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The Review of Neuromorphic Circuit with Astrocyte-Based Self-Repairing Mechanism

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ABSTRACT: Research on neurophysiology reveals that the brain not only identifies damaged synapses during neural injury but also possesses the ability to repair these faulty connections. A key player in this repair mechanism is the astrocyte, which participates in retrograde signaling to regulate synaptic activity. Through this collaboration between astrocytes and neurons, the performance of intact synapses is enhanced, contributing to the restoration of neural function. Motivated by this natural repair process, this paper examines the development of neuromorphic circuits designed with self-repair capabilities, inspired by astrocyte-neuron communication. The paper highlights computational models that replicate synaptic repair, forming the basis for analog circuits that adjust the signals of healthy synapses to compensate for damaged ones across various frequencies. Additionally, the review covers the concept of self-repairing hardware, particularly focusing on spiking neural networks that integrate astrocyte-neuron networks for autonomous fault detection and correction. This method provides a more precise repair mechanism compared to traditional fault tolerance strategies, showcasing its potential in neuro-inspired computing, robotics, and the development of future neural systems.

KEYWORDS: Neuromorphic Circuits, Self-Repairing Systems, Astrocyte-Neuron Interaction, Spiking Neural Networks, Neuro-Inspired Circuit, Synaptic Repair Mechanisms, Astrocyte Signalling, Neural System Recovery

I. INTRODUCTION

Astrocytes, a specialized category of glial cells, that are instrumental in managing and sustaining neuronal networks in the brain, as indicated by neurophysiological studies. These cells, the most prevalent in the central nervous system, are deeply involved in promoting synaptic communication and adaptability. They establish intricate connections with neurons through gap junctions, forming the tripartite synapse. This model integrates astrocytes with presynaptic and postsynaptic neurons into a cohesive unit, enabling two-way communication that regulates both synaptic dynamics and overall neural network behavior. Unlike neurons that produce action potentials, astrocytes react to synaptic activity by exhibiting temporary elevations in intracellular calcium ion (Ca^{2+}) concentrations. These calcium fluctuations serve as key signals, initiating a series of intracellular processes. Among their significant functions, astrocytes release gliotransmitters such as glutamate and adenosine triphosphate (ATP) into the synaptic gap. These chemical messengers, while distinct from traditional neurotransmitters, play an important role in modulating synaptic communication through astrocyte-mediated mechanisms. A vital component of this interaction is retrograde signaling, where information flows in reverse, from the postsynaptic neuron back to the presynaptic terminal, challenging the conventional understanding of unidirectional synaptic communication. This process is initiated by the release of the endocannabinoid 2-arachidonylglycerol (2-AG) from the postsynaptic neuron, which triggers a pathway that activates presynaptic receptors, influencing neurotransmitter release. Astrocytes amplify this process, thereby enhancing the signaling cascade, which culminates in endocannabinoid-mediated synaptic potentiation (e-SP). Astrocytes also absorb 2-AG, triggering the production of inositol trisphosphate (IP3) and generating Ca^{2+} oscillations within the cell. These calcium waves are essential for the astrocytic release of glutamate, which further strengthens synaptic signaling. This mechanism significantly increases the transmission probability (PR) of synapses, reinforcing robust synaptic connections and contributing to the repair and stabilization of neural networks. This signaling becomes especially critical when synapses are damaged or function inefficiently. By amplifying the activity of unaffected synapses, astrocytes compensate for weakened connections, ensuring the neural network remains operational even in the face of damage. This self-repair capability highlights their role in maintaining the brain's resilience and adaptability. Inspired by these biological processes, researchers have developed neuromorphic circuits that replicate the self-healing features of the tripartite synapse in artificial systems. By incorporating astrocyte-like signaling pathways, including retrograde communication and synaptic modulation, these circuits can autonomously detect and address faults, maintaining

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functionality even as hardware degrades. These neuromorphic systems are designed to dynamically adjust to damage, much like the brain's natural ability to self-repair. By emulating key chemical signaling mechanisms, such as retrograde pathways and e-SP, these circuits enhance the performance of functional components while adapting to deficiencies. This bio-inspired design ensures robust and reliable operation under challenging conditions. This review highlights the role of the tripartite synapse in self-repairing neural networks, focusing on astrocyte-neuron interactions and critical signaling pathways, including those involving 2-AG and Ca2+. It also explores how these biological processes have been translated into neuromorphic circuit designs and their implications for robotics, artificial neural networks, and neuro-inspired computing. The potential of these innovations to advance fault-tolerant and adaptive technologies is discussed, emphasizing their transformative impact on future systems.

