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# Distributions in Plastic Neural Network: Unimodal to Bimodal Transition

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**Abstract:** Empirically observed mean membrane potential (MMP) has been the focus of numerous investigations related to neurons. The distributions in MMP inherently remain prime target of many studies. The MMP largely remains Unimodal until the synaptic plasticity starts playing role and alters the coupling strength. The changes in the coupling strengths subsequently lead to the Unimodal to Bimodal distributions. In this paper an attempt has been made to establish the role of synaptic plasticity in this transition. The underlying synaptic plasticity framework, resulting the changes in the coupling strength, may contribute in generation of Bimodality in MMP. The investigation advocates the vitality of synaptic plasticity in MMP bimodal distribution generation.

**Keywords:** Coupled Neuro-system, Synaptic plasticity, Coupling Strength, Bimodal distribution.

## I. INTRODUCTION

A vertebrate brain is composed of billions of connected units known as Neurons. These neurons are connected to each other with the average of 103 distinct connections per neuron. These connections are responsible for transmitting signals from one neuron to another in terms of ions or neuro-transmitters. Further, these connections play a vital role in controlling the signal transmitted from one neuron to another. Many biophysical models have been developed to express these neuronal activities. But, being a complex phenomenon, different aspects of neuronal activities are captured via different categories of models. Many of these models are suitable for single neuronal spiking while some others are suitable for neuronal network [1][2].

Many studies have been conducted to gain the insights in this [3][4]. Overall dynamics of a single neuronal model are limited to its behavior in isolation. Whereas networked neurons, which is the reality of human brain, have more rich dynamics. Possible number of activities increases as the number of neurons taken into account is increased. Whenever the network of neurons is talked about, the characteristics of a communication network are inherited. Being the natural phenomenon, the noise becomes the part of each and every network [5][6]. The network of neurons, each of which is denoted by set of differential equation(s), brings up dynamical systems no matter how simple the model is [7]. As soon as the number of neurons increased the system becomes dynamical itself. As the field is emerging, new dimensions are being introduced and more unexplored areas are being added as well.

Every communication channel has some capacity. This capacity is governed by the coupling strength in between concerned neurons. The communication between neurons is a kind of information processing too [8]. Consequently it may be said that the dynamics and information distribution in between neurons is regulated by the respective coupling strengths.

Synaptic plasticity is the ability of changing the synapses in between neurons be it structural or else. This ability allows the connections to be altered in between two set or population of neurons [9]. The alteration may be in terms of strengthening or weakening the existing connection or articulate a new connection. In either way the coupling strength gets affected. The connections keep changing throughout the lifespan due to multiple reasons [10][11].

The coupling strength directly dictates the intensity of signal transmission in between any neuron pair. Coupling strength varies with the time and spiking activities in between respective neurons. It is highly possible that coupling strength in between one pair of neuron is not identical to another pair of neuron, and in most of the time it is not. Since every active neuron generates spike and inactive neurons remain at the resting potential, the coupling strength changes in between one set of neurons independent of another one. The discussion regarding independent changes in the coupling strength convergences to some ideal state or not remains open ended. But the coupling strength itself may follow some distribution. The challenge is to find out the distributions in the coupling strengths of neuron population.

There exists number of models that can be considered for the required experiment. However, the initial challenge is to choose a suitable model to demonstrate the interconnected neuron population. At the same time, the model shall be computation friendly. Izhikevich compiled number of popular neuronal models available and portrayed their biophysical plausibility and implementation

cost [1]. Rabinovich et al also systematised the neuronal models in their paper [2] in which they also gave pros and cons of the models.

Hindmarsh-Rose (H-R) model is taken into account after thorough investigation as it was evolved to investigate the interactions of neurons. The H-R model covers larger range of functional behaviour of a neuron and it is computationally feasible as well. In 1982, two British researchers developed the H-R model as two coupled differential equations of first order and then improved it in 1984 as three coupled differential equations of first order [12][13]. It is purely mathematical equations based model leaving behind the ion wise calculations related issues.

