Noisy Neuromorphic Circuit Modeling Obsessive Compulsive Disorder

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Abstract—The CORTICO-STRIAL-THALAMO-CORTICAL (CSTC) circuit in the brain has an important role in controlling movement and thought. As a consequence, any dysfunction in this circuit may cause movement and psychological disorders. For example, one hypothesis to explain Obsessive Compulsive Disorder (OCD) is that alteration in this circuit causes a positive feedback loop (direct pathway) to become strengthened and a negative feedback loop (indirect pathway) to become weakened, which causes hyperactivity in the cerebral cortex resulting in OCD symptoms. We have designed and simulated a biomimetic electronic circuit with a Carbon Nanotube synaptic layer and CMOS neurons that mimics the CSTC loop in healthy and OCD conditions. This circuit simulation demonstrates the utility of neuromorphic circuits in brain modeling, since varied behavior can be demonstrated via modification of synaptic strengths. We further demonstrate the role noise (variability) plays in the CSTC circuit function and dysfunction and describe a noise-generating circuit implementation to inject noise into neuromorphic circuits based on photons emitted by an LED.

I. INTRODUCTION

Obsessive Compulsive Disorder (OCD) is prevalent throughout the world, and the rate of occurrence is thought to be between 1 and 2 percent. While models of this disorder have been proposed, demonstration of the models using electronic circuits has not appeared in the research literature. The BioRC project [1] has a library of neuromorphic circuits that support modeling both typical and atypical neural circuits. Demonstrating simulations that exhibit disorder states is one current focus of the BioRC group [2], and OCD is an excellent candidate for inclusion in these experiments.

OCD is a debilitating neuropsychiatric condition characterized by intrusive distressing thoughts (obsessions) and/or repetitive ritualized mental or behavioral acts (compulsions), normally acted on to alleviate anxiety [3], [4]. There are numerous symptoms of OCD [5], [6], some related to obsession and some to compulsion. Behaviors characterized by obsession include excessive doubt about task completion, fear of contamination or uncleanness, need for symmetry or exactness, fear of causing harm to others, excessive concern over right and wrong, and intrusive inappropriate sexual thoughts. Compulsive behavior includes excessive or repeated checking, washing/cleaning, counting, ordering/arranging, repeating, hoarding or praying.

The CORTICO-STRIATAL-THALAMO-CORTICAL (CSTC) circuit controls thought and movement as a response of the brain to external stimuli. Lesions in this area, dysfunctional behavior of neurons, or imbalance in excitation and inhibition can cause movement disorders, neurological symptoms, and even psychological disorders. For example, in non-human primates, blocking the activity of the sub-thalamic nucleus (STN) produces a movement disorder similar to that seen in human hypokinetic and hyperkinetic movement disorders like Parkinson’s disease and Huntington’s disease [7], [8]. One explanation for Obsessive Compulsive Disorder (OCD) is that neurons in the thalamus and cortex keep firing due to a repetitive excited loop and SNr, a major output structure of the basal ganglia, becomes unable to inhibit the thalamus and slow down the hyperactivity. This hyperactivity is believed to cause OCD [3].

Here, we have designed a biomimetic circuit that mimics the mechanism of OCD. Our circuit has a hybrid structure. Excitatory synapses and inhibitory synapses have been designed using circuits containing Carbon Nanotube (CNT) transistors and neuron neuromorphic circuits have been designed using CMOS 45nm technology transistors.

II. THE HYBRID ELECTRONIC CORTICAL NEURON

![Fig. 1. Structure of our Hybrid Neuron](image1)

![Fig. 2. Two layer neuromorphic circuit, Carbon synapses on top and Silicon neurons below](image2)
The human brain has around 100 billion neurons. Each one of these neurons can have an average of 10,000 up to 100,000 synaptic connections to it from other neurons. \(^1\) Realizing synapses with a nanolayer on the surface of the CMOS die is used here to enable complex neural networks to be fabricated. Synapses are designed with CNT transistors. CNT technology is a suitable choice for the synaptic layer in neuromorphic circuits due to the lower parasitics of CNTs, lighter weight and biocompatibility, while CMOS is used for dendritic potential adders and axon hillocks that require complicated computations. There are emerging applications for this hybrid architecture in nanotechnology. Hybrid architecture combines inherent advantages of both CMOS and CNTs. It uses CNT transistors as sensors and CMOS as complex electronic control and processing units \([9]\), while supply voltages are identical for both technologies. There are techniques for independent VLSI fabrication and CNT processing so that the CMOS portion can exploit advantages of VLSI scaling like cost, flexibility and predictable performance \([10]\). While demonstration of hybrid structures is not relevant to demonstrating the OCD mechanisms via simulation, such combined technologies might make prosthetic devices more feasible in the future.

