Ph. D. Dissertation

Theoretical study on physical properties of carbon nanotubes

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Department of Bio and Brain Engineering

KAIST

2003

Theoretical study on physical properties of carbon nanotubes

Theoretical study on physical properties of carbon

nanotubes

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A thesis submitted to the faculty of KAIST in partial fulfillment of the requirements for the degree of Doctor of Philosophy in the Department of Bio and Brain Engineering . The study was conducted in accordance with Code of Research Ethics1.

2002. 12. 5. Approved by

Professor Chang, Kee Joo

[Advisor]

1 Declaration of Ethical Conduct in Research: I, as a graduate student of KAIST, hereby declare that I have not committed any acts that may damage the credibility of my research. These include, but are not limited to: falsification, thesis written by someone else, distortion of research findings or plagiarism. I affirm that my thesis contains honest conclusions based on my own careful research under the guidance of my thesis advisor.

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ABSTRACT

Correlate neural activities, such as oscillation and synchronization are one of the important keys for communication in the brain. However, the question on how to modulate the synchronization level and how these synchronized activity affect spike transfer from one layer to another layer in different convergent connection conditions are not clearly understood. In this paper, we employ computer simulation of realistic neural network to address the question.

The neural network consists of two layers of conductance based single cell model, source layer and target layer. The interlayer connection follow statistical wiring diagram, for which, strength and connectivity of the connection depend on the distance between the target cell when pro jected on source layer and the source cells. For controlling, we built two other connection methods while fixed constant connectivity. One is the constant strength of connection and another the random strength of connection that follow negative exponential distribution. The response function of the neural network was measured with various pattern of input to the source layer. Then, the level of spike transfer from one layer to another was measured from various conditions. To study how one specific type of ions channel controls level of synchronization in the network, we conduct the experiment when T-type calcium channel functions and when it is not.

We showed that synchronization facilitates spike transfer between neuronal layers. We compared level of spike transfer in the controlled neural network system with independent input spike trains and the neural network with oscillating input. We found that when the synchronization between inputs to the system is high, the spike information can transfer to target network with less convergent connection.

In summary, we found that neural network could selectively allow the information from specific syn- chronization level to be transfer to another layer. And this level of synchronization in the network can be modulated by T-type calcium channel. This paper suggested more study on the potential of T-type calcium channel and level of synchronization as the key to understand information transfer in the brain.

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Chapter 1. Introduction

1.1 Old Intro

Correlated neural activities such as synchronizations and oscillations are observed in various areas in the brain. A number of studies suggest that this neural synchronization might be a key to understand various brain functions and brain diseases, but the detailed mechanism of how the synchronized neural activity can be systematically controlled is not completely understood yet. In this study, we use the simulated model neural network to understand how the synchronization of spike activities in local neural population can be modulated by the activity of a specific ion channel. Then, we examine the role of synchronization in transmission of information between the neuronal layers. In addition, we examine how different types of interlayer connection affects the level and speed of information transfer across neuronal layers.

The ob jectives of the work

o Reproduce the neural network of Parkinson’s Disease animal model

o Study bursting behavior in thalamic layer with and without T-type calcium channel

o Simulate the thalamocortical network of Parkinson’s Disease animal model using statistical wiring diagram

The justification for these ob jectives: Why is the work important?

Existing experiments in animals’ brain require much human effort and time in preparing different types of genetic manipulated animals

Outcomes of most experiments target on only a limited number of species

Results of those experiments are not sufficient to explain neural mechanisms in general

Many simulations use the parameters of experiments from the literatures without considering prop- erties of individual animal species Suggests the need to bridge the gap between simulations and experi- ment effort on animals

Background: Who else has done what? How? What have we done previously?’ Guidance to the reader: What should the reader watch for in the paper? What are the interesting high points? What strategy did we use? Summary/conclusion: What should the reader expect as conclusion? Research Question o RQ1 : How much can computational simulation resemble experimental results of animal study? o RQ2 : Can simulation predict functional connection between thalamus and motor cortex in the real animal? o RQ3 : Is the neural population with higher synchronization level better than the un-synchronized neural population in information transfer to another layer? o RQ4 : Would the neural population with high synchronization level require small level of convergence input to another layer given the same net level of presynaptic inputs compare to the neural population with unsynchronized activities.

