A Carbon Nanotube Cortical Neuron with Excitatory and Inhibitory Dendritic Computations

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***Abstract*—The design of a cortical neuron with carbon nanotube circuit elements that performs nonlinear dendritic computations with excitatory and inhibitory post-synaptic potentials is presented. The inhibitory synapse with controllable parameters that implement plasticity is described in detail. The circuit design was simulated using carbon nanotube spice models. Simulations show that the neuron fires as long as the inhibitory post-synaptic potential is weak or absent. Strong inhibitory post-synaptic potentials prevent the neuron from firing.**

I. Introduction

The cortex is a complex system, and each cortical neuron is itself complex. The scale of the cortex is immense, with an estimated 100 billion neurons interconnected by trillions of synapses. In addition, each neuron performs nonlinear, location-specific dendritic computations on the potentials generated at each synapse. Much of the complexity of the neural behavior is due to the computations involving the post-synaptic potentials (PSPs) arising from the stimulation of excitatory and inhibitory synapses [1]. These potentials combine on the dendritic arbor in complex ways. Dendritic computations include linear, sublinear and superlinear additions and subtractions of postsynaptic potentials depending on the relative locations and nature of the synapses, affecting the probability and the frequency of neural firing.

The complexity of these neural computations presents engineering challenges to the construction of a future synthetic cortex. Of course, a future intelligent synthetic cortex built with neuronal circuits that captured every detail of a biological neuron's physiology would be impractical. Nevertheless, certain aspects of synaptic and dendritic behavior contribute in an important way to learning via short and long-term mechanisms. Capturing those aspects might make it possible to construct a future intelligent synthetic cortex.

A nanotechnological solution could allow the construction of a synthetic cortex containing trillions of synapses. Carbon nanotubes that can behave as metallic wires as well as FETs are a promising technological option. Carbon nanotubes may support the scale of a synthetic cortex, being extremely small (a few nm. in diameter). Current flow is largely ballistic (comparable to the flow of electrons in free space), capacitances are in attofarads, and rise and fall times in picoseconds. Channel resistance is primarily due to the quantum resistance at the junction between the nanotubes and metallic connections, related to the differences in electron energy levels. This creates a challenge for biomimetic neural circuit design since resistance cannot be adjusted easily. Current flow between drain and source is typically increased by using parallel nanotubes, although small adjustments can be made by varying nanotube length. Appropriate interfaces could be used to convert to/from biological signal levels and delays. Finally, nanotubes have been shown to induce minimum immune system reactions in living tissue, making carbon nanotube prosthetic devices desirable [2].

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A central goal of the BioRC project is to construct neural circuits that are biomimetic, that capture variations in neural behavior as a result of learning, and that could lead to a synthetic cortex in the future. We design CMOS circuits to be fabricated, as well as circuits using nanotechnologies like carbon nanotubes for which SPICE models exist, and for which transistor behavior and the operation of simple circuits has been previously demonstrated.

This paper presents The BioRC project's archtypical carbon nanotube neuron design with inhibitory and excitatory dendritic computations. We use carbon nanotubes as circuit elements, and demonstrate the operation of this neuron with SPICE simulations. Carbon nanotube excitatory synapse circuits and their effects on dendritic computations have been a subject of previous publications [3], [4]. The focus of this paper is on the inhibitory synapse and its effect on neural firing, along with the dendritic computations that can be implemented in our neuron. The inhibitory synapse circuit described here mimics the modulation of neurotransmitter quantity and reuptake rate, while exhibiting the variations in hyperpolarizing potential that occur. Shunting inhibition has been implemented as a simplification of the hyperpolarizing inhibition shown here, and will be described in a later publication.

II. Background

The most notable research in neuromorphic engineering includes Meads artificial retina [5]. This significant work originated with Mahowald and Mead [6], followed by Boahen [7], Zaghloul and Boahen [8] and more recently Farquhar and Hasler [9]. Hynna and Boahen report on a circuit that generates a calcium spike with attention paid to exact replication of waveforms, and describe incorporation of the calcium spike circuit in an entire neuron circuit [10]. Some mixed-signal

