

A Directionally-Selective Neuromorphic Circuit Based on Reciprocal Synapses in Starburst Amacrine Cells

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Abstract—Starburst Amacrine Cells (SACs) play a major role in the detection of directional motion in the biological retina. The starburst amacrine cell has intrinsic electrical mechanisms for producing directional selectivity (DS). GABA transmitter-receptor interactions between two overlapping SACs make DS more robust. We present a compartmentalized CMOS neuromorphic circuit that models a portion of two biological starburst amacrine cells in the retina and includes a simplified model of reciprocal interaction between the dendritic branches of SACs. We demonstrate that a neuromorphic circuit incorporating the reciprocal synapses enhances the responses in the neuromorphic dendritic tip and generates robust directional selectivity.

Index Terms—Neuromorphic Circuit, Directional Selectivity, Starburst Amacrine Cell, Reciprocal Synapse, Retina, Motion.

I. INTRODUCTION

Starburst Amacrine Cells (SACs) play a major role in the detection of directional motion in some mammalian retinas. Neuroscientists' experiments suggest that SACs perform the initial neural computations that induce directional selectivity in the ganglion cell. The computation of directional selectivity (DS) occurs at individual dendritic branches of each SAC and each branch acts as an independent computation module [1]. Both the dendritic calcium signal and membrane voltage in the dendritic tip generate a stronger response with the light stimuli moving from the soma towards the dendritic tip (centrifugal motion) than moving in the opposite direction (centripetal motion) [1]. In this paper, we present a neuromorphic circuit that emulates the directional selectivity response in biological SACs. Modeling this aspect of the mammalian vision system and cortex could be useful for service robots because early and rapid detection of moving objects is essential for safe, reliable operation of the robots, and has not been incorporated in this manner in other robotic vision systems to date.

II. BACKGROUND

The starburst amacrine cell, with a characteristic radially-symmetric morphology, is thought to provide directional inhibitory input to direction-selective ganglion cells (DSGCs) [2] [3] [4]. SACs receive glutamate release from bipolar cells (BCs). Furthermore, the SAC dendritic tip releases and

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receives the GABA neurotransmitter. To explain the DS observed in the SACs, neuroscientists have proposed at least two fundamentally different mechanisms [5]: dendrite-intrinsic electrotonics [6] [7] and lateral inhibition [8] [9]. Euler et al. demonstrated that the intrinsic electrical mechanisms of SACs may produce DS without inhibitory network interactions [5]. However, lateral inhibition between two SACs may enhance the difference in response and generate a robust directional selectivity [9]. They also found that SACs may have directional responses even if GABA inhibitory interactions between the SACs are blocked pharmacologically [1]. A centrifugal (CF) motion generates an in-phase response that is summed effectively with the response in the distal compartment. However, centripetal (CP) motion generates an out-of-phase response that is not summed effectively.

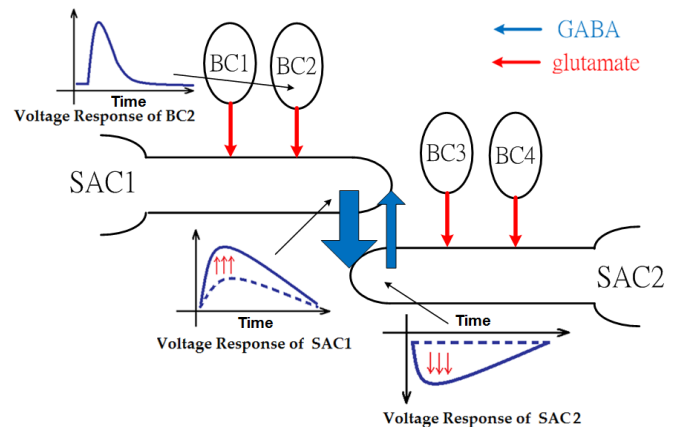


Fig. 1. A reciprocal synapse between two SACs may enhance the difference in responses of the distal tips of the two SACs' dendrites.

