, v \geq 30mV, then, v \leftarrow c, u \leftarrow u + d \tag{2.19}$$

Here v is membrane voltage, we get it by solving of the differential equation 2.17. While I represents the injected DC-current.

u is the time-recovery variable, which is the time required to the membrane voltage to return to its original value after one pulse, which represents the activation of K+ ionic currents and inactivation of Na+ ionic currents. We can get u by solving the differential equation 2.18.

Other parameters, a, b, c and d are dimensionless parameters, they are important in the configuration of the pulse shape, different pulses can be obtained by changing these parameters [8], were parameter a describes the time scale of the recovery variable u, parameter b describes the sensitivity of the recovery variable u to the sub-threshold fluctuations of the membrane potential v, parameter c describes the after-spike reset value of the membrane potential v caused by the fast high-threshold K+ conductances, parameter d describes after-spike reset of the recovery variable u caused by slow high-threshold Na+ and K+ conductances, as shown in Figure 2.8 [28].

We implied the parameters given in Table 2.2.

When the spike reaches (+30 mV), the membrane voltage v and the recovery variable u are reset according to the 2.19. The resting potential in the model is between -70 and -60 mV depending on the value of b

In the next sections we will show how we modeled these APs for Pyramidal neurons starting from the available simulators in section 2.3 , section 2.4 the models of single spike neuron APs of Pyramidal neurons we be explained.

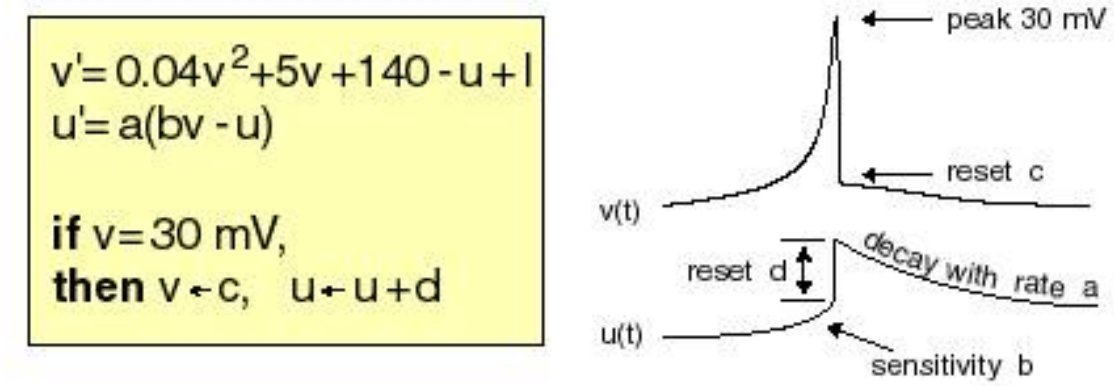

Figure 2.8: A plot showing the effects of different parameters of Izhikevich model parameters [28].

2.3 The Simulators Developed for Modeling in Neuroscience

Simulation means substitution of experimentation with the real system for analysis of arbitrary system behavior. Simulation is needed when the real system does not exist and it is costly or impossible to build and experiment with prototypes , or when other modeling methods are impossible.

In order to be sure that expected accuracy of simulation is consistent with needed requirements and get the right results we need first to formulate of the problem, identifying the type of desired model , also the questions to be answered and hypothesis to be tested.

Simulation process have many advantages, simulation generates data by using existing preliminary data decreasing need to collect other data, also can build system model in modules, run independently and join together, like we did in this thesis, we used data of single spike neurons and rebuilt them in neuron groups using Brian simulator to study there behavior [29].

The following list are the most used neuron simulation programs:

1-BRIAN Brian is a simulator for spiking neural networks available on almost all platforms. The motivation for this project (BRIAN) is that a simulator should not only save the time of processors, easy to learn and use, highly flexible and easily extensible. The Brian package itself and simulations using it are all written in the Python programming language [30].

2-NEURON: For empirically-based simulations of neurons and networks of neurons [31].

3-GENESIS: Is a simulation environment for constructing realistic models of neurobiological systems at many levels of scale including sub-cellular processes, individual neurons, networks of neurons, and neuronal systems [32].

4-NEST: Is a simulator for spiking neural network models that focus on the dynamics, size and structure of neural systems rather than on the exact morphology of individual neurons [33].

5-SPLIT 23: Is a tool specialized for efficiently simulating large-scale multi-compartmental models based on HH formalism.

6-Mvaspike: Mvaspike is a general purpose tool aimed at modeling and simulating large, complex networks of biological neural networks [34].

We choose to work on BRIAN simulation program which has been written in Python for the chase of ease and to save time, Brian is easy to learn and use, highly flexible and easily extensible. The Brian package itself and simulations using it are all written in the Python programming language [35].

2.4 The Firing Patterns In Cortex

The main identically neuron cells are the Pyramidal cell, specially in the layers III and V. Pyramidal neurons, also known as Pyramidal cells, are neurons with a Pyramidal shaped cell body (soma) and two distinct dendritic trees.

The basal dendrites emerge from the base and the apical dendrites from the apex of the Pyramidal cell body. The dendrites is considered as the input part of the Pyramidal cell and receives its stimulus from the synapses with the other cells, while the axon is the output and connect the cell to the other cells. Pyramidal neurons have been observed in birds, fish, reptiles, and all mammals studied. They are found in forebrain structures such as the cerebral cortex, hippocampus, and amygdala, but not in the olfactory bulbs, striatum, midbrain, hindbrain, or spinal cord. They are the most numerous excitatory cell type in mammalian cortical structures, suggesting that they play important roles in advanced cognitive functions.

The Pyramidal cells have two properties:

1. have a retrograde signaling molecules.

2. have extensive branching in both dendrites and axons, which gives the property that one neuron can connect with thousands of other neurons in a network.

The firing patterns in the cortex of the mammalians can be classified into six classes, although most of the biologists agree with this classification, some of them think it's oversimplified and the each class can be further subdivided into more subclasses, and that each neuron can change its firing pattern according to the state of the brain [13,14].

We took all these the parameter values of the spikes types from Izhikevich model of cortex and did them in BRAIN Simulator according to the values shown in Table 2.2, where Izhikevich did them in MATLAB [8].

We draw the obtained results as shown in the next figures from Figure 2.9 to Figure 2.13.

Table 2.2: Single spike values.

Spiking Type	a /msec	b /msec	I mA	c mV	D V/sec
RS: regular spiking	0.02	0.2	10	-65	8
IB: intrinsically bursting	0.02	0.2	10	-55	4
CH: Chattering	0.02	0.2	10	-50	2
FS: fast spiking	0.1	0.2	10	-50	2
LST: low-threshold spiking	0.1	0.25	10	-50	2

RS: regular spiking: The neuron fires a tonic pulses with decreasing frequency, in response to injected pulse of DC current. These types of neurons could be found in the 2, 3, 5 and 6 layer in the cortex and called the Spiny Stellate cells.

IB: intrinsically bursting: This kind of spiking found in the layer 5, and also distributed in all the other layers of the cortex. In this type of spiking, a burst is start at the beginning of a strong depolarization pulse of current, then turn to a tonic spiking mode.

CH: Chattering Neurons fires another type of high- frequency bursts of spikes with short inter bursts period. These types of neurons could be found in the visual part of the cortex of adult cats, morphologically they are spiny Stellate or Pyramidal neurons located in the layers 2-4, and mainly in layer 3.

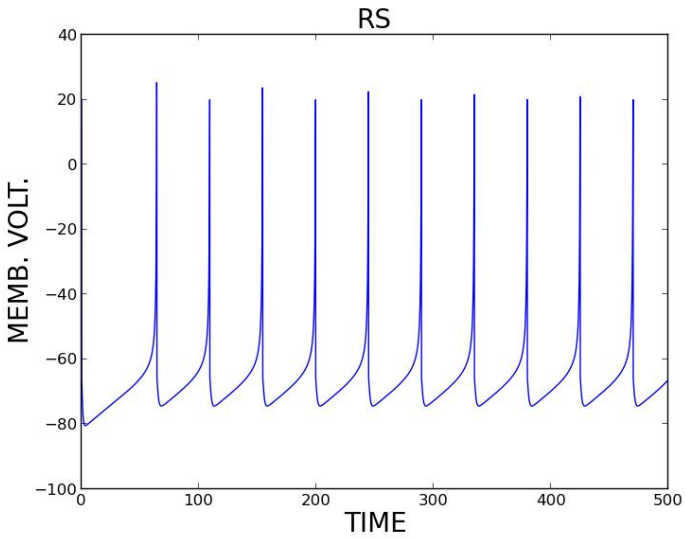

Figure 2.9: Regular spiking.