Figure 1 shows our hybrid electronic neuron and figure 2 shows the architecture of our neuromorphic circuit.

### III. Synapses and Noisy Neuron Transistor Circuits

The BioRC group has designed synapses with CNT transistors \([11]\) whose simulation models are used in this research. The adder \([12]\) is used as a Dendritic Arbor compartment with CMOS technology. Three of these adders are used in a two stage configuration to make a four input adder.

Figure 3 shows the Axon Hillock that is used in the circuit with CMOS technology.

The input to each synapse is an action potential (AP) and the output is a postsynaptic potential (PSP). Synapses can be excitatory or inhibitory. Excitatory synapses receiving an input AP signal produce a positive voltage which is called an excitatory postsynaptic potential (EPSP) and inhibitory synapses receiving an AP input produce a negative voltage which is called inhibitory postsynaptic potential (IPSP). In living neurons, amplitudes of these PSP signals can be altered which means that synapses can be weakened or strengthened by changing neurotransmitter availability or receptor concentration. We mimic this alteration in this case by changing the receptor concentration voltage knobs on the CNT synapse circuit. Every neuron in our circuit is connected to four synapses. If the algebraic summation of PSP signals to a neuron exceeds the firing threshold of that neuron, it will fire and produce an AP otherwise it will ignore the stimulus. Therefore, these particular neurons have all or none response to input spikes. Synaptic connections between neurons and strengths and types of synapses can exhibit different behavior in biological nervous systems and similarly in our neuromorphic circuits.

Neurons have stochastic behavior and their firing rate and the times they fire are random variables. In the BioRC group, different types of approaches have been taken to induce noise in the neuron to imitate biological neurons with stochastic behavior, including variable-response synapses and variable-threshold axon hillocks \([14][15]\). Here, we connect our bio-mimetic neuron to noise sources that have been generated externally to observe noise effects at a higher level (neural network). Also, we introduce an electronic noise generator circuit that can be used in neuromorphic systems.

### IV. A Noise Generator for Neuromorphic Circuits

Noise affects the nervous system at every level, from the receiving sensory signals to the sending motor response signals\([16]\). Most of noise is simulated as a true random Poisson or Gaussian process to fit naturally occurring biological variations \([17]\). However, in modern digital systems, randomness usually generated by a look-up table or linear feedback shift register and true random seeds \([18]\), which is actually pseudo-random and may have problems with speed, power, and area cost. The digital nature of the signals may also cause accuracy issues when used with analog neurons. Moreover, the true randomness of the memristor, which shows a logarithm distribution, is insufficient for modeling neural noise. \([19]\)

We present the use of a CMOS-compatible true randomness generator using an optoelectronic device as a noise source for neuromorphic components in CMOS circuits \([20]\). Photons generated by an LED as a provably random quantum process provide a way to realize a random pulse generator (RPG). Related work has been widely reported. In neuromorphic circuits, independent randomness is important for noise to be a significant factor in neural information processing. Thus, in extensive neural networks, a large number of RPGs integrated in circuits can be expected, and, as a result, the RPGs must be power and area efficient.

The RPG device consists of three parts: LED, single photon avalanche diode device (SPAD), and waveguide. This structure has been experimentally verified by others \([21]\). Based on this structure, we assume a red LED and single photon avalanche diode device to implement an on-chip random pulse generator. The cross-section of the device is shown in Figure 4. Through the the platform SOLES \([22]\), the LED can be compatible with CMOS technology.

The novel SPAD structure was emulated in SPICE using a CMOS 180 nm process \([23]\). A planar pn junction is biased
above breakdown in the core of SPAD, thus operating in Geiger mode. In this regime of operation, electron-hole pairs generated by photons can stimulate an avalanche breakdown by impact ionization. Because conventional avalanche photodiodes operate just below the breakdown voltage, the optical gain of the SPAD is high enough to enable single-photon sensing. Each photon absorbed in the active region can allow a large current to flow through the SPAD. In the 180nm CMOS circuits, both active and passive avalanche quenching schemes can be integrated.