As long as the processes of two neighboring SAC overlap, they are likely to form reciprocal connections in a positive feedback loop. In Figure 1, when the light moves to BC2, the dendritic tip of SAC1 produces a voltage response and releases more GABA which inhibits the response of dendritic tip in SAC2. SAC2 in turn produces less GABA release which enhances the response of the dendritic tip in SAC1.

Research on silicon retinas, beginning with Mahowald and Mead [10] and followed by Zaghoul and Boahen [11] as well as others, involves human vision, which is not thought

to involve directional selectivity in the retina [12]. Human artificial retinas are in clinical trials as well [13]. Andreou and Strohhahn designed an analog VLSI processor for computer vision based on the Hassenstein-Reichardt-Poggio models for information processing in the visual system of a fly [14]. In their model, the delayed photoreceptor responses may correlate with the current responses of neighboring receptors and perform directional selectivity. Liu’s neuromorphic vision chip was also inspired by motion computation in the fly’s visual system [15]. However, the fly doesn’t have SACs in the retina. Benson and Delbruck proposed a silicon retinal model for direction selectivity [16], using inhibitory connections in the null direction to perform direction selectivity. They included only photoreceptor cells and direction-selective ganglion cells (DSGCs) in their model and omitted SACs, simplifying the direction selectivity in the retina. Etienne-Cummings assumed primate motion detection is performed in the cortex and modeled insect and primate visual motion detection in hardware implementations [17]. His comparison does not include any designs having SACs. Wang and Liu designed an analog VLSI network using spiking neurons for motion detection [18] which is based on the model proposed by Rao for explaining the formation of direction- and velocity- selective cells in the visual cortex [19]. Since they are modeling the cortex and not the retina, they do not include SACs in their circuits.

III. CIRCUITS MODELING STARBURST AMACRINE CELL PORTIONS, WITH A RECIPROCAL SYNAPSE

We constructed a compartmentalized neuromorphic circuit for modeling portions of two biological starburst amacrine cells (SACs) found in the retina. To emulate the DS response of each SAC, our circuit model incorporates two major mechanisms, dendrite-intrinsic electrotonics and lateral inhibition. We emulated the dendrite-intrinsic electrotonics by including the propagation of membrane voltage along the dendritic branch of a SAC. With regard to lateral inhibition, we incorporated GABA transmitter-receptor interactions between the two overlapping SAC portions in our circuit. The positive feedback of lateral inhibition behaves as expected in our circuit simulations and appears to be stable. We describe the circuit and analyze its stability in detail in the next sections.

Figure 2 shows two overlapping dendritic branches of two simplified biological SACs and a block diagram of the corresponding circuit implementation. Each branch of the SAC model consists of an intermediate compartment and a distal compartment. The soma is not modeled. Both compartments receive glutamate inputs from our bipolar cell circuits (not described here) in the form of voltage waveforms. The signals first propagate through the wave-shaping circuits that convert the glutamate input voltages into cation concentration represented by voltages inside the SAC cell. The cation concentration voltage at the intermediate compartment propagates to the distal compartment through a delay circuit and is summed with the cation concentration voltage at the distal compartment. While the propagation is bi-directional, we include propagation only towards the dendritic tip for this

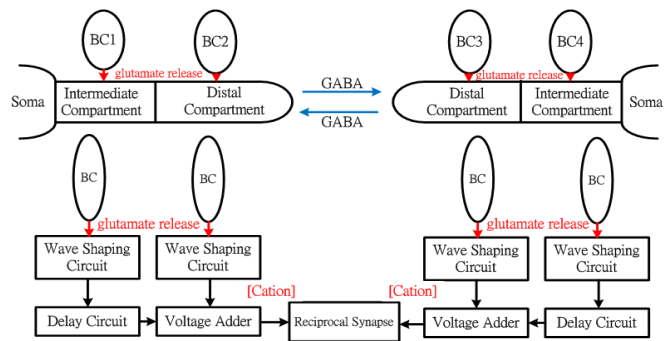


Fig. 2. The top diagram represents two SACs interacting through a reciprocal synapse. The bottom diagram depicts the correspondence in our circuit implementation. The somatic compartments and signal propagation toward soma are not being modeled in our circuit.