II. REVIEW OF LITERATURE

Neuromorphic systems, designed to replicate the brain's structure and function, have become a focal point in electronics and computer science. These systems aim to model neural processes through circuits that perform computations similar to how the brain operates. The inclusion of self-repair capabilities is a recent development, enhancing the durability and resilience of these circuits, which are essential for both artificial and biological systems. This review highlights key advancements and identifies research gaps.

2.1 Development of Neuromorphic Circuits

The concept of neuromorphic circuits was introduced by Mead in 1990, using analog circuits to emulate biological neural structures. This led to the development of spiking neural networks (SNNs). Indiveri et al. (2011) advanced this field by designing energy-efficient neuromorphic silicon neurons. Recent research has focused on improving learning algorithms, such as the hybrid memristor-CMOS spiking system proposed by Serrano-Gotarredona et al. (2013), which combines non-volatile memristors with CMOS technology to enhance flexibility and efficiency.

2.2 Astrocyte-Neuron Interactions in Self-Repair

Astrocytes, glial cells that regulate neuronal activity, play a crucial role in self-repair. Lichtsteiner et al. (2008) examined how astrocyte-mediated regulation could improve performance and fault tolerance in neuromorphic systems. Liu et al. (2016) expanded this work by demonstrating that astrocyte-based neuromorphic circuits could enhance neuronal synchrony and support recovery. Additionally, Amiri et al. (2020) explored bio-inspired astrocyte-neuron networks to improve pattern recognition and enable self-repair in spiking networks.

2.3 Research Gaps and Future Directions

Despite progress, several gaps remain in the research of self-repair mechanisms in neuromorphic systems. One limitation is the scalability of self-repair in large systems, as most studies focus on small-scale models. More research is needed on practical applications in complex environments like robotics and AI. Another gap is the integration of memristors with astrocyte-based models. While memristors have shown potential in synaptic plasticity, combining them with astrocyte-based self-repair mechanisms could lead to more efficient and adaptive circuits, improving the robustness of neuromorphic systems.

III. METHODOLOGY

The neuromorphic circuit design described here models the behavior of biological neurons and astrocytes, with a focus on implementing self-repairing capabilities to enhance network stability. The system incorporates postsynaptic neurons (PSNs), presynaptic neurons, synapses, a 2-AG generator, an astrocyte circuit, and a PR (Probability Rate) signal generator. Each component serves to replicate biological processes and ensure fault tolerance in the neural network.

3.1 Presynaptic Neuron and Synapse Interaction

The presynaptic neurons generate spike trains that act as the input to synapses. Each synapse has a unique Probability Rate (PR) that determines the likelihood of transmission. The synaptic activity is governed by the comparison between the presynaptic voltage ($V_{pre\text{-}syn}$) and the PR value. If $V_{pre\text{-}syn}$ is lower than PR, the synapse activates and allows a current of 80 nA to flow into the postsynaptic neuron. A differential amplifier with a current mirror configuration compares these values, emulating how biological synapses transmit signals based on their probability of firing.

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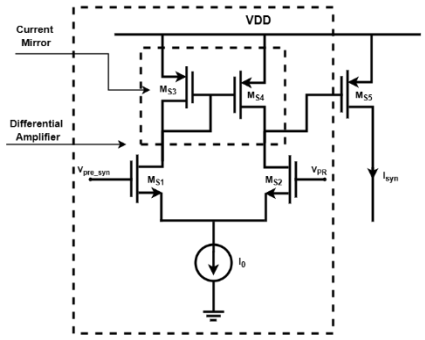

Fig.3.1. Presynaptic Neuron

3.2 Postsynaptic Neuron (PSN)

The PSN circuit is based on the Izhikevich neuron model, which mimics the spiking behavior of biological neurons. Each PSN is connected to multiple synapses, each contributing a current based on its activation state. The total current entering the PSN is the sum of the active synapse's currents. If more synapses are active, the PSN's likelihood of firing increases, similar to the way biological neurons integrate inputs from various synapses to generate a response.