1.2 New Intro according to professor on 15.05.29

¡Re-arrange the wording & sentences again ¿ Mention that you get inspired by the manuscript(?) findings(?) of Dr.Kim that they found the animal with T-type calcium channel get blocked lost the correlation between VL and M1 and resulted in reduce in motor output compare to the normal function

case . This lead to the question of, what is the role of synchronization in information transfer. In addition, the relationship between the neural synchronization and interlayer connection are not clearly understood. Note : Clearly mentioned that we do not use any of their data in the analysis. Just use it for reference.

Then talk about basic comp neuro analysis information and technique - Response Function - Any others Spike Statistics

1.3 Computational Neuroscience

1.4 Realistic Neural Network simulation

1.5 The NEURON simulator

1.6 The Correlated Neural Activities

1.7 The synchronization

1.8 Parkinson’s Disease

Chapter 2. Methodology

(Intro of methodology ) - Two layers, with two types of neurons WT & KO - Order of methodology

1. Single Cell

2.1 Prof ’s suggestion

General Note : If you fix trial number to some n value –¿ explain why it is enough. Never say it is due to the time limit that you cannot run more simulation.

2.1.1 Single Cell Model

2.1.2 Network Model

2.1.3 Connectivity

- Include lateral connection -¿ because you spent a lot of time building up the system - Show how to make connection / generate connection in detail (Just figure is not clearly enough) , may just show one sample and average of sample

................ How to do each analysis (the analysis that going to be shown in Result part) ................

2.1.4 Activity of VL itself ( osc and not osc)

- Definition of Efficacy

- Response Function

- Why are you trying the osc input (change parameters)

- Give the ”reason” in every thing you do - what are the expected output with different strength of oscillation - In and output oscillation

2.1.5 Distribution of Mosaics

(JS work, unpublished data, don’t mention in detail)

2.1.6 Activity of VL - M1 and connection type

- How to make contour line , the meaning of contour plot

- Why act when changing osc F and osc Amp change

What do you expect, why are you trying this. What are the meaning of these result in biological system

2.2 Single Cell Model

The single cell in this work has been model based on the Hodgkin-Huxley model, the conductance- based single cell model with addition of T-Type calcium channel and input synaptic connections [1, 2, 3]. The differential equations for the model can be shown as the following.

*dv*

*C dt* = − *g*L (*v* − *V*L ) − *G*N a (*v* − *V*N a ) − *G*K (*v* − *V*K ) − *X G*C aT (*v* − *V*C aT )

− *g*σE (*t*)(*v* − *V*E ) − *g*σI (*t*)(*v* − *V*I ) − *g*input (*t*)(*v* − *V*E )

where, *σ* : type of neuron, excitatory(E) or inhibitory(I),

*g*L : leakage conductance,

*g*σE or *g*σI : synaptic conductance providing excitatory or inhibitory input

*C* : membrane capacitance, *G*N a : Na channel conductance, *G*K : K channel conductance,

*G*C aT : T-type Calcium channel conductance,

*X* : T-type Calcium controlling factor;

X=1 for normal functioning case (WT) X=0 for not functioning case (KO)

The equations for sodium, potassium, and T-type calcium voltage-gated channel conductances( *G*N a

, *G*K and *G*C aT respectively ) are shown as the following

*G*N a = *g*¯N a *m*3 *h, G*K = *g*¯k *n*4 *, G*C aT = *g*¯C aT *r*3 *s*

Where m,h,n,r and s are the channel activation variable. For sodium and potassium channel,

*dx*

*dt* =*α*x (*v*)(1 − *x*) − *β*x (*V* )*x, x* = *m, h, n*

For T-type calcium channel, the mechanism has three state kinetic process

*dr*

*dt* =*α*r (*v*)(1 − *r*) − *β*r (*V* )*r*

*ds*

*dt* =*α*s (*v*)(1 − *s* − *d*) − *β*s (*V* )*s*

*dd*

*dt* =*β*d (*v*)(1 − *s* − *d*) − *α*d (*V* )*d*

Where the *α*x and *β*x are rate constants for each type of channel. The form for these rate constants are

taken from the existing empirical measurements [1, 4, 2].