simplified SAC because the dendritic tip is where GABA is released. The summation is implemented using a voltage adder. In the reciprocal synapse circuit shown in Figure 3, the subscript of the parameters for the left SAC is 1 and the subscript of the parameters for the right SAC is 2. Here, we use only the names of the parameters without the subscripts. The output of the voltage adder labeled $[Cation]$ represents intra-cellular cation concentration at the distal compartment. The membrane potential of the distal compartment labeled by V_d is positively modulated by $[Cation]$ and negatively by $GABA_{IN}$. $GABA_{IN}$ represents the effect on the distal compartment of the GABA release from another SAC. $GABA_{release}$ is represented by the voltage output that models GABA release and is modulated positively by V_d and negatively by $GABA_{reuptake}$. The voltage adder circuit modified from [20] performs non-linear summations of intra-cellular cations. In the wave-shaping circuit, the rise of glutamate induces more current which charges the output capacitor C quickly to $(V_{glutamate} - V_{th})$, where V_{th} is the threshold voltage of the transistor. The pull-down transistor provides a resistive path for discharging output capacitor C when glutamate input decreases. Therefore, the wave-shaping circuit produces an output with smaller response and longer duration than the input. The delay circuit uses a current-mirror structure to model the propagation delay along a dendritic branch of the SAC.

IV. SIMULATION EXPERIMENT CONFIGURATIONS

Figures 2 and 3 illustrate the scenario to perform the simulation experiments and the circuits we used respectively. Consider the case in which $[Cation]_2$ remains the same and $[Cation]_1$ increases. At the outset, both V_{d1} and $GABA_{release1}$ increase. The increase of $GABA_{release1}$ pulls down V_{d2} and $GABA_{release2}$. The decrease of $GABA_{release2}$ pulls up V_{d1} . As a result, V_{d1} is increased due to the positive feedback loop. During this operation, transistor M8 enters the linear region as $GABA_{IN2}$ increases. Therefore, the gain of the amplifier consisting of M8 and M7 decreases. Meanwhile, transistor M2 enters the subthreshold region which allows V_{d1} to increase more quickly (transistor M1 is in the saturation

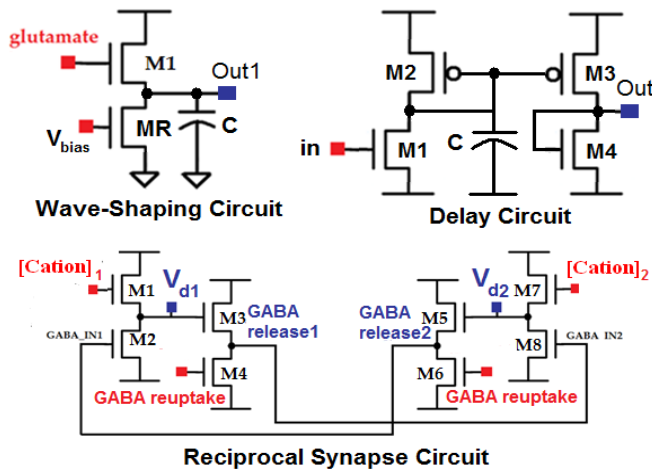


Fig. 3. Wave-shaping circuit, delay circuit, and reciprocal synapse circuit.

region). However, the amount of increase is limited by the decrease in gain of the amplifier consisting of M8 and M7. Eventually, the whole loop reaches a stable state without divergence or oscillations. V_{d1} is still proportional to the amplitude of $[Cation]_1$. We conclude that the SAC design with the reciprocal synapse still appears to possess the property of graded potential output and the operation appears to be stable.