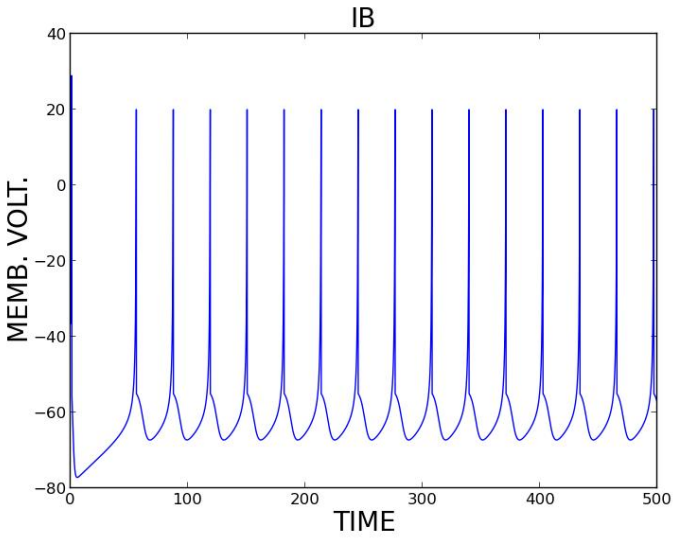

Figure 2.10: Intrinsically bursting.

FS: fast spiking: The neurons firing such kind of spikes are normally sparsely spiny and a spiny non Pyramidal. And fires a high-frequency tonic spikes with constant period. But when the injection DC current fails below some threshold value, the spiking types become an irregular tonic spikes switching randomly between firing and resting.

LST: low-threshold spiking: In this kind of spikes, neurons fire tonic spikes with a well-defined frequency and rebound (post-inhibitory) spikes. They also have the ability of firing a low-frequency spike trains, by unknown excitability class. These neurons are located in the vertical inter-laminar direction of the neocortex, and its non-Pyramidal inter-neurons.

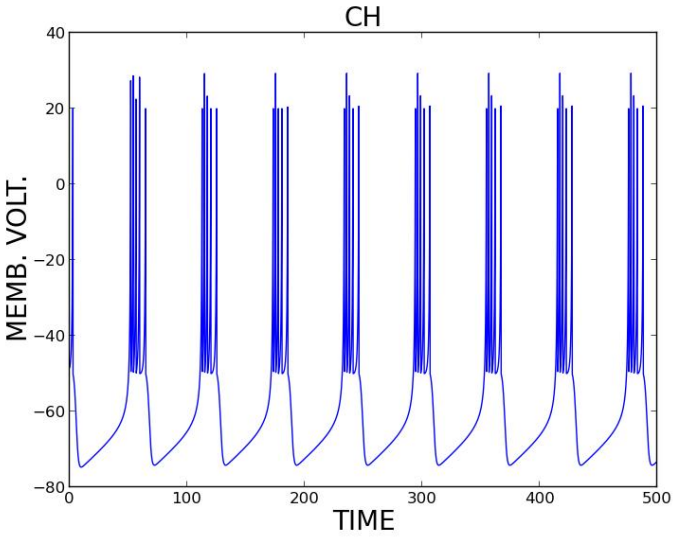

Figure 2.11: Chattering spiking.

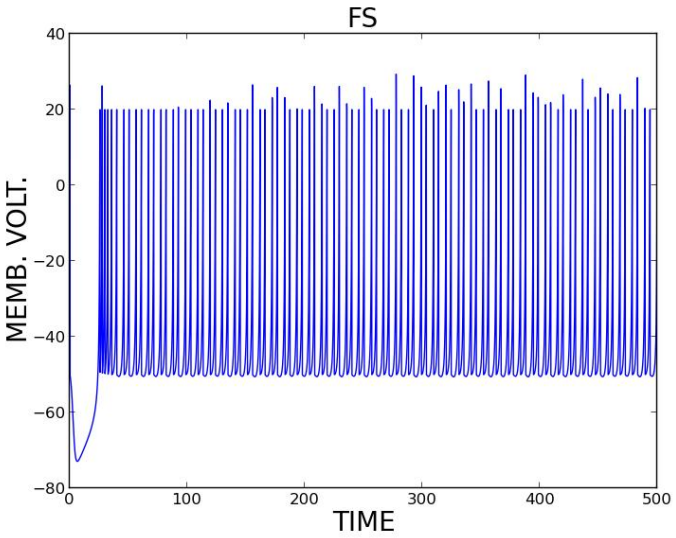

Figure 2.12: Fast spiking.

LS: late spiking: The neurons exhibits such a kind of spiking are the non-Pyramidal neurons, which located mainly in layer(1), this kind of spiking is significant in that, it happens as a ramp response to the injected DC current have a value near the rheobase, which result in a delayed spike as much as 1 sec. Also the voltage shows sub-threshold oscillation during the ramp [1]. While forming the network, from these different patterns of firing, three of them are considered: regular spiking, fast spiking and chattering.

In the next section we will make the network of these single neurons using Brian simulator program and for both human and rat cortex. We made two groups, one

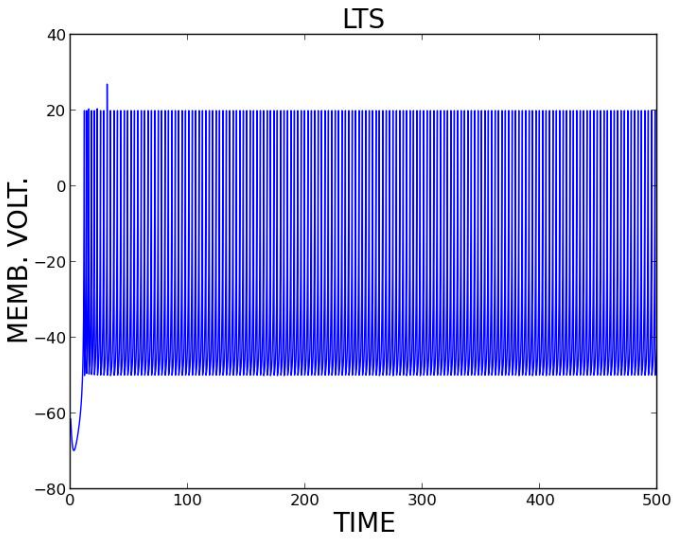

Figure 2.13: Low-threshold spiking.

represents excitatory action and simulates the Pyramidal neurons in the cortex, the other represents the inhibitory action and simulates the Stellate cells in the cortex.

We made this model using RS and CH single spikes seperately to make two excitatory groups, and FS to make the inhibitory group in both of the models. Since the RS, CH and FS spiking types are the ones recorded in the cortex as excitatory and inhibitory respectively [1].

3. MODELLING CORTEX BEHAVIOUR

The human cortex have six layers, distinguished from each other morphologically, and consist of two main types of neurons, Pyramidal and Stellate type. While the Pyramidal neurons plays as excitatory neurons, the Stellate neurons plays as inhibitory neurons [27]. In this section we will talk about how we created the network composed of Pyramidal and Stellate type of neurons to model cortex and how we measured synchronization due to activation between the neurons.

3.1 Modelling Cortex Behaviour

In the previous section, we explained how we obtained five different behaviour of Pyramidal and Stellate type neurons using Izhikevich neuron model, these five different type of spiking neurons are most seen in the cerebral cortex [8]. In this chapter we will explain how we created a model to simulate the spiking neuron network behaviour of the cortex neurons as a tissue. This model contains the same neuron types (characterized by its spike type) with the same percentage of presence and with the same type of connections. The connection probability is random [10]. While we were forming the network we formed two groups one of them is composed of excitatory firing neurons represented by RS spiking type or CH spiking type, while the other group is inhibitory firing neurons represented by FS spiking type in all simulation. We based our choices of neuron model on ‘ model which suggested that cortex spiking can be classified into two main groups, excitatory and inhibitory [8]. We make two models to simulate the human cortex and two models to simulate the rat cortex.

In our connections we used BRAIN connection codes to make a network of neurons as shown in Figure 3.1, and this is done by simulating Equation 3.1.

$$dg/dt = -g/\tau \tag{3.1}$$

Where g is conductivity variable, it represents the change in the conduction between two neurons endings due to the effect of interaction of neurotransmitter with and

opening postsynaptic membrane of the targeted neuron (the dendrites of the targeted neuron) [26]. The value of τ is a time constant, it differs from type to type of spiking and where the considered neuron in [36]. In this network we ensured that each single neuron has the ability to connect to all the other neurons whether they are in its group on in the other groups, but still the connections are sparse since the occurrence of the connections will be controlled by the sparseness variables, which is the situation in the brain cortex [10]. This modeling strategy ensures the happening of (DLA) phenomena which had been mentioned in Section 2 [16]. These connections are controlled by changeable connection weights (one weight for each group). The connections between groups and in the groups are determined randomly and the connectivity probability is taken as a variable to see the role of it on overall behaviour of cortex dynamics. We noticed that when we increase the weight of connection between neurons, the synchronization also increases to some extend, in a nonlinear relationship.

In order to get the DLA phenomena we created four connections (two for each group) in each simulation, each one of them is connecting the neuron with its following neurons (in the same group) and the other is connecting the neuron to neurons of the other group. The difference between the human cortex model and the rat cortex model is in the ratio of neuron type in the excitatory and inhibitory groups. While modeling the human cortex inhibitory neuron number taken as 800, and the excitatory neuron number taken as 200, in the rat cortex it is vice versa. These numbers are determined following the ratio of inhibitory and excitatory neurons in rat and human cortex as mentioned in [37] and [38], respectively.