For sodium channel,

*α*m (*v*) = 0*.*1 − (*v* + 40)

exp

−(*v* + 40)

10 − 1

*β*m (*v*) = 4 exp

−(*v* + 65)

18

*α*h (*v*) = 0*.*07 − (*v* + 65)

exp

−(*v* + 65)

20 − 1

*β*h (*v*) = 1*/*

exp

−(*v* + 35) + 1

10

For potassium channel,

*α*n (*v*) = 0*.*1(−(*v* + 55))

exp

−(*v* + 55)

10 − 1

*β*n (*v*) = 0*.*125 exp

−(*v* + 65)

80

For T-type calcium channel,

−(*v* + 28*.*2)

*α*r (*v*) = 1*.*0

1*.*7 + exp

13*.*5

*β*r (*v*) = exp

−(*v* + 63*.*0)

7*.*8

(*v* + 160*.*3)

exp

−(*v* + 28*.*8)

13*.*1

+ 1*.*7

*α*s (*v*) = exp ( −

17*.*8

*v* + 83*.*5

−(*v* + 160*.*3)

*β*s (*v*) =

0*.*25 + exp

6*.*3

− 0*.*5 ∗

exp

17*.*8

*v* + 37*.*4

*v* + 83*.*5

*α*d (*v*) = (1*.*0 + exp

30*.*0

*v* + 83*.*5

240*.*0 ∗ (0*.*5 +

0*.*25 + exp

6*.*3

*β*d (*v*) =

0*.*25 + exp

6*.*3

− 0*.*5

∗ *α*d

Each cell can receive both types of synaptic input, excitatory and inhibitory input. Upon the arrival of spike, or spike event, the membrane potential at the postsynaptic cell response to the input. They are called excitatory postsynaptic potential(EPSP) and inhibitory postsynaptic potential (IPSP) for excitatory and inhibitory input respectively. The conductance that responsible for EPSP and IPSP were

modelled as the following function, *G* = *w* ∗ (exp( *−*t ) − exp( *−*t ) where, w is weighting factor, *τ*1 is the

τ2 τ1

rise time constant, and *τ*2 is the decay time [4]. The value of *τ*1 and *τ*2 are 1 and 3 millisecond(ms)

respectively for EPSP, 1 and 7 ms respectively for IPSP.

2.2.1 Parameter search for single cell model

Most of the parameters were set with the well-known values. Some parameters - which are *g*C aT , *g*N a - were optimized so that the model single cell shows the same behavior with the experimental results. The criteria for choosing parameters are 1. The number of burst spike 2. The number of tonic spike 3. The drop in voltage after current injection

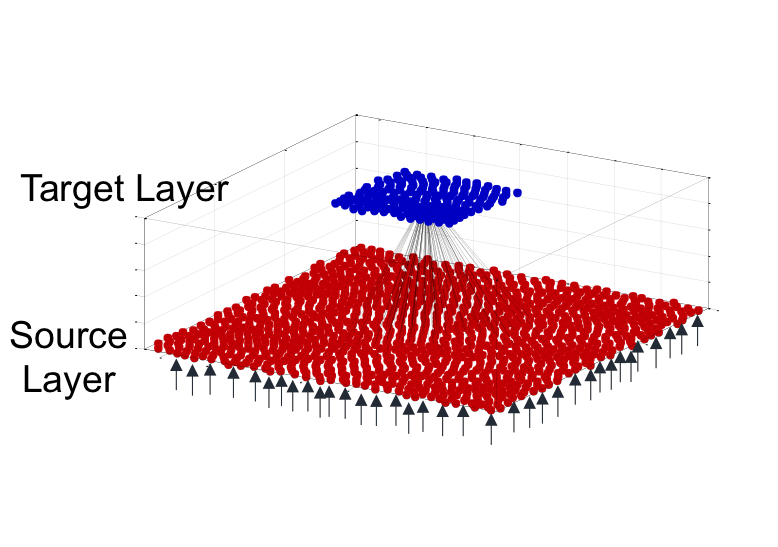


Figure 2.1: Network Sample

Behavior of single cell with and without T-Type calcium channel

2.3 Population Model

2.3.1 Model the neuronal mosaics

–¿ Repulsive interaction // Reference - PIPP model

2.3.2 Model Baseline activity

- Poisson input and - current fluctuation

2.3.3 Parameter search for population model

Parameters for cell in population model include input spike frequency and their weighting factor. The criteria to select parameters are output frequency and standard deviation of output frequency compare to the baseline activity recorded in experiments