The simulations were conducted with TSMC 18 CMOS (180nm) technology using SPECTRE with two branches (Figure 2) and also with only one branch. To demonstrate the dendrite-intrinsic electrotonics of the SAC design, we applied moving stimuli, centripetal motion and centrifugal motion, to the configuration with only one branch and measured the responses of the distal compartment for both cases. The results are plotted in Figure 4. The glutamate inputs are the graded potentials generated by an outer-retina circuit including bipolar cell circuits we designed earlier. The moving stimulus from the intermediate compartment to the distal compartment will first evoke a response at the intermediate compartment. After some delay, the signal reaches the distal compartment. Meanwhile, the moving stimulus has reached the distal compartment and the evoked response is therefore summed with the response from the intermediate compartment. This results in a larger voltage response at the distal compartment than at the intermediate compartment. For the opposite moving stimulus, the response is not summed optimally because the signal from the intermediate compartment cannot reach the distal compartment on time. The simulation shows that the stimulus moving centrifugally evokes a stronger response than moving centripetally.

To demonstrate lateral inhibition between the SACs, we tested the two configurations and compared their responses. We applied a stimulus moving from the intermediate compartment to the distal compartment to both configurations. We measured the responses of the distal compartments (Figure 5). The results visually indicate that the positive feedback of the reciprocal synapse effectively enhances the response of the

distal compartment in our circuit simulation.

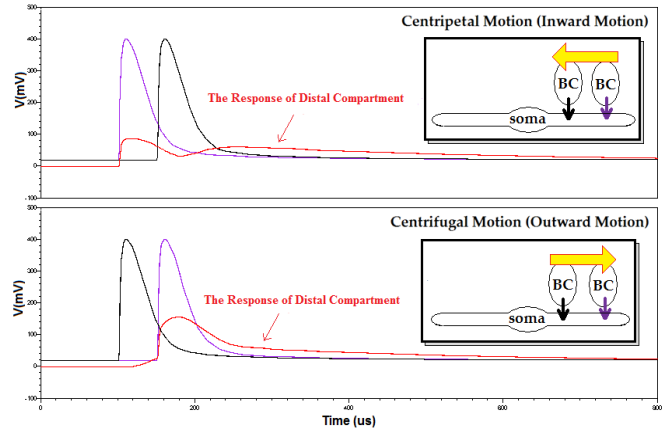


Fig. 4. The simulation results of a single SAC dendritic branch with respect to both centripetal and centrifugal motion. The black trace and purple trace represent the inputs to the simulation that are the outputs from the bipolar cells connecting to the intermediate compartment and distal compartment respectively. The red trace is the response of the distal compartment.

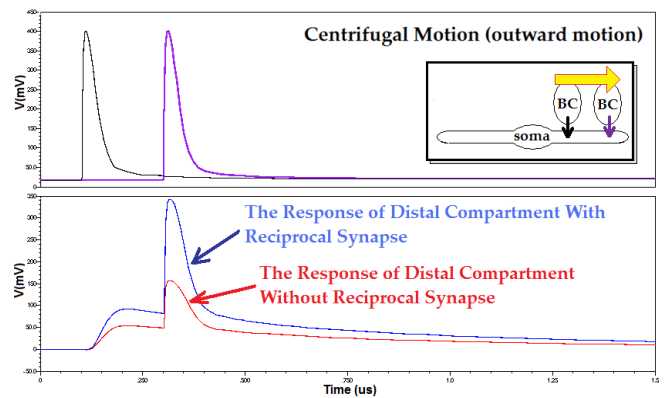


Fig. 5. Comparison of the simulation results both with and without reciprocal synapse to centrifugal motion. The black trace and purple trace represent the inputs to the simulation that are the outputs from the bipolar cells connecting to the intermediate compartment and distal compartment respectively.