The following codes are BRIAN codes for connection.

```
Conneci=Connection(Pe,Pi,'gi',structure='sparse'
,sparseness=s, weight=w1)
Connece=Connection(Pi,Pe,'ge',structure='sparse'
,sparseness=s, weight=w2)
Connecee=Connection(Pe,Pe,'ge',structure='sparse'
,sparseness=s, weight=w2)
Connecii=Connection(Pi,Pi,'gi',structure='sparse'
,sparseness=s, weight=w1)
```

Where *Pe* and *Pi* represent excitatory and inhibitory groups respectively, 'weight' refers to the connection weight, where the weight of connection is the factor ffecting the value of g. Whenever a spike is formed the value of g is increased by the weight factor. So weight effects the term I in Eq 2.17. 'Source' and 'target' stands for the start and targeted networks respective. 'Structure' refers to the type of connection weather its random, dense, dynamic. We chose random or 'sparse' type of connection, since this is the type of connection noticed in the cerebral cortex [10].

The importance of connection weights is that it is related to the brain plasticity which controls the brains capability to learn and form new relations as suggested by new studies [39].

In our codes, we implied the values of weights in a loop starting from 20 mV up to 120 mV, since lower than 20 mV the connection will be so weak and no synchronization will happen, and higher that 120 mV the amount of measured synchronization tends to be fix about 0.7-0.8.

Sparseness is the probability of establishing a connection between two neurons from different groups, increasing the sparseness will also increase the connection which in turn will increase the synchronization between the neuron groups. We changed the sparseness values from 0.01-0.3 to study its effect. The important values are 0.1-0.2, since these are the values appears in biological brain cortex [10]. In order to ensure that the results are accurate we made two loops, internal loop to change the sparseness and external loop to change the weight values. In this way each connection weight value will be examined with all the changeable sparseness values and vice versa.

So, with the model we created, we investigated thoroughly the effect of the weights and sparseness of connections on synchronization.

Synchronization is a correlated appearance in time of two or more events associated with various aspects of neuronal activity. Neuronal synchronization depends on chemical and electrical synaptic interaction [18]. We investigated the synchronization by looking at its variance as following [40]:

$$V(t) = \frac{1}{N}\sum_{i=1}^{N} V_i(t) \qquad \textbf{(3.2)}$$

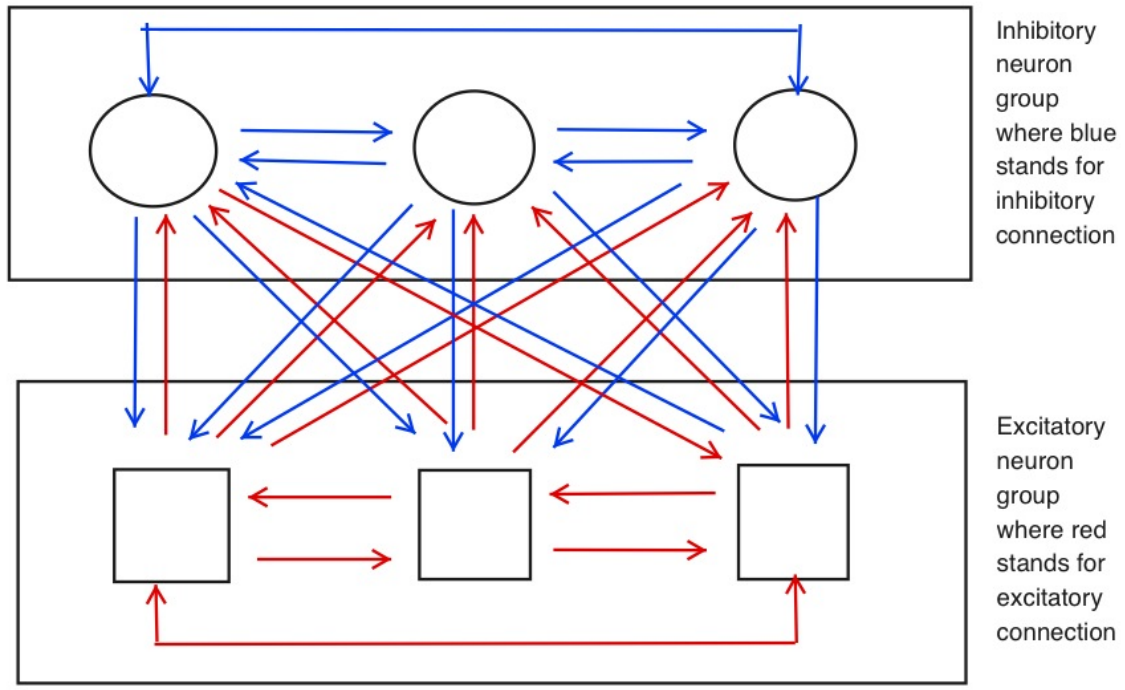

Figure 3.1: Demonstration of the connections between groups in the model. The arrow stands for the excitatory connections and line stands for inhibitory connections.

In equation 3.2 we have normalized the calculated membrane voltage of all neurons. This will be done by using two loops since we have two groups of neurons.

$$\sigma_V^2 = \langle [V(t)]^2 \rangle_t - [\langle V(t) \rangle_t]^2 \tag{3.3}$$

Equation 3.3 is for the calculation of the variance of normalized values of all recorded voltages for each neuron. First the summation of all voltage in time series for each neuron was calculated, while in the first term the square of each voltage has been taken before the summing all the terms. In the second term the voltage values had been summed first and then it has been squared. After making both of the two terms as matrices, the final result where taken by making a loop and subtracting each corresponding values from each other.

$$\sigma_{V_i}^2 = \langle [V_i(t)]^2 \rangle_t - [\langle V_i(t) \rangle_t]^2 \tag{3.4}$$

After getting all the required values, now we submitted them in the next equation which is for calculating the synchronization factor X(N).

$$\mathscr{X}^2(N) = \frac{\sigma_V^2}{\frac{1}{N}\sum_{i=1}^{N} \sigma_{V_i}^2} \tag{3.5}$$

The last equation 3.5 is to measure the synchronization of a neuron group we need to establish a specific normalized indicator between 0-1, such that 0 indicates no synchrony while 1 indicates a full synchrony.

The synchronization affected by other factors like type of spiking. During simulation we noticed that RS and FS types of spiking shows higher values of synchronization compared to other types of spiking with the same values of weight and sparseness.

Obviously in large scale networks, the synchrony needs a high number of connections between neurons and to overtake some threshold value [10], which is controlled by the changing of the weights of connection and the sparseness.

3.2 Simulation Results

In order to work on BRIAN, Python has to be installed and other programs that give support the mathematical and matrix side, like Scipy, Numpy and Pylab. After installing Python, Scipy, Numpy, Pylab, Sympy and finally BRIAN and doing the required tests to ensure that BRIAN has engaged correctly to all of these programs.

The first steps of the program are the setting of the required running duration time and running times. The running duration which represent the simulated time of interactivity between the two groups in the real tissue, and is limited by the ability and the properties of the computer.

The running times, represents the number of times the entire calculations will be repeated, and its related to the number of required changes to be done.

Here we are investigating the change in the weight of connections between both groups, including the internal and external connections, i.e. the connections between the two groups and the connections inside each groups itself.

The next steps are to define the matrices that will be used in the program, this step is required in Numpy, which is used by Python in the calculations of matrices.

Our work falls into two categories, the first one is building the single spike neuron model as has been mentioned in section 2, we did this by building the required neuron model, calculating of the single neuron membrane voltage. This is realized as in Figure 3.2.

Algorithm 1: calculate and draw the membrane voltage of the single neuron using Izihekevich model.

information: these codes written in BRIAN simulator which is a special library of Python Programming Language.

ensure: in order to get the required results properly, the following libraries should be imported:
NumPy and Scipy packages for Python: an efficient scientific library.
PyLab package for Python: a plotting library similar to Matlab.
SymPy package for Python: a library for symbolic mathematics (not mandatory yet for Brian)

ensure: set the duration of running.
ensure: the parameters required to solve the equation in order to get the membrane voltage.
require: v and u in Eqs 2.17 and 2.18

ensure: create the initial conditions required in the differential equations.
ensure: the requires will be get by solving these two differential equations:

dv/dt= 0.04v2 +5v+140-u-I .... to calculate the membrane voltage.
du/dt= a(bv-u) ..... to calculate the recovery time.

ensure: plot the obtained results of membrane voltage (v) against time of duration (t).

Figure 3.2: Algorithm to calculate and draw the membrane voltage of the single neuron using Izihekevich model.

The results of this first part of our work can be found in section 2, Figure 2.9 to Figure 2.13.

The second category, after generating the single spiking neurons we used these single spiking neurons in forming groups of neurons and measuring the synchronization occurred between these neural groups. This is realized as follows as in Figure 3.3.

The creating of neural groups starts by setting a loop to satisfy the condition of running times, which is equal to number of recording times of membrane potential. To have a high accuracy, more number of values should be recorded. In our case we recorded 120 values for membrane potential for each one of the neurons.