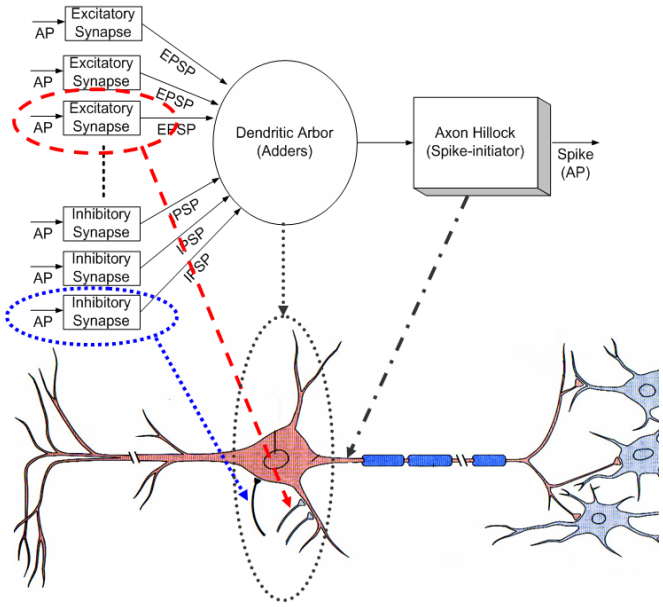

Fig. 1. A system block diagram of the cortical neuron model with a pyramidal neuron cartoon

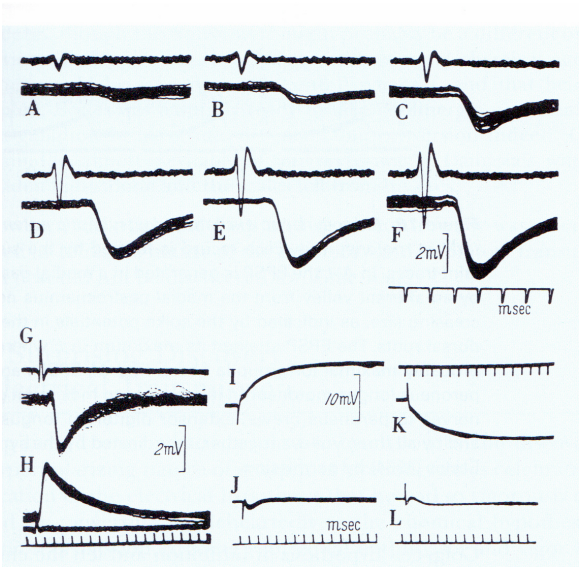

Fig. 2. Biological inhibitory postsynaptic potentials

electronic models close to biological neurons include Liu and Frenzel's spike train neuron, with a 10-transistor mixed-signal synapse [11], and Pans bipolar neuron [12]. An 8-transistor excitatory CMOS synapse [13] is close in scale and nature to our synapses, although they plan to use the synapse for summation of inputs from many pre-synaptic sites.

Electronic inhibitory synapse circuits have been traditionally modeled in combination with excitatory synapses, and have primarily been shunting synapses. Elias was one of the first researchers to propose electronic models for dendritic computations resulting from the interactions between excitatory and inhibitory synapses [14]. Grattarolla et al. simulate a neuron with inhibitory synapses constructed using integrators and comparators [15]. An electronic neuron design with both inhibition and excitation models a bursting oscillator with a depressed synapse constructed of a current mirror and amplifier, with the circuit modulating the output current of the synapse, and the lowest potential being ground [16]. Although they describe an inhibitory synapse it is not clear if that synapse exhibits plasticity. Lee models a central pattern generator that employs an inhibitory synapse [17]. This synapse uses operational amplifiers and multipliers to emulate predefined equations and is extensive in terms of transistors used. Shi and Horiuchi model both shunting and hyperpolarizing inhibitory synapses [18].

III. The Carbon Nanotube Neuron Circuit

Our basic model for a cortical neuron, shown in Figure 1, consists of three types of sub-modules: the excitatory ([3], [4]) and inhibitory synapses, the dendritic arbor [4] and the axon hillock. Circuit models for the dendrites and axon themselves are not shown in this basic model.

A. The Inhibitory Synapse Circuit

The biological inhibitory synapse itself has a complex structure [19] [20]. Inhibitory synapses can be shunting, effectively vetoing depolarization caused by excitatory postsynaptic potentials, or hyperpolarizing, subtracting potential from the dendrite. The modulation of quantity of neurotransmitters released at the presynaptic terminal affects the actions of the receptors on the postsynaptic side that control ligand-gated ion channels, resulting in a variation in hyperpolarizing potentials across the cell membrane. The rate that neurotransmitters are reuptaken into the presynaptic terminal also affects synaptic behavior by delaying the fall in postsynaptic potential. Example biological IPSP measurements are shown in Figure 2 [20].

The work presented here is based on the biomimetic behavior of a hyperpolarizing inhibitory synapse circuit designed to be compact, with correspondence between biological mechanisms and circuit structures. This synapse circuit evolved from an earlier excitatory synapse [4]. Synapse behavior is controlled by voltages on the gates of the transistors, acting as control knobs. The neurotransmitter concentration and the spread of the IPSP (delay of return to resting potential) can be varied by controlling the neurotransmitter release and reuptake rates. The synapse also exhibits temporal summation of the IPSPs when action potentials impinge on the synapse at close intervals. This circuit models cell potentials and neurotransmitter concentrations with voltages, along with the correspondence between circuit elements and biological mechanisms.