A. Discrete Noise Signals

LE diodes are direct band-gap devices that produce incoherent light by spontaneous emission, essentially a random process. If operated at sufficiently low power, a LED emits photons that are virtually independent of each other. This photon emission is a Poisson process and the wavelength of photons is a Gaussian distribution. The exponentially-distributed time intervals of the Poisson-process photon emission and the photon loss in the waveguide are neglected, because the quenching time of SPAD is much longer.

In this RPG model, the output is uniformly-distributed discrete pulses with Gaussian-distributed amplitude in uV. To use these signals in our neuromorphic circuits, we have to amplify it to mV pulses. The complete circuit of the discrete noise generator is shown in Figure 5.

Fig. 5. Discrete noise generator circuit

The photons are simulated as a voltage response in SPAD. The strength of photons depends on the wavelength, which is Gaussian random. The photon detection efficiency of SPAD is also a Gaussian process related with wavelength. Thus we choose 615 nm peak wavelength and $\sigma = 30$ for both LED and SPAD as the simulation parameters. The simulation results of simulated photon voltage and noise signals are shown in Figure 6. The random pulses in the figure are the output of the noise generator, and the trace below shows the simulated photon voltage used as input to the SPAD.

Fig. 6. Discrete noise signals

The above noise signals only include positive pulses. In neural systems, the noise can be both positive and negative [16]. To accomplish it with our RPG, we use two RPGs and connect them to one amplifier. The output can be calculated with Equation:

$$V_{out} = -A(V_1 - V_2) + V_1$$

The corner situation is the two inputs having the exact same phase, so that the pulses could cancel each other. But if the amplitude of the two signals is different, it allows the circuit to generate robust random positive and negative pulses (Figure 7).

Fig. 7. Corner situation of random pulses with positive and negative values

B. Continuous Noise Signals

To generate continuous noise signals, we connect the RPG to a current mirror. (Figure 8) The charge accumulates at the output, and gradually is discharged by the leakage signal. Thus
we create a continuous noise signal (Figure 10a), but in this manner, the noise cannot be negative. The solution is the same as the method we used for discrete noise signals. Two continuous signals are connected to an amplifier (Figure 9), one of the signals is inverted and the output is the summation of inputs. The final result of the continuous noise is shown in Figure 10b. This noise-generation strategy can be incorporated into the neurons with noisy inputs, as described next.

Fig. 9. Circuit for continuous noise

Fig. 10. Continuous noise signal and source random pulses signal

V. BIOLOGICAL OCD CIRCUIT AND ELECTRONIC MODEL

Figure 11 (a) shows a simplified block diagram of a mouse brain. It shows the loop between cortex, striatum (which is a part of Basal ganglia), and thalamus. In a healthy condition, the striatum inhibits GPe, and therefore GPe stops inhibiting STN. Then, STN excites GPi/SNr and GPi/SNr inhibits the thalamus. The indirect pathway on the right in Figure 11 (a) acts like a brake and inhibits hyperactivity in the thalamus and cortex. If the direct pathway on the left becomes strong, it will inhibit GPi/SNr and therefore the thalamus becomes hyperactive. The level of thalamus and cortical activity is controlled by a "gas-brake" mechanism of direct and indirect pathways [3]. Figure 12 also shows the simplified block diagram of the two loops that control response of brain to coming sensory signals. All sensory signals (except Olfactory) first go to the thalamus and from the thalamus they get distributed to corresponding regions in the Cerebral Cortex. The block diagram of our circuit design is shown in Figure 11 (b). D1 is the neuron that inhibits GPi/SNr and accelerates the activity of the thalamus. D2 is the neuron that excites GPi/SNr through an indirect pathway, inhibiting the thalamus and reducing its activity. GPi/SNr is connected to the thalamus through inhibitory synapses, inhibiting the thalamus and consequently the cortex.

It should be noted that many neurons are involved in the biological circuit modeled here, while we have simplified the circuit to single neurons in each brain region. More-extensive neuromorphic circuit models can be constructed to model more detailed behavior. We ran simulations for this OCD circuit to demonstrate the overall behavior of the circuit when certain neurons D1 and D2 have weak or strong synapses, and we also introduced noise to break the OCD loop. We show details of the simulations in the following scenarios.