In the first model we took the Regular Spiking (RS) as the excitability group, and the Fast Spiking (FS) as the inhibitory group. The values of the parameters used to built the single neuron model was taken from [8] and [36] and are mentioned in Table 2.2.

The change in the membrane voltage was calculated using the following differentiation equations: Eq 2.17 to Eq 2.19 that are responsible for the calculating the membrane voltage and the recovery time after spiking.

Algorithm 2: calculate and draw the synchronization of two groups of neurons.
==========

information: these codes written in BRIAN simulator which is a special library of Python Programming Language.
ensure: in order to get the required results properly, the following libraries should be imported:

NumPy and Scipy packages for Python: an efficient scientific library.
PyLab package for Python: a plotting library similar to Matlab.
SymPy package for Python: a library for symbolic
mathematics (not mandatory yet for Brian)

ensure: set the duration of running.
ensure: the parameters required to solve the equation in order to get the membrane voltage.
require: v and u in Eqs 2.17 and 2.18
ensure: create the initial conditions required in the differential equations.

ensure:
dv/dt= 0.04v2 +5v+140-u-I .... to calculate the membrane voltage.
du/dt= a(bv-u) .....to calculate the recovery time.

ensure: number of neurons in each group.
Ni=800, Ne= 200

ensure: creating the groups
Pe=NeuronGroup(Ne,diff eq of RS, reset cond.excitatory)
Pi=NeuronGroup(Ni,diff eq of FS, reset cond.inhibitory)

ensue: creating the connection
connection1=(Pe to Pi,conductivity variable.inhibitory, weights.inhibitory, sparseness)
connection2=(Pi to Pe,conductivity variable.excitatory, weights.excitatory, sparseness)
connection1_1=(Pe to Pe,conductivity variable.excitatory, weights.excitatory, sparseness)
connection2_2=(Pi to Pi,conductivity variable.inhibitory, weights.inhibitory, sparseness)

require: calculate the synchronization of the two neuron groups.

ensure: plot the obtained results of membrane voltage (v) against time of duration (t).

Figure 3.3: Algorithm to calculate and draw the synchronization of two groups of neurons.

Eq 2.19 is the reset condition, that is when membrane voltage reaches 30 mV, the membrane voltage and the recovery time will be reset to a certain values (c and u+d) respectively, these values (c and d) determines the spiking behaviour as given in Table2.2.

Eq 3.1 is to calculate the differential change in the synaptic connection between neurons, where *g* represents the change in the conduction between two neurons endings due to the effect of interaction of neurotransmitter with the opening postsynaptic membrane of the targeted neuron (the dendrites of the targeted neuron) [26].

In Figures 3.4 and 3.5, the results for human and rat cortex are give respectively.

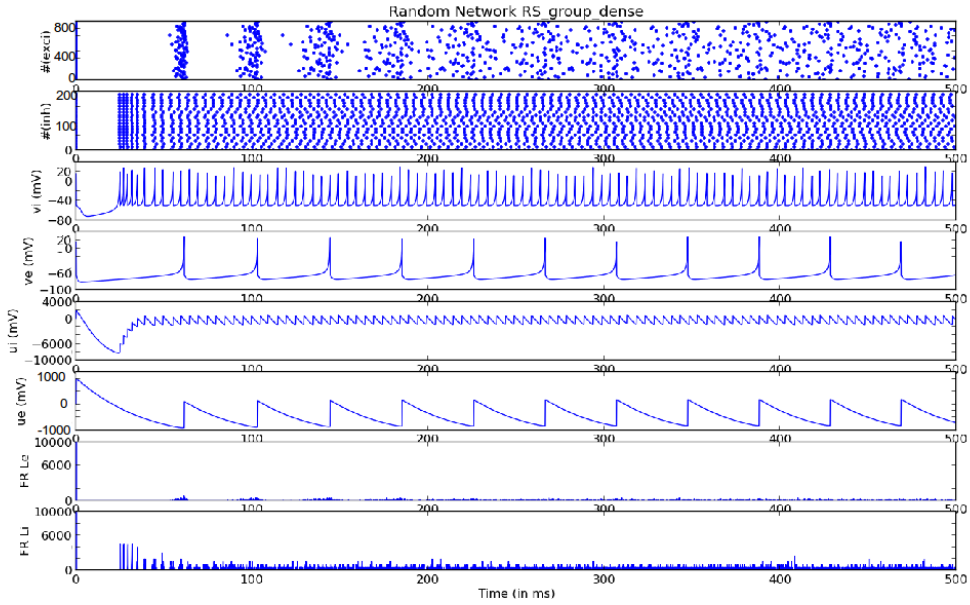

Figure 3.4: (RS-FS Human cortex): The network results for the network where the excitatory neurons are regular spiking, the weight is 20 mV ,duration is 500 msec, and the sparseness is 0.1.

The difference between human cortex and rat cortex where discussed in the previous section 3.1.

In Figures 3.4 to 3.7 we can see the upper two rows which represent the raster plots of the two connected neuron groups, RS as excitability and FS as inhibitory. Raster plot shows us the which one of the neurons has fired a spike at specific unit of time which is here 'second'. We made the number of RS (excitatory) group neurons as 800 neuron and 200 for FS (inhibitory) group neurons, this is identical to percentage of excitatory and inhibitory neurons in real in human cortex [38].

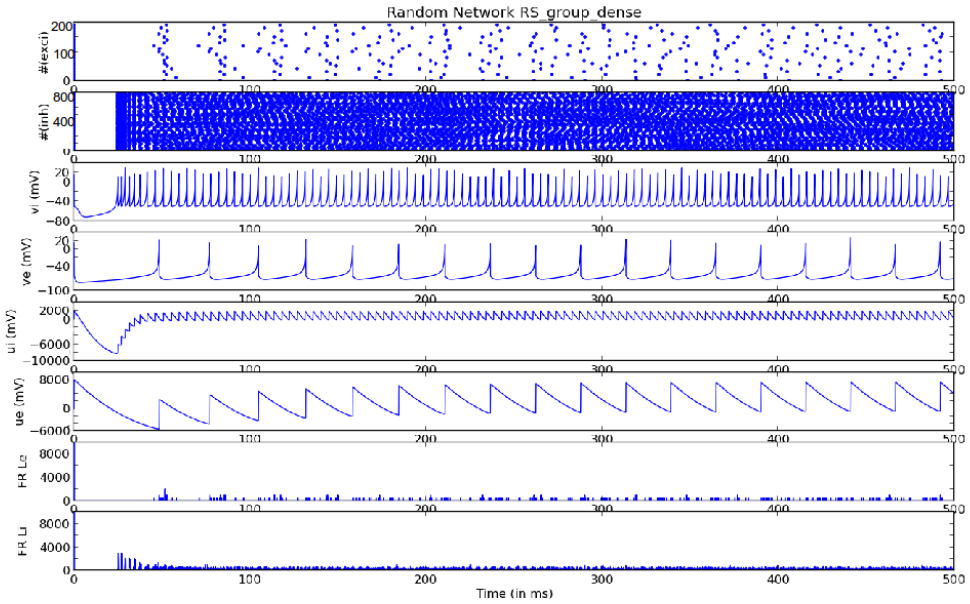

Figure 3.5: (RS-FS Rat cortex): The network results for the network where the excitatory neurons are regular spiking, the weight is 20 mV, duration is 500 msec, and the sparseness is 0.1.

The next two rows (v_i and v_e) represents the membrane voltage plot of a randomly selected single neuron, where v_i stands for voltage membrane for single neuron which taken as inhibitory (FS) and the v_e stands for voltage membrane for single neuron taken as excitatory (RS). The other two rows are u_i and u_e, represents the recovery time for both the inhibitory group and the excitability group.

The last two rows represent the Firing Rate, which is the sum of the neurons that has been recorded as successful spike in the raster plot (the most upper field).

The raster plot helps us to estimate roughly whether there is a synchronization between two neuron groups or no. Where, the left side of the raster plot rows shows the recording of regular repetitive spikes with almost identical spaces between spike and another, which indicates that at this period of time the group were in synchrony. This happened due to that the connecting weights is so low, that means practically there is not connection between the two groups. On the other hand, the more the right side we go, we can notice that there is a more random spiking depictions, which indicates low synchronization.

The sparseness where taken as 0.1 for the excitability group and 0.02 for the inhibitory group, since this is the values can be seen in real human cortex [10].

The second model we made, was by implying CH spiking type as excitatory group and keep FS as inhibitory group. Since CH spiking (same as RS spiking) is identical to the behaviour of excitatory Pyramidal neurons in cortex. Again the calculation over this model were taken twice, and the resultant figures were as follows, Figure 3.6 for human cortex while Figure 3.7 for rat cortex.