Figure 3 presents the inhibitory synapse circuit that displays plasticity. The design is segmented into parts that facilitate biomimetic behavior corresponding to biological mechanisms. The action potential impinges on two sections of the synapse as shown, namely the neurotransmitter section and a delay mechanism (*delay 1*) that delays the reuptake of neurotrans-

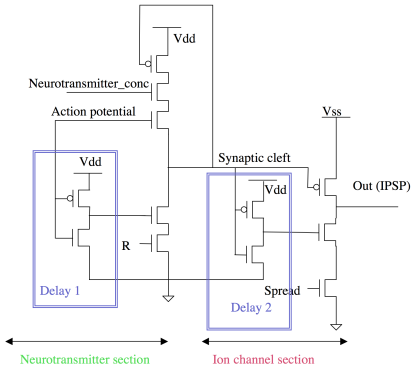

Fig. 3. The Carbon Nanotube Inhibitory Synapse

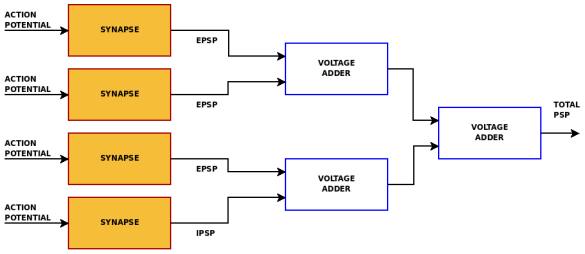

Fig. 4. The Dendritic Arbor Portion

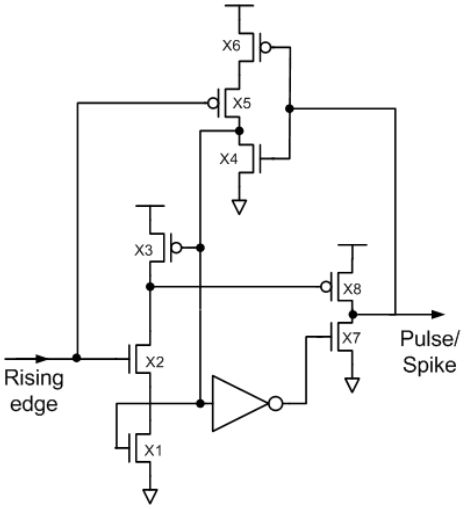

Fig. 5. Circuit diagram of the axon hillock module

mitters. The pull up transistor in the Neurotransmitter section controls the actual neurotransmitter concentration in the synaptic cleft, modeled by the voltage at the synaptic cleft node, whereas the pull down transistor models the reuptake mechanism that controls the drop in neurotransmitter concentration in the cleft. The chronological occurrence of reuptake is controlled by the rise time of the delay circuit, by varying the length of its PMOS transistor to indirectly control the falling RC time constant of the neurotransmitter concentration. The neurotransmitter release cause one or more ion channels to open; hyperpolarization is modeled by the pull down transistor in the ion channel section tied to negative potential (Vss). The ion flow responsible for the rise of the IPSP to the resting potential is modeled by the pull up to ground in the same section. The time delay between the negative peak of the IPSP and its rise up to ground potential is modeled by a second delay circuit (*delay 2*) that is tunable to vary the synapse properties. Variation in neurotransmitter concentration in the synaptic cleft causes a change in the IPSP negative peak amplitude thus directly altering the synapse strength [20]. Also the reuptake mechanism *R* and *spread* input control the spread of the IPSP, which modulates the temporal summation of the synapse output when action potentials impinge on the synapse or multiple synapses are stimulated at close intervals. The voltage across the gate labeled *neurotransmitter_conc* controls the current that models the neurotransmitter release while the voltage across the gate *R* controls the reuptake. Varying these two voltages controls the IPSP amplitude and the spread of the IPSP respectively.

B. The Dendritic Arbor

The adder circuit [21] is shown in an earlier publication [4]. A block diagram of the dendritic arbor portion is shown in Figure 4.