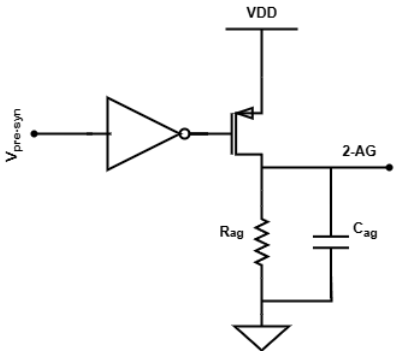

Fig.3.3. 2-AG Signal Generator

3.3 2-AG Signal Generation

The 2-AG Generator is used to simulate retrograde signaling, akin to biological 2-AG signaling in neurons. The output spikes from the PSN are inverted and applied to a PMOS transistor, driving current into a capacitor that forms an RC network. The capacitor accumulates charge over time, with the voltage across it representing the 2-AG signal. This integration mimics how 2-AG levels rise with increased neuronal activity. The 2-AG signal is then passed through a current mirror and nMOS transistors to generate a stable voltage that serves as input to the astrocyte circuit.

3.4 Astrocyte Circuit

The astrocyte circuit simulates the regulatory function of biological astrocytes in controlling synaptic activity through calcium (Ca^{2+}) signaling. The 2-AG signal triggers oscillations in intracellular Ca^{2+} levels in the astrocyte. Elevated Ca^{2+} levels stimulate the release of gliotransmitters, which modulate synaptic activity, balancing network activity. This mechanism replicates how astrocytes control neurotransmitter release and enhance synaptic strength through retrograde signaling, ensuring network stability.

3.5 e-SP Signal Generation

The e-SP signal is generated based on the calcium oscillations from the astrocyte circuit. The Ca^{2+} signal is amplified using a common-source amplifier and converted into a pulsatile waveform through an inverter. The waveform is processed through an RC network and a current mirror, producing the final e-SP signal. This signal is akin to the

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potentiation effect seen in biological networks, where astrocytes release gliotransmitters like glutamate to enhance synaptic strength.

3.6 PR Signal Generator

The PR Signal Generator controls the likelihood of synapse activation. It uses the DSE (Depolarization-Induced Suppression of Excitation) and e-SP signals to adjust the PR value. When a synapse receives more activity, the PR value decreases, making it more likely to fire. The PR signal is generated by balancing these inputs using current mirrors, providing a dynamic threshold that controls synaptic activation.

3.7 Self-Repair Mechanism

The self-repair mechanism is at the heart of this neuromorphic design. If some synapses fail (low PR), the astrocyte circuit compensates by enhancing the PR values of the remaining synapses. This mechanism mimics how biological systems maintain stability despite neuronal damage, ensuring the overall network activity remains functional. Adjusting the PR values helps ensure that synaptic activity is sustained, even in the presence of faulty synapses.