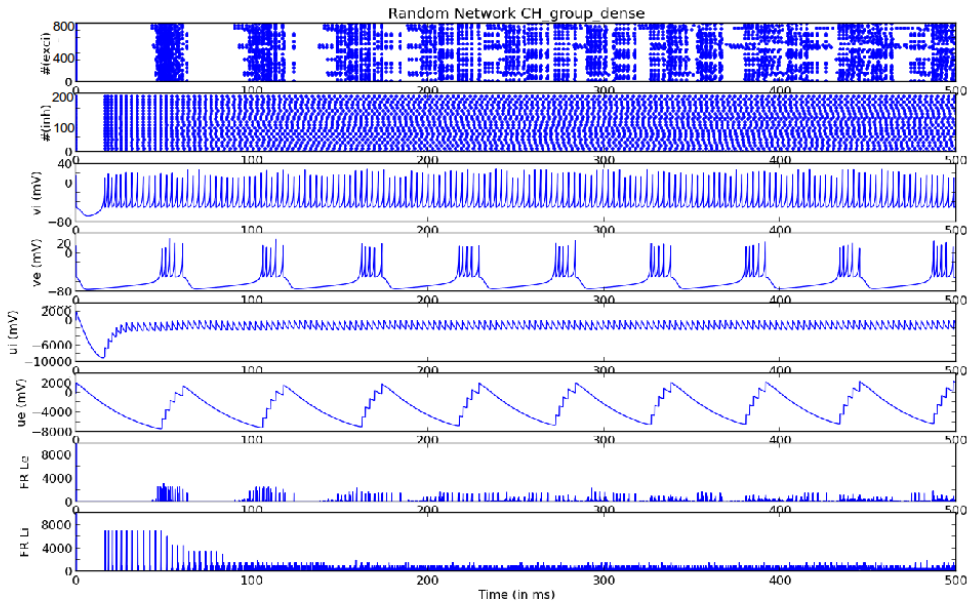

Figure 3.6: (CH-FS Human cortex): The network results for the network where the inhibitory neurons are chattering, the weight is 20 mV, duration is 500 msec, and the sparseness is 0.1.

Here also we can notice that there is a low synchronization since the connecting weights is so low, that means practically there is not connection between the two groups, but the synchronization in RS-FS model were a little more that CH-FS model, since there have different spiking frequencies came from the fact that they have different parameters.

Again sparseness where taken as 0.1 for the excitability group and 0.02 for the inhibitory group [16].

The importance of synchronization measurement to the modelling of neuron groups have been discussed in the previous section.

To obtain the synchronization measurement, we were recording the resultant membrane voltages in matrices during the running of the program. These matrices will have a size of [5000x200] for the excitability group, and [5000x800] for the

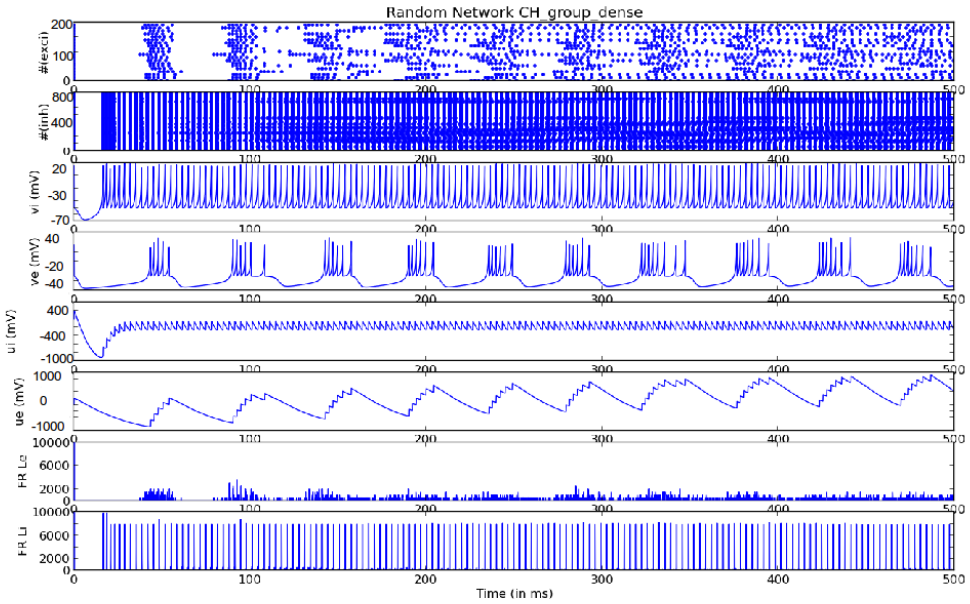

Figure 3.7: (CH-FS Rat cortex):The network results for the network where the inhibitory neurons are chattering, the weight is 20 mV, duration is 500 msec, and the sparseness is 0.1.

inhibitory group (for rat cortex and vice versa for human cortex model). Where the 5000 represents the number of membrane voltage values that has been recorded, it comes from (500/0.1), where 500 is the duration time (running time) and 0.1 is the recording resolution.

So in each second, we had 10 membrane voltage values for each single neuron in both of the groups. While the 200 and 800 represents the number of neurons in the excitability and inhibitory groups respectively.

We used previously mentioned synchronization measurement factor, which we obtained by implying Eqs 3.2 through 3.5 [40].

The results were as following:

The obtained results show that, a synchronization appears between the neural groups, this synchronization increases as connection weights increase with keeping the other variables constant as shown in the Figure 3.8 for human cortex, also in Figure 3.9 for rat cortex.

Figure 3.8 is for the human cortex for both RS-FS model and CH-FS model respectively, while Figure 3.9 is for the rat cortex for both RS-FS model and CH-FS model.

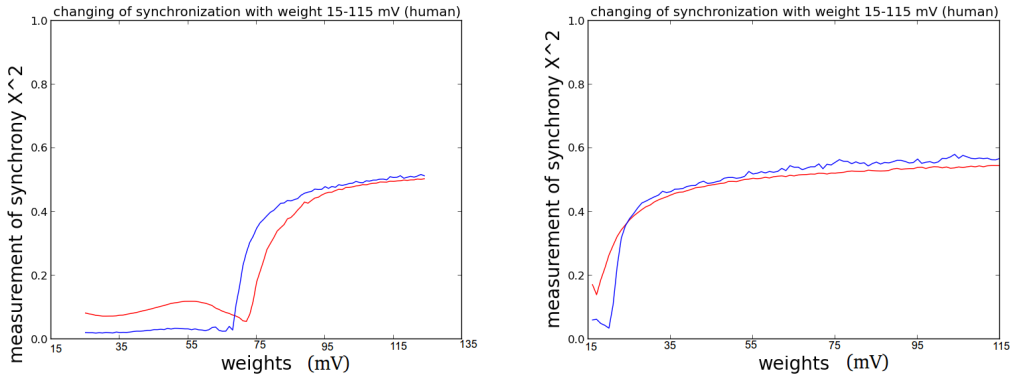

Figure 3.8: (RS-FS and CH-FS for Human cortex): The change in the measure of synchrony due to weights, where the excitatory group has regular spiking neurons. Red line gives result for excitatory and blue line gives result for inhibitory group.

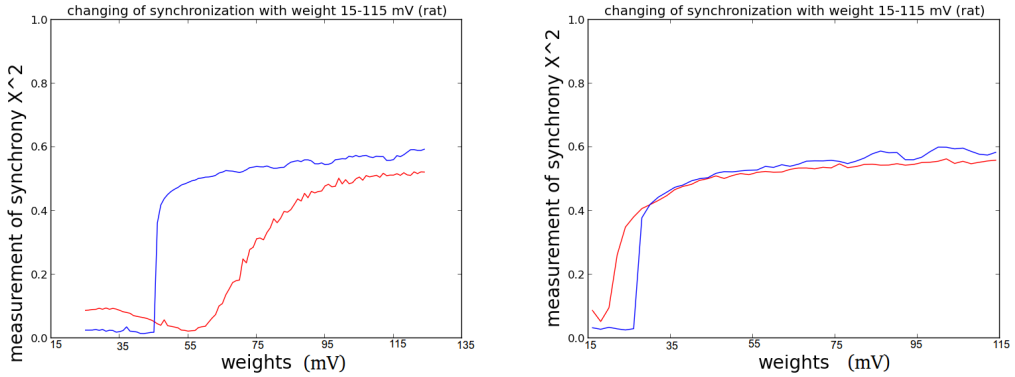

Figure 3.9: (RS-FS and CH-FS for Rat cortex): The change in the measure of synchrony due to weights, where the excitatory group has regular spiking neurons. Red line gives result for excitatory and blue line gives result for inhibitory group.

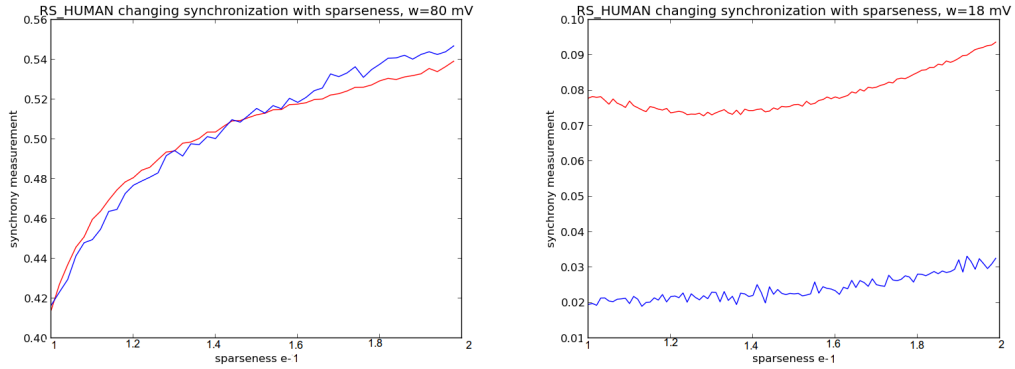

Figure 3.10: (RS-FS for Human and Rat cortices): The change in the measure of synchrony due to sparseness, where the excitatory group has regular spiking neurons. Red line gives result for excitatory and blue line gives result for inhibitory group.