There are four synapses (three excitatory and one inhibitory) in the arbor, each on a separate dendritic branch. Our axon hillock circuit is shown in Figure 5. In a biological neuron, the axon hillock has the highest density of sodium channels, resulting in the lowest threshold (-55mV compared to elsewhere in the neuron) to initiate an action potential. If the summation of post-synaptic potentials (PSPs) reaches a threshold value, the axon hillock circuit will generate a spike. This circuit behaves in a similar fashion to a self-resetting CMOS circuit, receiving a rising edge and producing a pulse whose width is controlled by the gate delay of the inverter shown in Figure 5. To mimic a fast rising phase (due to the rapid increase of the sodium channel conductance) and a slower falling phase (due to the slower increase of the potassium channels conductances) of an action potential, we adjusted the pull-up and pull-down strength of transistors X8 and X7. All the other transistors were tuned to model the time courses (time constants) in the dynamic mechanisms of the voltage gated ion channels.

In our archetypical biological neuron, potentials range from around -75mV to +40mV with action potentials peaking around +40mV. Since the carbon nanotube neuron is designed to operate with Vdd around 0.9V as the peak action potential voltage, and with 0.0V (Ground) as the resting potential, the post-synaptic potentials were scaled accordingly, with 0.0V circuit potential corresponding to -75mV biological potential and 0.9V circuit potential corresponding to 40mV biological potential. Likewise, we scaled the delays with about 1 ms in the biological neuron scaling to about 10 ps in the nanotube neuron [22]. The postsynaptic potential appearing at the den-

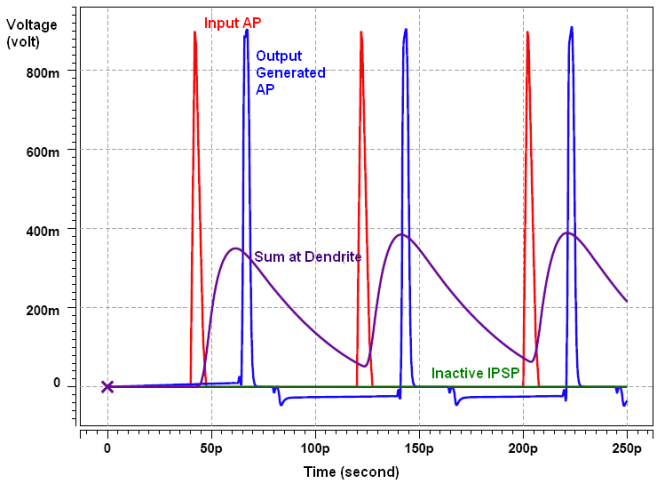

Fig. 6. Firing of the neuron with inhibitory synapse inactive

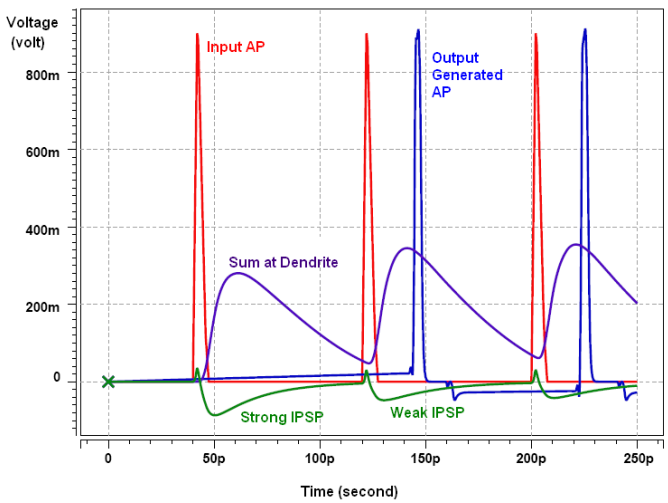

Fig. 7. Firing of the neuron with weak and strong IPSP

dritic trunk is approximately 14% of the action potential and the duration is about 6 times as long as the action potential, similar to EPSPs described in the literature. It should be noted that the massive fanin and fanout of cortical neurons are sufficient to significantly slow operation of artificial cortical neurons in practice.

IV. Experiments With The Cortical Neuron

We performed several experiments. The neuron was tested with action potentials input to each synapse, and the output of the neuron measured. The input action potential (the red trace) is applied to each synapse module and the generated action potential (the blue trace) is captured at the axon-hillock node. First we tested the neuron with an inactive inhibitory synapse (Figure 6). Second, we show that when the inhibitory synapse is strengthened, it prevents the neuron from firing since the summation of PSPs is below the threshold to initiate a spike at the axon hillock (Figure 7). Third, again shown in Figure 7, we repeated the previous experiment with the strength of the Inhibitory synapse set to a low value using the neurotransmitter concentration knob, showing the neuron firing in spite of the small IPSP generated.

Many other experiments have been performed with variations in neurotransmitter concentration, receptor variations and non-linear dendritic computations. These experiments demonstrate the behavior of our excitatory and inhibitory synapses, and are a subject of future work.

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