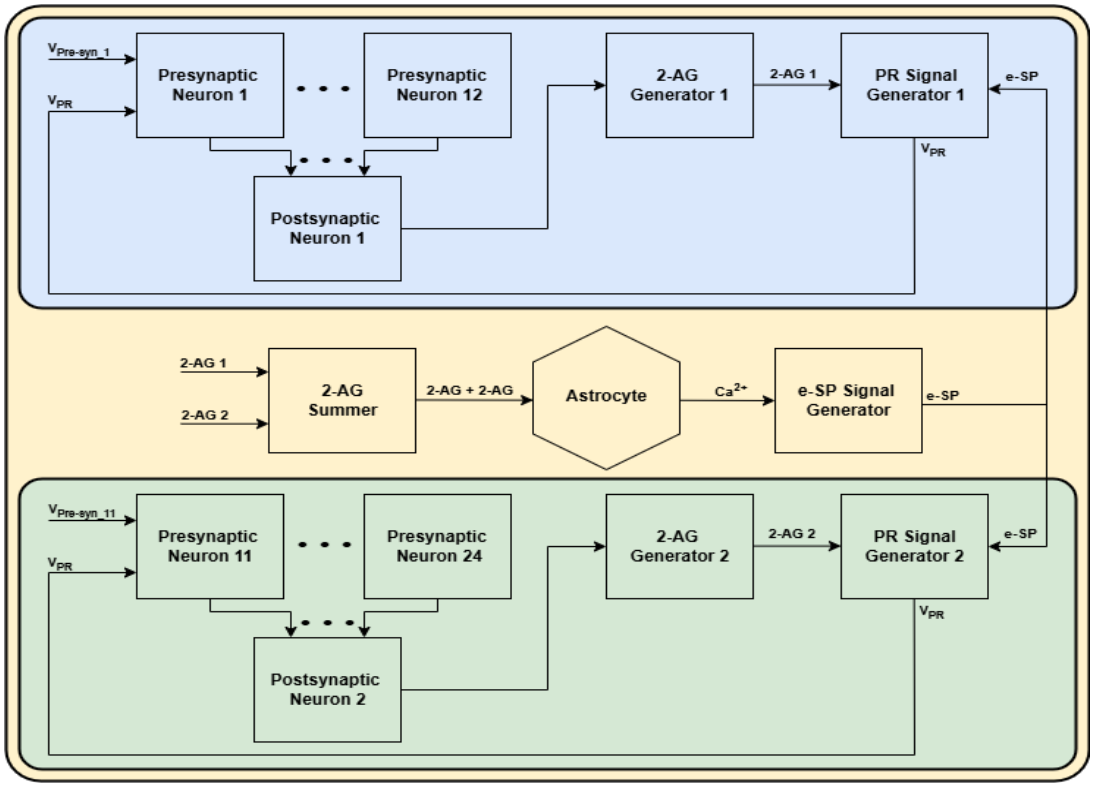

Fig.3. The Complete block diagram of a self-repairing capability CMOS Neuron

IV. RESULTS

The proposed neuromorphic CMOS circuit, developed to model a self-repairing tripartite synapse, was extensively analyzed through simulations under multiple scenarios, including healthy operation, partial synaptic damage, and complete synaptic failure. The circuit successfully demonstrated an adaptive self-repair mechanism driven by the astrocyte-inspired module, which redistributed synaptic probabilities among the remaining functional connections to restore neural firing activity. Detailed post-layout simulations incorporated parasitic effects, confirming the robustness of the self-repair process even under degraded conditions. Phase portrait analysis highlighted the dynamic recovery of synaptic activity during and after fault events, illustrating the circuit's capacity for adaptive compensation. Monte Carlo simulations further validated its resilience, showing consistent performance despite variations in device parameters and process mismatches. These findings establish the circuit’s potential for enhancing fault tolerance in neuromorphic systems, paving the way for more reliable and efficient artificial neural networks.

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V. CONCLUSION

This paper presents a neuromorphic circuit model that mimics the behavior of a tripartite synapse, integrating presynaptic neurons, postsynaptic neurons (PSNs), astrocytes, and self-repair mechanisms. Using CMOS transistors, the design replicates key aspects of biological neural networks, including fault tolerance and adaptability. The Probability Rate (PR) modulation ensures synaptic health is reflected in the network's activity, with healthy synapses contributing more to the overall signal. The astrocyte circuit enhances system reliability by compensating for malfunctioning synapses, similar to natural neural processes. Additionally, the 2-AG signaling pathway activates the astrocyte feedback loop, helping restore balance in the network by boosting the activity of healthy synapses. With low power consumption and compact design, this circuit is a strong candidate for applications in neuromorphic computing, neuroprosthetics, and adaptive systems. This work not only offers insights into fault-tolerant computing but also lays the groundwork for future developments in artificial neural networks. Future studies could focus on scaling the model, incorporating machine learning for more dynamic self-repair, and optimizing it for specific neuromorphic applications.

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