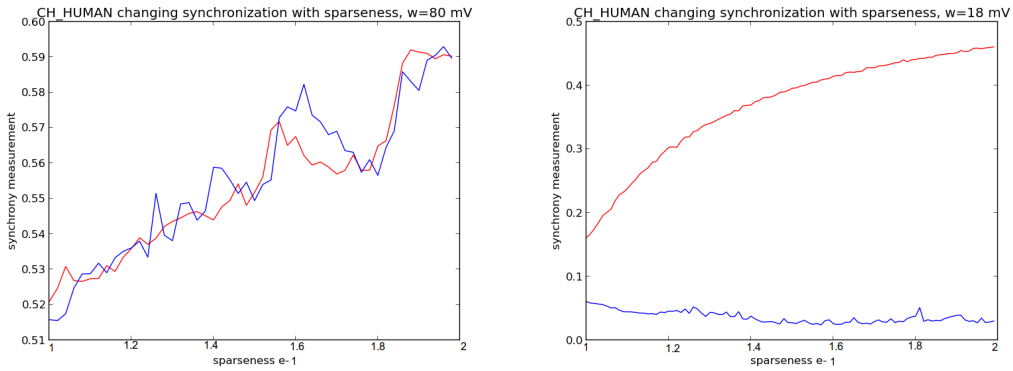

Figure 3.11: (CH-FS for Human and Rat cortex): The change in the measure of synchrony due to sparseness, where the excitatory group has regular spiking neurons. Red line gives result for excitatory and blue line gives result for inhibitory group.

Synchronization increase also with the increase of sparseness with keeping the other variables constant as can be seen in Figure 3.10 to Figure 3.11 for human cortex, also in Figures 3.12 to Figure 3.13 for rat cortex.

In Figure 3.10 to 3.13 we plotted the changing in synchronization vices changing sparseness from 0.1 to 0.2, since these are the real avialable values in the cortex [1], keeping the weight of connection fixed. The connection weights was fixed to two different values, 18 mV and 80 mV. The idea of taking these values is to make a comparison and get a better understanding of Figures 3.8and 3.9. If we look at these two figures, we can see that when the connection weight equal to 18 mV, there is no synchronization, the calculated values shows 0.02-0.1. While, when the connection weight increase up to 20 mV synchronization starts gradually to appear as in CH part of Figures 3.8 and 3.9, and up to 40 mV in the RS part. So, we chose connection weight values 18 mV and 80 mV to be sure that the first value fails in the non-synchronization field and 80 mV fails in the synchronization field, so that we can study the effect of sparseness purely without any interaction from the connection weight.

The importance of calculating the relation between synchronization and sparseness is to determine the required value of sparseness to obtain the synchronization, any value of sparseness upper than that critical recorded value at the synchronization-starting point, will ensures us a tight synchronization, this stands for both models and for both human and rat cortices [41].

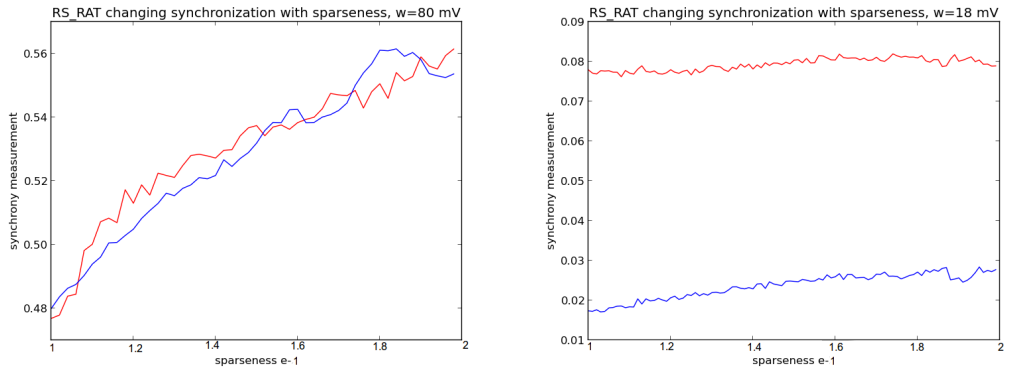

Figure 3.12: (RS-FS for Human and Rat cortices): The change in the measure of synchrony due to weights, where the excitatory group has regular spiking neurons. Red line gives result for excitatory and blue line gives result for inhibitory group.

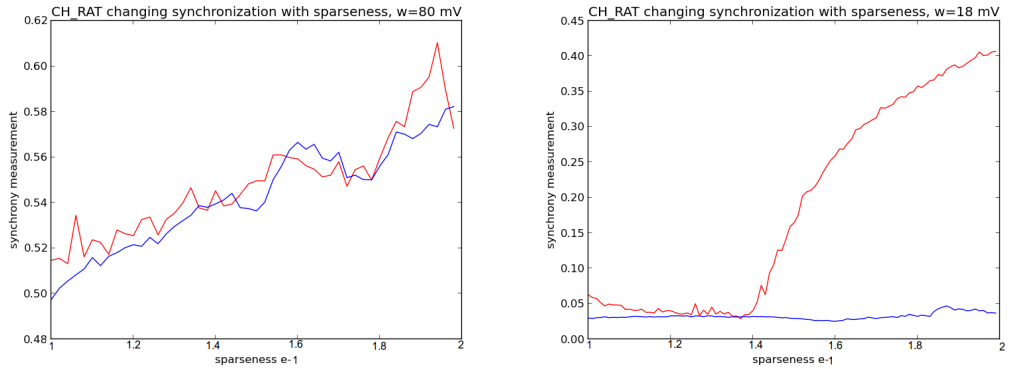

Figure 3.13: (CH-FS for Human and Rat cortices): The change in the measure of synchrony due to weights, where the excitatory group has regular spiking neurons. Red line gives result for excitatory and blue line gives result for inhibitory group.

We can notice that the CH-FS model reaches higher synchronization values compared to RS-FS model as can be easily noticed in Figures 3.10 to 3.13.

In Figures A.2 to A.4, we can see the effect of changing the value of sparseness. As we can see, changing the sparseness from 0.1 to 0.2 have significant effect of synchronization. It changed the threshold value at which the synchronization happens.

In general, we can conclude that the effect of sparseness on synchronization is more recognizable than the effect of weights of connection.

Finally, we can conclude that increasing the weight or the sparseness of connection will increase the connection between neurons, the synchronization also increases to some extend, in a non-linear relationship.

4. CONCLUSION

The cerebral cortex considered as the most advanced part of the brain since it is responsible and included in many difficult functions like, sensory functions interpreting, cognitive functions include thinking, analysing, and understanding language which considered as the most complex and difficult jobs done by the brain. Simulating the brain cortex have a special importance since by studying brain cortex we can learn more about brain dysfunction in mental disorders and neurological disease.

In our work we made the model of the single Pyramidal Neuron and then study the behaviour of a group of these neurons when connected to each other in a way similar to the real connections in the original cortical tissue. We also studied the degree of synchronization occurred and the factors affecting it including the effect of connection weights and connection sparseness.

Synchronization is an indicator of a coherent behaviour of a number of different dynamical systems, where the solution of each separate system converges to the same solution [42]. That is when the spiking of two neuron groups shows an accepted level of synchrony or coherent in their spiking, it indicates that they are well connected in some level of disciplinary and not behave in a completely random way [10]. The obtained results of synchronization factor given in Section 3 show that the synchronization between neuron groups increases as the connection weights increases.

These simulation results are obtained in BRIAN environment where networks of neurons are realized with the different ratios of excitatory and inhibitory neurons. These excitatory/inhibitory neurons are modelled using Izhikevich model and different neuron behaviours are obtained by changing parameter values.

Synchronization also helps to keep the signals between interconnected neurons robust to the intrinsic noise (the inter-neuronal noise), which allow a reliable communication and signal transition even in a noisy environment [20].

We investigated also the effect of sparseness on synchronization, we concluded that synchronization increase also with the increase of sparseness with keeping the other variables constant. The importance of calculating the relation between synchronization and sparseness is to determine the required value of sparseness to obtain the synchronization, any value of sparseness upper than that critical recorded value at the synchronization-starting point, will ensures us a tight synchronization

Synchrony also used to detect the coordinate activity between different parts of of the brain, like when someone wants to do something, it needs the cooperation of different parts of his body.

It is has strongly thought that the cortex plays a role which leads these different parts of the brain to work in coordinate by prompting them through excitatory spikes, which will appears eventually in the way of synchrony in their spiking [17].

As a future work, we can change the conductivity variable of connection and study its effect of synchronization.

