# The Neuronal Communication System: Midterm Report

Jeffrey Mehlman
Electrical Engineering Department, Stanford University
Stanford, California 94305
jmehlman at stanford dot edu

## I. PROBLEM STATEMENT

Discovering the complete functionality of the neuron has been a major research effort for nearly 100 years. In recent years, electrical engineers have developed *neuronal networks*. They utilize simple neuron models called ”integrate-and-fire” neurons, which add up weighted inputs and ”fire” an output if the sum exceeds some threshold. In neural networks, multitudes of these model neurons are connected together to perform complicated adaptive tasks. They have been used in a variety of fields.

Recently biophysical research has indicated that the function of a single neuron is much richer than initially thought; each neuron is endowed with internal computational abilities far exceeding that of the simple integrate-and-fire neuron. Each dendritic synapse communicates both with the soma and with other nearby synapses. Additionally, the soma communicates with dendrites for long-term computational functions. We aim to investigate this rich functionality and model it as a **neuronal communication system** in order to learn more about the functionality of the neuron as well as to discover new possiblities for communication protocols.

## II. BACKGROUND & LITERATURE SURVEY

Beginning with Hodgkin & Huxley and their peers in the 1930s [1], it was theorized and then shown that the basis for neuronal communication was electrical in nature. Since then, electrical engineers have been major contributors to neuroscience. An entire field of electrical engineering, called ”neural networks,” has developed out of this interdisciplinary field. Engineers mimic the ability of the brain to learn through simplistic models of neurons and networks of them. Signal processing is one area which has benefited greatly from these developments.

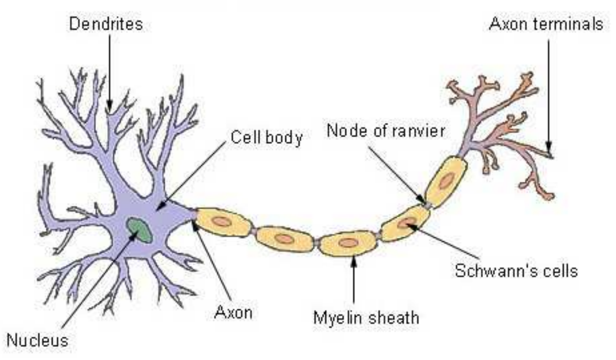

Fig. 1. The Neuron

As shown in Figure 1, a single neuron is comprised of three main sections - a dendritic tree, a soma (cell body), and an axon [2]. The axon generally functions as the ”output” and the dendritic tree takes ”inputs” from other neuron’s axonal outputs. At the interface, the axon terminates in a *synapse*. The space between the synapse and the dendrite is called the *synaptic cleft*. Nearly all of the signaling in the human brain takes places via electrochemical transfer. The axon synapse releases chemicals, called *neurotransmitter*, which migrate across the synaptic cleft and have either an excitatory or inhibitory effect on the dendrite. In the excitatory case, the dendrite generates an electrical current by altering properties of the cell membrane to allow ionic currents to flow into the cell. The size of the electrical pulse seen at the soma falls off with the distance of the synapse from the soma. In the inhibitory case, the dendrite can both alter the properties of the cell membrane to have a shunting effect and suppress any currents generated nearby as well as generate a negative (outgoing) current. These pulses are termed Post Synaptic Potentials (PSPs). One neuron may have thousands of synaptic inputs!

Originally, it was believed that the dendritic tree functioned as a set of passive cables, whereby each input was independently propagated through the tree to the soma and summed linearly there. This model is called the ”point” neuron, and is described mathematically as

$$r = \sum_{i=1}^{n} \alpha_i x_i \tag{1}$$

where $\alpha_i$ and $x_i$ are the synaptic weights and synaptic inputs respectively, and $r$ is the signal seen at the soma [3]. if $r$ exceeds some threshold, the axon hillock generates an Action Potential (AP), which propoagates down the axon as well as back up into the dendritic tree. This latter fact will be discussed in Section III.

In recent years, researchers have discovered that function of the dendritic tree itself is much richer than originally believed. In fact, it has been shown in simulation and through experiment that each dendritic branch functions as a computational subunit which can perform supra-linear summations of dendritic PSPs on that branch [3]–[8]. It is this fact upon which we develop our research efforts.

## III. SYSTEM MODEL

In figure III, this rich functionality is shown as a two-layer neural integration and computational system. Each dendritic branch performs it's own computation, defined mathematically as

$$b_i = s\left(f\left(d_{i,1}, \ldots, d_{i,n}\right)\right) \tag{2}$$

where $b_i, \quad s(-), \quad f(-), \quad$ and $d_{i,j}$ are the output of the $i^{th}$ branch, sigmoidal amplification function, dendritic arithmetic function, and $j^{th}$ dendritic input on the $i^{th}$ branch, respectively. The function s is a sigmoidal function, which determines whether the amplifier for that branch will indeed amplify-and-forward the signal. The function f determines how each set of branch inputs sums together. In most cases, these branches are in fact correlated with each other. A number of efforts have been aimed at developing simple arithmetic rules for this summation which we will leverage for our model.