To characterize the circuit behavior, we applied the moving stimulus at different speeds and different input intensities. We observed the amplitude of the response of the distal compartment and plotted the simulation results in Figures 6 and 7 respectively. In Figure 6, the responses are measured using a stimulus with photocurrent of 250 nA and the speed (as shown in x-axis) has been normalized on a scale from 1 through 100. For centripetal (CP) motion, the evoked responses are not sensitive to the speed in either case of having a reciprocal synapse or no reciprocal synapse. For centrifugal (CF) motion, the evoked response is maximum when the speed is tuned to the propagation delay of the cation voltage in the dendrite, and is reduced gradually when the speed is slower or faster. The results visually illustrate that, for the experiments performed, CF motion evokes a larger response than CP motion within a range of speed and the amplitude is enhanced by the presence

of the reciprocal synapse.

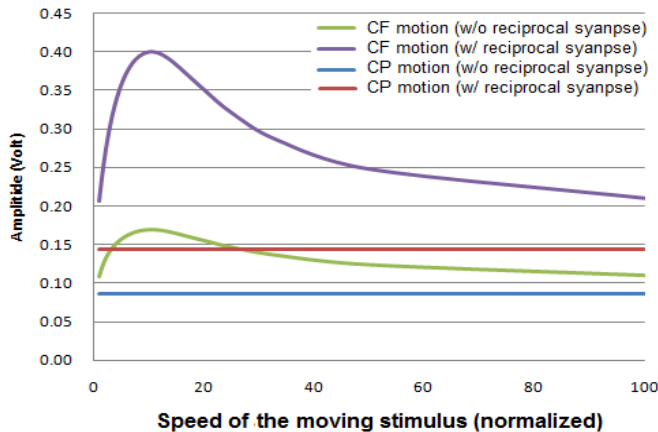


Fig. 6. The response of the distal compartment swept by the moving stimulus at different speeds (Given a photocurrent of 250nA as the input)

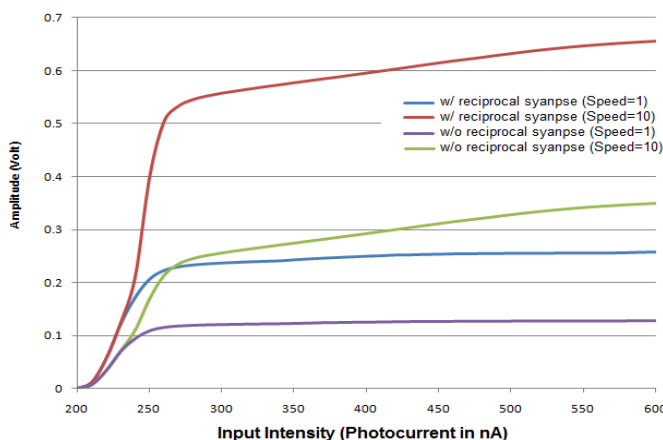


Fig. 7. The responses of the distal compartment swept by the moving stimulus with different input strength along the centrifugal direction

In Figure 7, the simulations are conducted by sweeping the CF motion with different amounts of photocurrents as the input. We used the moving stimulus at two speeds, 1 and 10, corresponding to speeds not tuned and tuned to propagation delays. In each case, we plotted two curves reflecting dendritic tip response in the presence and absence of a reciprocal synapse. Given the presence of a reciprocal synapse and the moving stimulus at a matched speed (i.e. 10), the response of the distal compartment is stronger than other cases. Moreover, the responses are enhanced by the presence of the reciprocal synapse across the entire input range that we swept.

V. CONCLUSION AND ACKNOWLEDGEMENTS

We have presented a neuromorphic circuit that emulates the directional selectivity response in biological SACs and demonstrated that incorporating the reciprocal synapses in the circuit increases directional selectivity. Incorporating SAC responses in retinal neuromorphic circuits has not been performed previously and is an important step towards circuits

that implement the directional selectivity of the vertebrate retina.

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