## II. HINDMARSH ROSE (H-R) MODEL

The H-R model defines membrane potential  $x(t)$  as

$$\dot{x} = y - ax^3 + bx^2 + I - z \quad (1)$$

Where  $\dot{y} = c - dx^2 - y \quad (2)$

And  $\dot{z} = r(s(x - x_1) - z) \quad (3)$

H-R model mainly focuses on single neuronal spike bursting scenario with the eight parameters taken together into account. Where  $I$  is the input current to the neuron and fast and slow ion channels are denoted by  $y(t)$  and  $z(t)$  respectively.

The proposed method considers a population of connected neurons with certain coupling strength assigned initially to each connection. The coupling strength gets changed during the experiment.

## III. PROBLEM FORMULATION

A population of coupled neurons is taken with the self-feedback loop possibility for each neuron. Each neuron has its own excitatory and inhibitory inputs. Presumptions are as following:-

- A. There is a possibility for each neuron to connect to another neuron with distinct coupling strength and has its mean membrane potential at sub-threshold level.
- B. The coupling strengths of neurons are set to follow uniformly distributed random numbers initially.
- C. Input from one to another neuron is the fraction of total input required for the spiking in the neuron.
- D. Coupling strengths are updated after certain iterations

The  $i^{th}$  coupled neuron is written as:

$$V(i) = \dot{x} + \sum_j C_{ij} V_j \quad (4)$$

Where

$V_i$ : Membrane Potential of  $i^{th}$  neuron and

$C_{ij}$ : Coupling strength towards  $i^{th}$  neuron from  $j^{th}$  neuron.

Initially the population of coupled neurons are assigned with specific coupling strength. The Coupling constant  $C_{ij}$  keeps the control over the signal received from  $j^{th}$  neuron. In other words, the coupling constant dictates the information transmitted from one neuron to another in terms of signals.

## IV. UNIFORM DISTRIBUTION

The uniform distribution is a distribution in the interval (a b) such that density of the event is constant over the interval. This distribution is taken to fix the coupling strengths of the connections initially.

Probability Density Function for the uniform distribution is given by

$$f(x) = 1/(b - a) \text{ for } a \leq x \leq b \quad (5)$$

As the equation (5) says, the pictorial depiction of uniform distribution shall remain a constant straight line for a sufficiently large generated numbers.

## V. SIMULATION METHOD AND RESULTS ANALYSIS

The key issue addressed here is to investigate the role of synaptic plasticity in the spiking of neuron. With this objective, the detailed simulation based study is carried out. The scope of the study is enlarged and bounded according to the hypothesis in the previous section. The Classical Runge-Kutta approximation method is used to solve the model depicted in terms of ordinary differential equations (1), (2) and (3). Therefore, the results obtained are the numerical approximation of the coupled Hindmarsh-Rose neuronal model for 120 neurons. The neurons are coupled in such a way that coupling strength may be modified.

Whole population of neurons is simulated according to Equation (4) with the model described by equations (1), (2) and (3). Each of the figures 1, 2 and 3 shows the spiking pattern generation of 3 individual neurons chosen from the population of 120 neurons. The simulation results are depicted in fig.1, 2 and 3. The results may be divided into three categories as Case 1, 2 and 3.