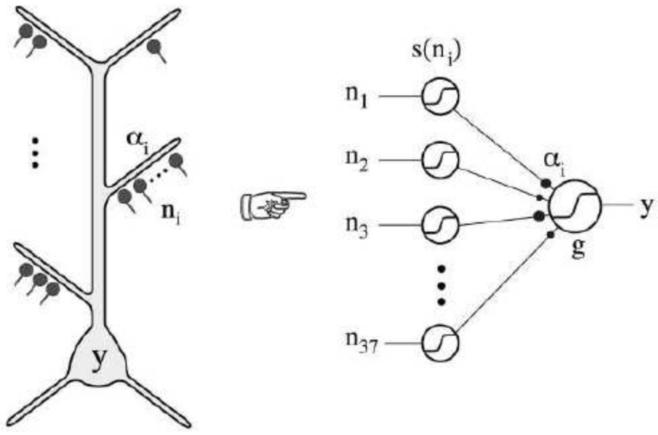

Fig. 2. Single Neuron Computation [5]

Our system aims to model this neuron functionality as a communications network. Each dendritic branch functions as a selection amplify-and-forward (AF) node, by taking the input signals and then deciding if they exceed a given threshold. If they do exceed the threshold, the signal is amplified and forwarded to the collection node (the soma). This amplification is called a ”spikelet.”

We model the nodes as selection-AF because of an additional interesting neural phenomenom. If a given dendritic branch develops a spikelet, and that spikelet leads to a full blown AP at the soma, that branch modifies it's amplification threshold and gain. This is called long term potentiation (LTP) and long term depression (LTD) [6]. Basically, these two phenomena lead to a change in the threshold and amplification gain at each dendritic branch. If a dendritic spikelet causes an AP, that branch reduces it's gain, and vice versa if it does not cause an AP.

This *neural sensor network* will be modeled in MATLAB.

## IV. PROGRESS & REMAINING WORK

The majority of work so far has involved a literature survey in order to become familiar with the latest research in neuronal biophysics. This is in line with the originally developed schedule, which reserved the first portion of this term for a literature study. With our system model in hand, the next step is to develop a MATLAB model which matches the system model derived above.

The next steps are as follows:

- Develop models for dendritic ”nodes,” including LTP and LTD
- Develop model for soma (collector) node
- Obtain neuron morphology and model from Duke/Southampton NEURON library [9]. This repository has a significant number of both complicated and simple neuron morphologies freely available.
- Develop code to parse NEURON morphology model and import to MATLAB as a communication neural sensor network
- Simulate network in MATLAB using the communication network derived above
- Compare to simulation in NEURON environment of actual neuronal biophysics. NEURON is a modeling environment developed specifically for neural research. I have learned how to use this tool as part of a different course (BIO 217).

## REFERENCES

[1] A. Hodgkin and A. Huxley, “A quantitative description of membrane current and its application to conduction and excitation in nerve (reprint),” *Bulletin of Mathematical Biology*, vol. 52, no. 1-2, pp. 25 – 71, 1990. [Online]. Available: http://www.sciencedirect.com/science/article/B6WC7-4GP2506-4/2/8a68ac710fc6dd933c684747f30d96f3

[2] M. F. Bear, B. W. Connors, and M. A. Paradiso, *Neuroscience: Exploring the Brain (Book with CD-ROM)*. Lippincott Williams & Wilkins, March 2002.

[3] P. Poirazi, T. Brannon, and B. W. Mel, “Arithmetic of subthreshold synaptic summation in a model ca1 pyramidal cell,” *Neuron*, vol. 37, no. 6, pp. 977 – 987, 2003. [Online]. Available: http://www.sciencedirect.com/science/article/B6WSS-488M518-C/2/4d8b0a26fb22cc6d9828bfa0cbe57ae7

[4] J. Haoa, X. dong Wanga, Y. Danb, M. ming Poo, and X. hui Zhanga, “An arithmetic rule for spatial summation of excitatory and inhibitory inputs in pyramidal neurons,” *PNAS*, 2009.

[5] P. Poirazi, T. Brannon, and B. W. Mel, “Pyramidal neuron as two-layer neural network,” *Neuron*, vol. 37, no. 6, pp. 989 – 999, 2003. [Online]. Available: http://www.sciencedirect.com/science/article/B6WSS-4CVXF15-1/2/64d4ca3eaa0bc4f91086aa23ebd40a60

[6] Z. Wang, N. long Xu, C. ping Wu, S. Duan, and M. ming Poo, “Bidirectional changes in spatial dendritic integration accompanying long-term synaptic modifications,” *Neuron*, vol. 37, no. 3, pp. 463 – 472, 2003. [Online]. Available: \url{http://www.sciencedirect.com/science/article/B6WSS-47WC1B4-C/2/1e5fe77b84030746b29cd3f1ab65b5af}

[7] N. Spruston, “Pyramidal neurons: dendritic structure and synaptic integration.” *Nature reviews. Neuroscience*, vol. 9, no. 3, pp. 206–221, March 2008. [Online]. Available: http://dx.doi.org/10.1038/nrn2286

[8] A. Polsky, B. W. Mel, and J. Schiller, “Computational subunits in thin dendrites of pyramidal cells,” *Nat Neurosci*, vol. 7, no. 6, pp. 621–627, June 2004. [Online]. Available: http://dx.doi.org/10.1038/nn1253

[9] “Duke / southampton archive of neuronal morphology.” [Online]. Available: http://neuron.duke.edu/cells/