REFERENCES

[1] **Eric R. Kandel, James H. Schwartz, T.M.J.** (2010). Principles Of Neural Science, McGraw-Hill Professional, p.292.

[2] **Url-1**, `http://thebrain.mcgill.ca/flash/d/d_02/d_02_cl/d_02_cl_vis/d_02_cl_vis.html`, date at which being taken: 16.03.2013.

[3] **CompaniontoBrainFacts.org, A.** (2009). Brain Facts A Primer On The Brain And Nervous System, USA.

[4] **Dean, T.** (2005). A computational model of the cerebral cortex, *The Twentieth National Conference on Artificial Intelligence*, Pittsburgh, Pennsylvania.

[5] **Ashby FG, H.S.** (2011). Simple Model of Spiking Neurons, ***55***(4), 273–289.

[6] **Mark F. Bear, Barry W. Connors, M.A.P.** (2010). Neuroscience Exploring The Brain, 4.

[7] **Gustavsson A., e.** (2012). Cost of disorders of the brain in Europe 2010., *European Neuropsychopharmacology, Elsevier*, ***22***(3), 237–238.

[8] **Izhikevich, E.M.** (2003). Simple model of spiking neurons, *Neural Networks, IEEE Transactions*, ***14***.

[9] **Url-2**, `http://bluebrain.epfl.ch/page-56882-en.html`, date at which being taken 16.03.2013.

[10] **Wang, X.J.** (2010). europhysiological and Computational Principles of Cortical Rhythms in Cognition, *American Physiological Society*, ***110***(31), 12828–12833.

[11] **Mark F. Bear, Barry W. Connors, M.A.P.** (2010). Neuroscience Exploring The Brain, 7.

[12] **Url-3**, `http://www.studyblue.com/notes/note/n/brain--cranial-nerves/deck/1242177`, date at which being taken: 16.08.2013.

[13] **Charles Watson, Matthew Kirkcaledie, G.P.** (2010). The Brain An Introduction to Function Neuroanatomy, Elsevier, USA.

[14] **Wells, R.B.** (2005). Cortical Neurons and Circuits: A Tutorial Introduction, *University of Idaho*.

[15] **Mark F. Bear, Barry W. Connors, M.A.P.** (2010). Neuroscience Exploring The Brain, 8.

[16] **Malsburg, C.** (2003). The Handbook ov Brain Theory and Neural Networks.

[17] **Mark F. Bear PhD, Barry W. Connors PhD, M.A.P.P.** (2010). Neuroscience Exploring The Brain.

[18] **Igor Timofeev, Maxim Bazhenov, J.S.T.S.** (2012). Neuronal Synchronization and Thalamocortical Rhythms in Sleep, Wake and Epilepsy, USA, pp.104–113.

[19] **Womelsdorf, T. and Fries, P.** (2007). The role of neuronal synchronization in selective attention, *ELSEVIER, ScienceDirect, CONEUR*, ***459***, 7.

[20] **Nicolas Tabareau, Jean-Jacques Slotine, Q.C.P.** (2010). How Synchronization Protects from Noise, *Computational Biology*, ***6***(e1000637), 156–165.

[21] **Mark F. Bear, Barry W. Connors, M.A.P.** (2010). Neuroscience Exploring The Brain, 9–10.

[22] **Url-4**, `http://www.indiana.edu/~p1013447/dictionary/assn_cor.htm`, date at which being taken: 16.12.2012.

[23] **Arnould-Taylor, W.** (1998). A Textbook of Anatomy and Physiology Paperback, Trans-Atlantic Publications, Inc.

[24] **Eric P. Widmaier, Hershel Raff, K.T.S.** (2010). Human Physiology The Mechanisms of Body Function.

[25] **Eric P. Widmaier, Hershel Raff, K.T.S.** (2010). Human Physiology The Mechanisms of Body Function.

[26] **David Sterratt, B.G. and Gillies, A.** (2010). Principles of Computational Modelling in Neuroscience.

[27] **Izhikevich, E.M.** (2005). Dynamical Systems in Neuroscience: The Geometry of Excitability and Bursting, San Diego, California, USA.

[28] **Izhikevich, E.M.** (2003). Simple model of spiking neurons, *Neural Networks, IEEE Transactions*, ***14***, 156–165.

[29] **Gupta, A.** Simple Model of Spiking Neurons, *Lally School of Management and Technology*.

[30] **Romain Brette, D.G.** (August 29, 2012). Brian Documentation.

[31] **Sterratt, D.** Neural Computation, *University of Yale*.

[32] **Bower, J.M. and Beeman, D.** (2010). The Book of GENESIS: Exploring Realistic Neural Models with the GEneral NEural SImulation System, Springer.

[33] **Novere, N.L.** (2013). Computational Systems Neurobiology, Springer.

[34] **Odyssee, P.T.** Mvaspike documentation, *Inventeurs du Monde Numerique*.

[35] **Goodman, D. and Brette, R.** Brian: a simulator for spiking neural networks in Python, *NEUROINFORMATICS*.

[36] **Richard Naud, Nicolas Marcille, C.C. and Gerstner, W.** (2008). Firing patterns in the adaptive exponential integrate and fire model, Switzerland.

[37] **C. W. Gatome, L. Slomianka, H.P.L. and Amrein, I.** (2010). Number Estimates of Neuronal Phenotypes in Layer II of The Medical Entorhinal Cortex of Rat And Mouse, *Neuroscience*, ***170***, 156–165.

[38] **Jo Watts, A.M.T.** (2004). Excitatory and inhibitory connections show selectivity in the neocortex, *The journal of Physiology*, ***591***(17).

[39] **Tak Pan Wong, P.** (2002). Aging of the Cerebral Cortex, *MJM*, ***6***, 104–113.

[40] **Url-5**, `http://www.scholarpedia.org/article/Neural_synchrony_measures`, date at which being taken: 2013.

[41] **Christoph Börgers, N.K.** (2003). Synchronization in Networks of Excitatory and Inhibitory Neurons with Sparse, Random Connectivity, *Neural Compution*, ***15***(3), 509–538.

[42] **Pecora, L.M. and Carroll, T.L.** (1990). Synchronization in Chaostic Systems, *Physical Review Letters*, ***64***(8), 156–165.

APPENDICES

APPENDIX A.1 : Extra figures

APPENDIX A.1

In Figures A.1 to A.4, we can see the effect of changing the value of sparseness. As we can see, changing the sparseness from 0.1 to 0.2 have significant effect of synchronization. It changed the threshold value at which the synchronization happend.

In gerenral, we can conclude that the effect of sparseness on synchronization is more recognizable than the effect of weights of connection.

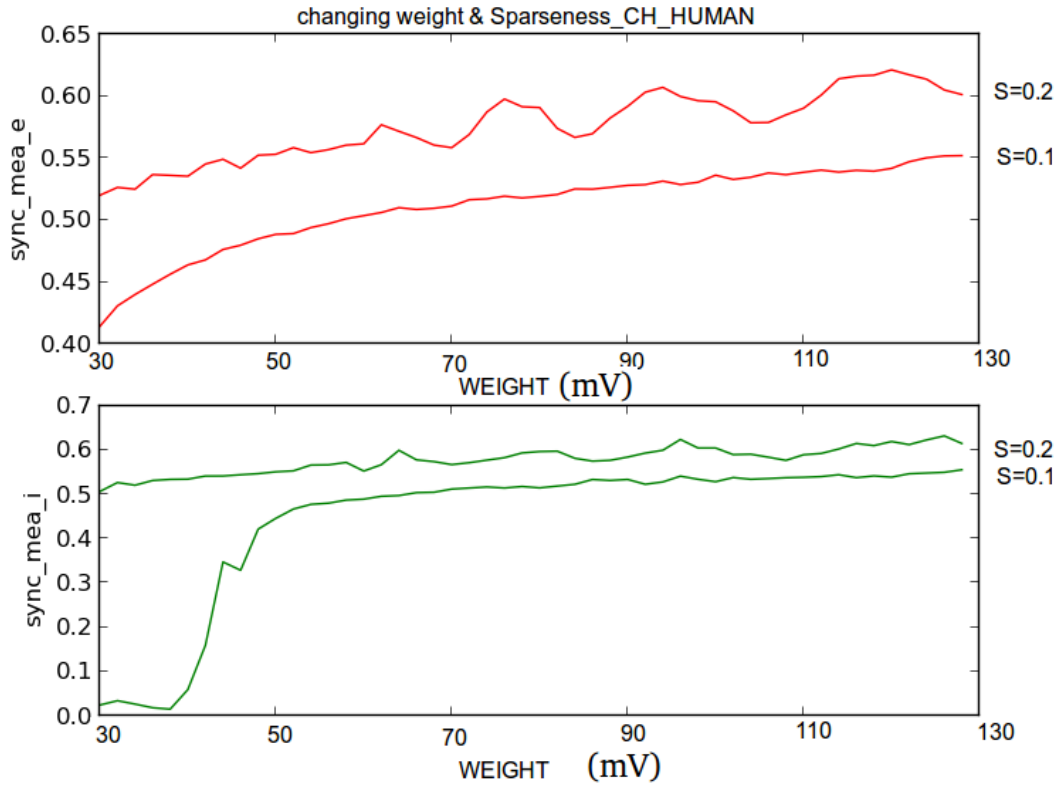

Figure A.1: (CH-FS Human cortex): change of synchronization with sparseness and connection weights togother, the taken values of sparseness where 0.1 and 0.2, since these are simiar to the real connection conditions in the human cortex.