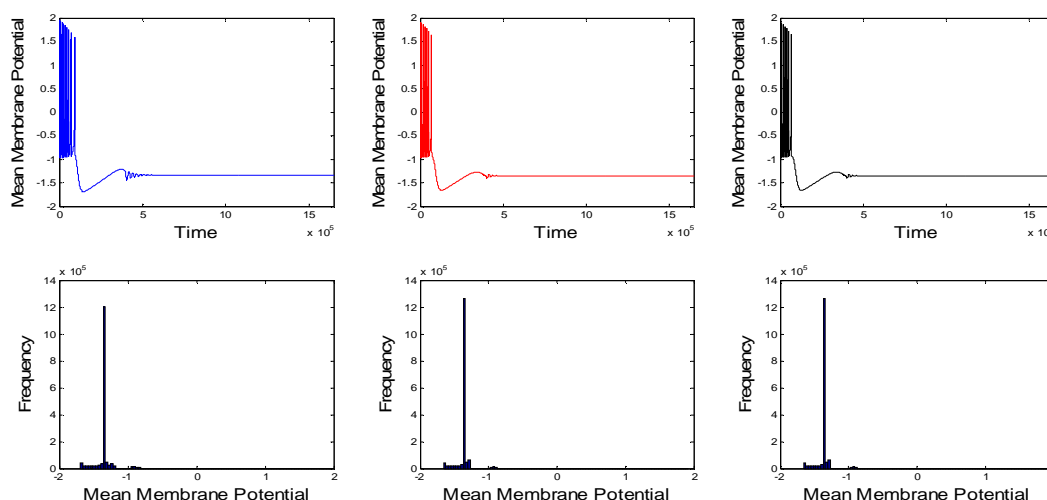


Fig. 1  $a = 3$ ,  $I = 2.60$ ,  $c = 0.0011$ ,  $d = 10$ , coupling strength increment factor = 0.000

#### A. Case 1

In this case the coupling strength remains unchanged as it happens when there is no plasticity in between neurons. The synaptic plasticity remains ineffective resulting in constant coupling strengths. As the neurons are set to sub threshold potential, none of them is able to generate spikes.

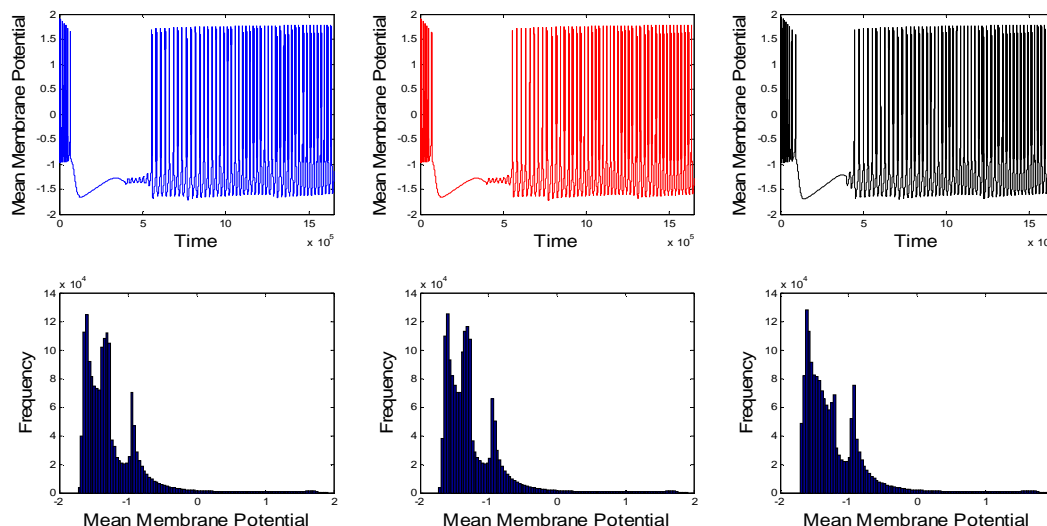


Fig. 2  $a = 3$ ,  $I = 2.60$ ,  $c = 0.0011$ ,  $d = 10$ , coupling strength increment factor = 0.001

#### B. Case 2

This scenario takes the account when there is a little bit of plasticity in between neurons. It results in the modifications in coupling strength. Therefore, synaptic plasticity starts playing role and changing coupling strengths in at small scale.