2.4 Connection modelling

Figure 2.4 The model neural network with sample connection

2.4.1 Statistical Wiring Diagram

Connectivity and Strength of connection depend on distance between cell [5, 6]

2.4.2 Lateral connection modelling

2.4.3 Thalamocortical Connection (interlayers connection)

Testing Figure! There are three types of connections. First, the interlayer connection type 1 : Con- nectivity and Connection Strength follows Gaussian distribution ( Figure 2.4.3 ) Second, The interlayer connection type 2 : Connectivity and Connection Strength follows uniform distribution. (Figure 2.4.3 )

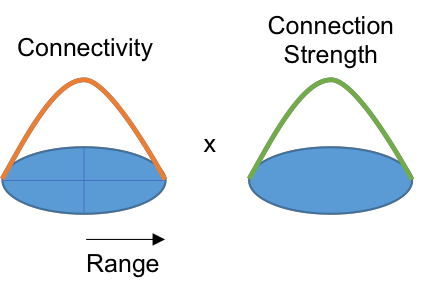


Figure 2.2: The interlayer connection type 1 : Connectivity and Connection Strength follows Gaussian

distribution

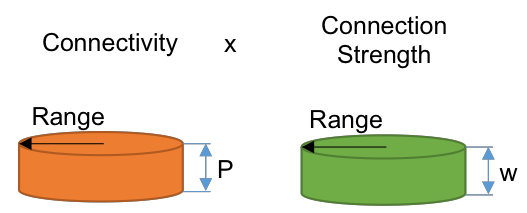


Figure 2.3: The interlayer connection type 2 : Connectivity and Connection Strength follows uniform

distribution

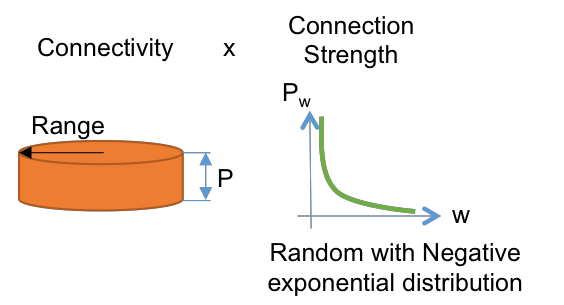


Figure 2.4: The interlayer connection type 3 : Uniform Connectivity and Random Connection Strength that follows negative exponential distribution

Last, The interlayer connection type 3 : Uniform Connectivity and Random Connection Strength that follows negative exponential distribution (Figure 2.4.3 )

2.4.4 Synchronization and information transfer in the network modelling

Chapter 3. Results

3.1 Prof ’s suggestion

General Note : If you fix trial number to some n value –¿ explain why it is enough. What are the meaning of these result in biological system

3.1.1 Proved your hypothesis

Hypothesis The brain (is it too big or too general?) need interlayer communication that optimized

cost of the connection which is achieved by the synchronized neural activity on source layer and statistical wiring diagram( with Gaussian distribution of connectivity and connection strength)

Proving the Hypothesis Compare differences in these three cases of connection (Gaussian, uniformly random, random with negative exponential) of input variations ( osc vs. no osc ; varies F and Amp ) The measurement can be 1) Fr , 2) Spike Correlation, 3) Spike Pattern (Ex. M1 activity according to Input oscillating pattern, how is the phase affect the result etc )

.................... OLD ....................

3.2 Answer to RQ1 : The computer simulation resemble ex- perimental results of animal study

3.2.1 The single cell properties of computational model and the whole cell patch clamp recording shows the same behavior

Figure .1.1: Comparing the sample membrane potential trace of WT and KO during hyperpolarize current injection and depolarize current injection

Figure .1.2: Comparing the number of bursting spikes and tonic spikes between the simulated cell and experimental data

3.2.2 The cell population properties of computational model and the MUA

cells recording shows the same behavior

Figure .1.3 : Comparison of mean and standard deviation of observed firing rate in cell population in computational model and experimental data

3.2.3 Neuron activities during light-off period (no photoactivation)

Figure .1.4 The baseline activities of neural population during light-off period show no significant different between WT and KO