VI. TYPICAL AND ATYPICAL RESPONSES

Figure 13 shows three different firing scenarios (A, B, and C) showing AP output (Action Potential) of all neurons. Thalamus receives external excitation that mimics sensory inputs. This external excitation, which is not shown in the simulation, causes thalamus to fire, unless it is inhibited. GPe and STN also are connected to external excitation and similar to thalamus they will keep firing unless inhibited. In other words, two synaptic inputs of the neurons representing these three regions (thalamus, GPe and STN) are excited by a spiking voltage source that mimics sensory signals.
A. Indirect pathway and direct pathway are both weak

The thalamus keeps firing unless inhibited, and each time the thalamus fires it excites the cortex. The cortex tries to excite neurons D1 and D2 in direct and indirect pathways but synapses connecting the cortex to D1 and D2 are weak and cannot cause them to fire. Therefore during scenario A, D2 does not fire. Since it does not fire, it cannot inhibit GPe. GPe fires unless inhibited, therefore during scenario A, GPe keeps firing. STN fires unless inhibited. During scenario A, GPe keeps inhibiting STN and therefore STN does not fire. Since STN does not fire, it cannot excite GPi/SNr. GPi/SNr does not fire and consequently cannot inhibit the thalamus. Thalamus fires unless inhibited, and therefore during scenario A it fires and causes cortex to fire in hyperactive mode. Figure 15 shows the simulation result for scenario A when the other two inputs to neuron D2R are connected to a white noise source with zero mean and 30 mV rms value. Occasional firings of D2R due to noise can interrupt hyperactivity of the cortex.

B. Indirect pathway is strong

Excitatory synapses connecting cortex to D2 have been strengthened and therefore each time the cortex fires, it causes D2 to fire. D2 starts inhibiting GPe. Since GPe does not fire, it stops inhibiting STN. STN fires and excites GPi/SNr. GPi/SNr inhibits the thalamus. The thalamus stops firing, therefore it does not excite the cortex. The thalamus and cortex do not fire for some time. Since the brake mechanism is indirect, it does not brake the cortex immediately. Since the cortex does not fire it cannot cause D2 to fire. D2 does not fire and GPe starts firing, and STN stops firing. This means that GPi/SNr will not inhibit the thalamus, the thalamus will fire again and will excite the cortex. So, even if scenario B lasts for some time, the thalamus and cortex will show activity, but this negative feedback mechanism reduces the hyperactivity and firing burst of the thalamus and cortex which is shown in Figure 16.

C. Both pathways are strong

In this scenario, the indirect pathway is strong but the direct pathway is stronger and D1 fires each time that the cortex fires. So, the cortex inhibits GPi/SNr. GPi/SNr cannot fire even
neurological disorders. In the brain can be modeled by neuromorphic circuits. These one neuron, it shows that healthy and unhealthy conditions additional physiological behaviors. Even though this circuit is neurotransmitter availability and reuptake rate, to demonstrate synaptic strengths to demonstrate more complex behaviors. can be used to probe further by strengthening and weakening that can be used in neuromorphic circuits to mimic biological breaks a repetitive loop. Also, we proposed a noise generator controls the level of activity in the brain and how noise can OCD, and simulations show how a “gas-brake” mechanism during scenario C, $D_2$ and $STN$ keep firing. However, $GPi/SNr$ does not fire and the thalamus shows atypical hyperactivity.

VII. CONCLUSION

We have designed a circuit with a CNT synaptic layer and CMOS neurons that mimics hyperactivity in the cortex linked to OCD, and simulations show how a “gas-brake” mechanism controls the level of activity in the brain and how noise can break a repetitive loop. Also, we proposed a noise generator that can be used in neuromorphic circuits to mimic biological noise and its role in biological behavior. This circuit simulation can be used to probe further by strengthening and weakening synaptic strengths to demonstrate more complex behaviors. Varying many more mechanisms in the neuromorphic circuits is possible, including firing threshold, receptor availability, and neurotransmitter availability and reuptake rate, to demonstrate additional physiological behaviors. Even though this circuit is oversimplified and regions in brain are represented by only one neuron, it shows that healthy and unhealthy conditions in the brain can be modeled by neuromorphic circuits. These kinds of circuits can be helpful in analyzing psychological and neurological disorders.

REFERENCES