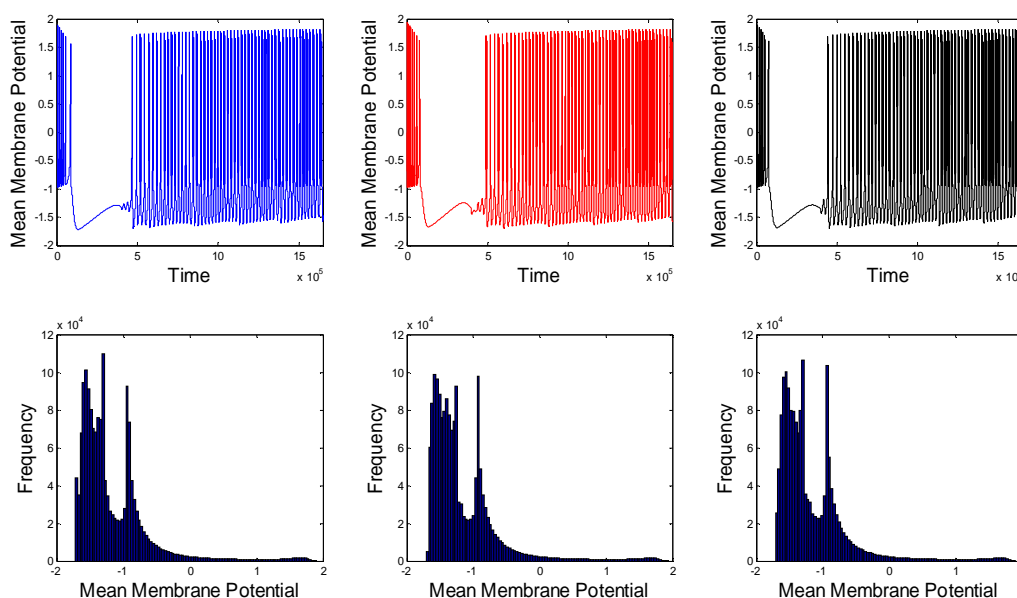


Fig. 3  $a = 3$ ,  $I = 2.60$ ,  $c = 0.0011$ ,  $d = 10$ , coupling strength increment factor = 0.002

### C. Case 3

In this case higher synaptic plasticity in between neurons is assumed. For that, coupling strength modification factor is increased. Hence the coupling strength is changing faster than in Case 2.

Coupling strength modifications are the result of synaptic plasticity. The synaptic plasticity gradually changes the coupling strength with the time and spiking activities between concerned neuron. Positive synaptic plasticity increases the coupling strength whereas negative synaptic plasticity does the opposite. Positive synaptic plasticity is taken into account for the investigation.

A pertinent question is to understand the reason of emergence of bimodality in MMP of a neuron. MMP is the feature of any neuron describing its potential strength. Its significance makes it crucial to study its every aspect. The experimental results based on simulation show that the probability density function (pdf) of mean membrane potential largely remains unimodal until coupling strength is increased. As the coupling strength starts increasing, the mean membrane potential pdf starts transiting from unimodal to bimodal. After significant number of iterations, the bimodality is distinctively visible. The shift of mean membrane potential from unimodality to bimodality signifies the positive shift of probability of neuronal spiking.

## VI. CONCLUSIONS

The synaptic plasticity governs the coupling strength in between any set of neurons. According to the simulation results, higher the synaptic coupling strength, higher is the chances of bimodality in the membrane potential. In other words, membrane potential, demonstrating the uni-modality in the frequency analysis transits to bimodality as the coupling strength increases.

Despite of sub threshold level of membrane potential initially, the neurons are able to generate spikes due to adequate amount of input charge received from other neurons. The amount of charge transmission may be governed by the coupling strength which is the result of synaptic plasticity. The investigation may further be extended by introducing the negative synaptic plasticity by which the coupling strength may decrease. The dynamics of inter-related negative and positive synaptic plasticity also remain the open ended.

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