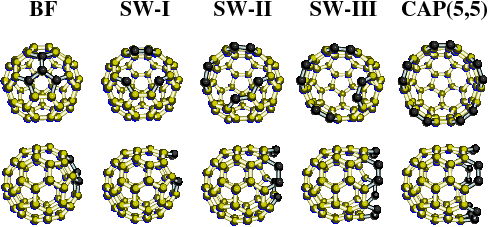


Figure 3.1: Sample Figure

3.2.4 Neuron activities during photoactivation

Figure .1.5 The delay and peak of rebound spiking activity of WT and KO are significantly different. The WT shows short rebounding period and higher peak of neural activities

3.2.5 The coherence between VL and M1 layer shows that correlated neural activities after photoactivation ( the rebound bursting spikes) drives activity in M1

Figure 3.2.5 The coherence between VL’s MUA and M1’s LFP

3.3 Answer to RQ2 : Computational simulation predicts causal relationship of exceed motor command in M1 from VL in Parkinson’s disease patient

3.3.1 The high synchronization (correlated neural activities) in VL but not average firing rate can drive M1’s motor command

Figure .2.1 The high synchronized neural activities during bursting can drive M1 in WT types but not KO, even though the average firing rate if WT and KO are not significantly difference

3.3.2 The high synchronization level can be achieved by bursting. The demolishing of bursting in WT neurons result in the absent of syn- chronization in neuron population

Figure .2.2 Schematic diagram of how the bursting activity cause high synchronization in neuron population

3.3.3 The Analysis of information transfer from VL to M1 : The informa- tion in VL can transfer to M1 when the neuron population in VL are synchronized

Figure .2.3 the neural information from VL is transferring to M1 only when the neural activities in

VL are synchronized.

Table 3.1: Energy stability *E* (eV) per molecule of all meta-stable isomer states of C60 opening process

for forming the (5,5) cap. In the SW-I and SW-II, both ferromagnetic (Ferro) and paramagnetic (Para) spin configurations are obtained, whereas only non-magnetic configuration is obtained in the BF, SW-III, and CAP(5,5). *M* is total magnetization *n*up -*n*down in unit of *µ*B , where *n*up(down) is the number of up (down) spins.

BF SW-I SW-II SW-III CAP

|  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- |
|  | | Para | Ferro |  | Para | Ferro |  | |
| *E* (eV) | 0 | 7.796 | 7.832 |  | 10.418 | 10.408 | 11.5 | 13.2 |
| *M* (*µ*B ) | 0 | 0 | 1.94 |  | 0 | 2.06 | 0 | 0 |

Figure .2.4 the level of information transfer between VL and M1 is proportionally to the synchro- nization level in VL

3.3.4 Artificially generated bursting in KO neurons result in high synchro- nization level of neuron population which can drive M1’s motor com- mand

Figure .2.5 successfully generated bursting behavior in KO with similar bursting behavior generated by T-Type Calcium channel in WT

Figure .2.6 The artificial bursting behavior in KO result in high level synchronization level of neural population and this high synchronized neural population can drive M1

3.3.5 Artificially activated VL neural population in theta and beta frequen- cies result in motor command from M1with the same frequency band with what observed in Parkinson’s disease patient.

Chapter 4. Conclusion

What does it mean? Successfully regenerate experimental results with computational simulation The simulation shows functional connection between thalamus and motor cortex in the real animal What hypotheses were proved or disproved? We can make computational simulation which resemble the experimental data The T-Type calcium channel generate bursting behavior of single cell The neural population are highly synchronized during bursting behavior The high synchronization level in neural population can transfer information from one layer to another layer What did I learn? I can make computational model that can regenerate experimental data and I can use it to predict new properties of neural system Why does it make a differences? The simulation predicts functional connection between VL and M1 neuronal layers The simulation suggest that reverse testing of KO cells to resemble WT can also drive motor command in M1. The finding suggests that the bursting is the important factor for high synchronization level of neural population and it is the key for neural network to transfer data from VL to M1

Add a new, higher level of analysis Indicate explicitly the significance of the work This work shows the potential of using computational simulation to regenerate experimental data in silico and employ it to manipulate properties of neuron network that are hard to do in the experiments and use it to predict new hypothesis

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Summary

Theoretical study on physical properties of carbon nanotubes

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