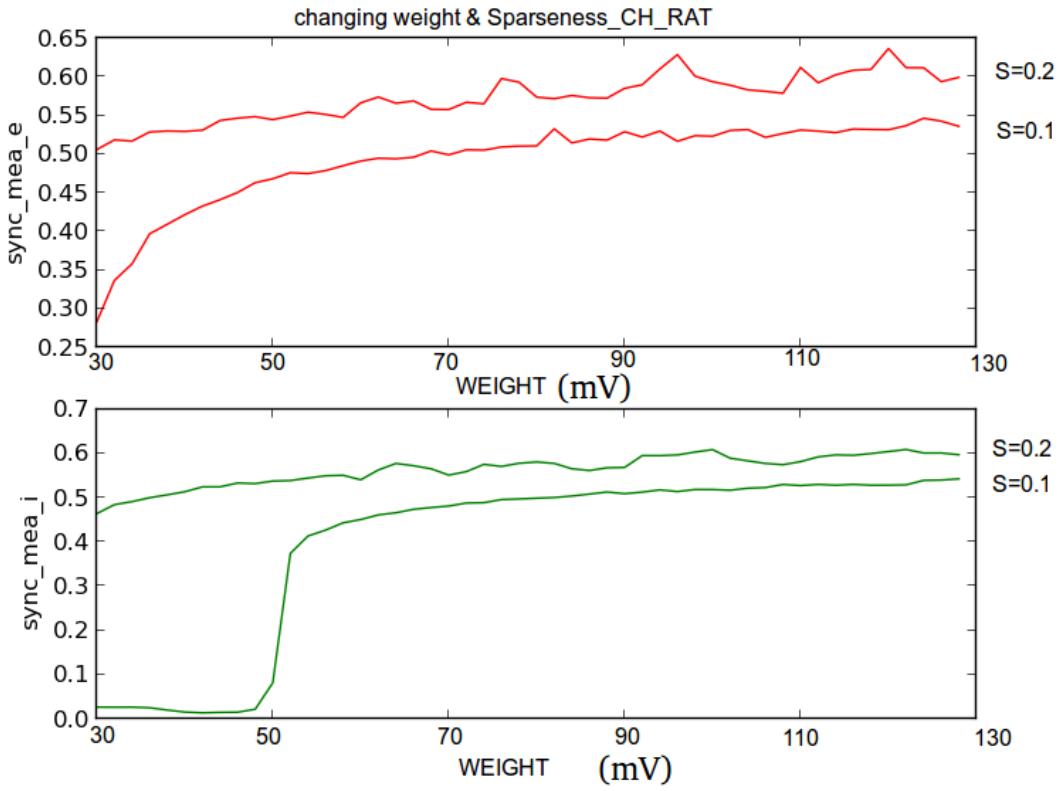

Figure A.2: (CH-FS Rat cortex): change of synchronization with sparseness and connection weights togother, the taken values of sparseness where 0.1 and 0.2, since these are simiar to the real connection conditions.

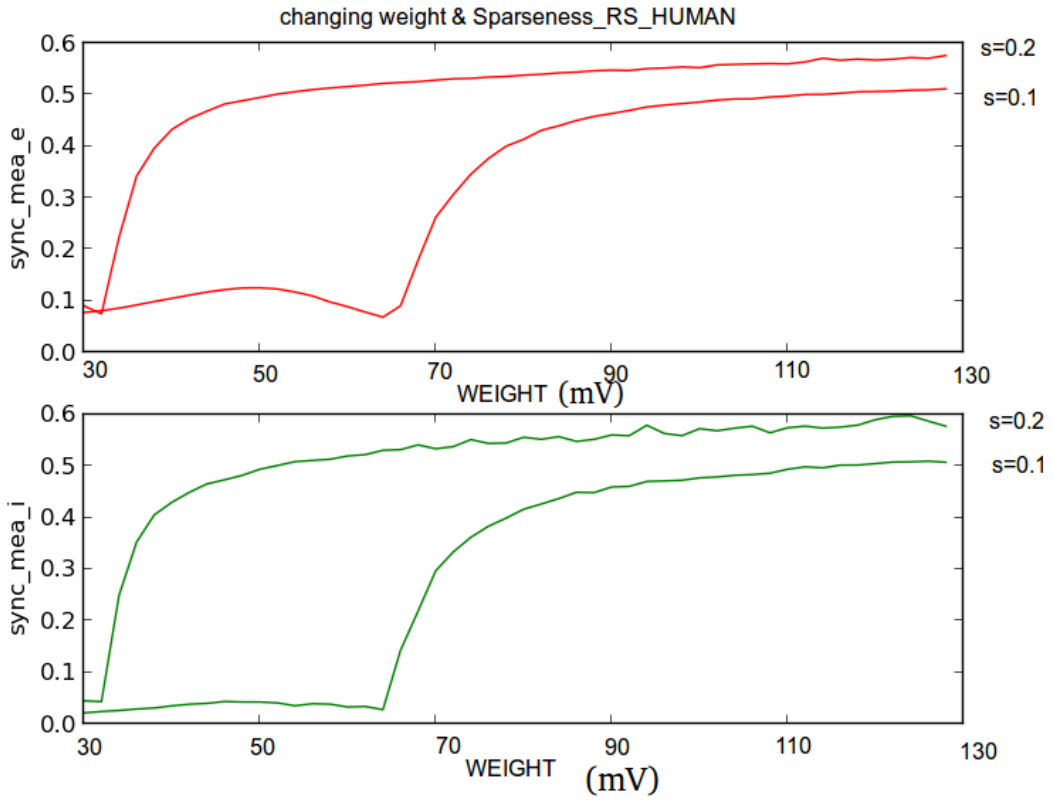

Figure A.3: (RS-FS Human cortex): change of synchronization with sparseness and connection weights togother, the taken values of sparseness where 0.1 and 0.2, since these are simiar to the real connection conditions.

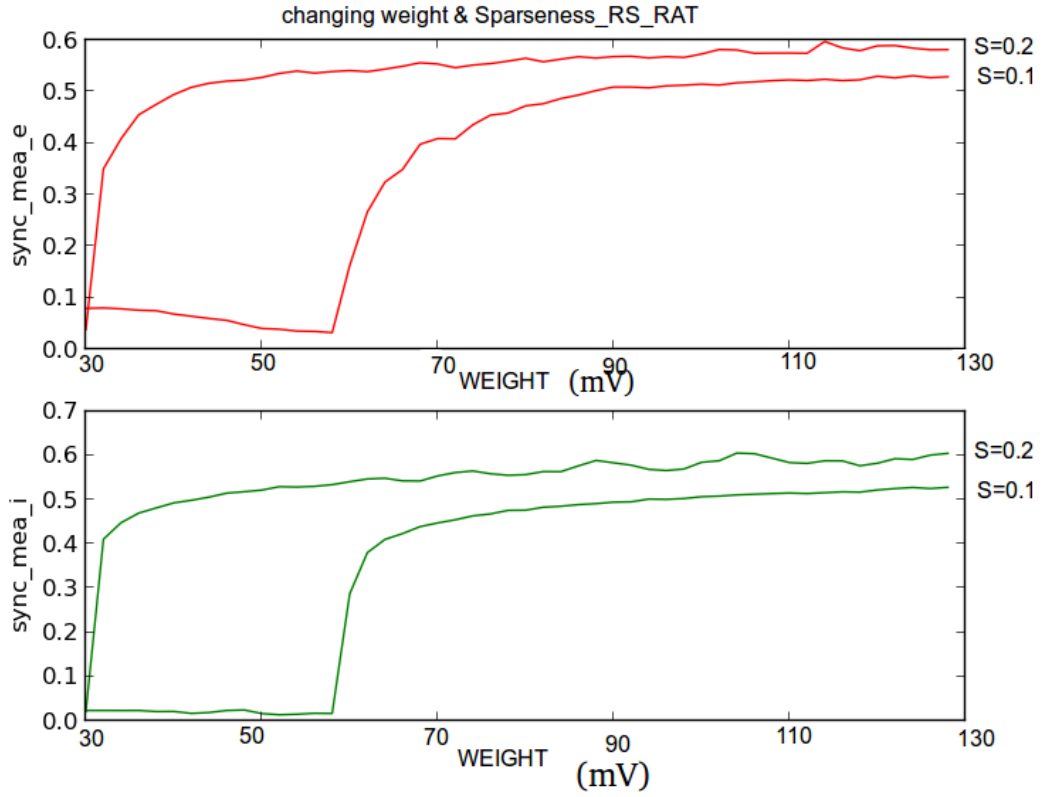

Figure A.4: (RS-FS Rat cortex): change of synchronization with sparseness and connection weights togother, the taken values of sparseness where 0.1 and 0.2, since these are simiar to the real connection conditions.

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