



Research paper

A new analog self-repairing algorithm based on a novel tripartite synapse circuit

Mahnaz Ranjbar^{a, *}, Gholamreza Karimi^{a, *}, Ali Khosravi^a, Derek Abbott^b^a Department of Electrical Engineering, Faculty of Electrical and Computer Engineering, Razi University, Kermanshah 6714967346, Iran^b School of Electrical and Electronic Engineering, University of Adelaide, Adelaide, SA5005, Australia

ARTICLE INFO

Keywords:

Biological neural systems
Astrocyte
Tripartite synapse
Self-repairing
Analog
CMOS

ABSTRACT

Self-repair is a biological mechanism through which the nervous system compensates for damage and maintains stable neural activity, a process that is partly mediated by astrocytes. Astrocytes play a critical role in synaptic regulation by releasing global feedback signals that contribute to error correction and the adjustment of neuronal firing-rate set-point. They receive signals from multiple connected synapses, such as 2-arachidonoylglycerol (2-AG), and subsequently release various neurotransmitters that modulate the activity of neighboring neurons. This astrocyte-mediated feedback is an essential component of biologically inspired self-repair mechanisms. When synaptic faults occur, the resulting disruption is sensed through changes in intracellular calcium (Ca^{2+}) levels in the postsynaptic neuron, which triggers adaptive adjustments in neuronal excitability. Together, astrocyte feedback and calcium-dependent regulation from complementary mechanisms facilitate postsynaptic homeostasis. In this work, we investigate the self-repair phenomenon and its relationship with astrocytes and tripartite synapses. First, a novel self-repair algorithm capable of detecting and compensating for synaptic faults is introduced, followed by the design of an analog circuit that implements this algorithm. In addition, a new tripartite synapse structure is proposed to mimic key biological interactions between neurons and astrocytes. The proposed circuits are implemented in a neural network consisting of three layers, 22 neurons, and five astrocytes. Simulation results demonstrate that healthy astrocytes and synapses can effectively compensate for synaptic faults by injecting feedback signals, thereby enhancing the self-repair capability of the network. The circuit was designed and simulated using HSPICE software with standard 0.35 μm CMOS technology.

1. Introduction

In recent years, considerable attention has been devoted to understanding the mechanisms that maintain stable neural function and enable self-repair in biological nervous systems. Such mechanisms have been observed in various contexts, including recovery processes after stroke [1], homeostatic plasticity [2], retrograde signaling pathways [3], and the role of glial cells in the formation and dysfunction of the neurovascular unit [4]. Neuronal self-repair has consequently emerged as a central topic in neuroscience, encompassing studies on synaptic homeostasis [5], investigations of neural homeostasis in regulating nervous system activity and preventing diseases [6], and analyses of presynaptic homeostatic plasticity mechanisms [7].

Biological neurons, despite their long lifespan and lack of cell division, maintain stable functionality for decades through multiple self-repair and homeostatic strategies. These include axonal and

dendritic regeneration [1], synaptic scaling [3], intrinsic homeostasis mechanisms, and stabilization supported by glial [8,9]. Such processes operate continuously and in a decentralized manner, enabling neural systems to compensate for perturbations such as noise, parameter drift, and structural damage without external supervision. Gliotransmission also contributes to maintaining effective signal transfer across network layers, although it can also decorrelate neuronal activity from the input signal pattern [10].

Theoretical studies suggest that if each neuron measures its average activity and adjusts its synaptic weights to maintain a target value, stability can naturally emerge at the level of the entire network [11–13]. This mechanism uniformly increases or decreases all synapses of a neuron [14,15], indicating that average neuronal activity is controlled by a homeostatic negative feedback mechanism [15,16].

Homeostatic regulation in biological neural systems is largely governed by negative feedback mechanisms that allow neurons to sense

* Corresponding author: Razi University, Kermanshah, Iran.

E-mail address: ghkarimi@razi.ac.ir (G. Karimi).

<https://doi.org/10.1016/j.rineng.2026.111492>

Received 7 July 2025; Received in revised form 10 May 2026; Accepted 9 June 2026

Available online 10 June 2026

2590-1230/© 2026 The Authors. Published by Elsevier B.V. This is an open access article under the CC BY-NC license (<http://creativecommons.org/licenses/by-nc/4.0/>).

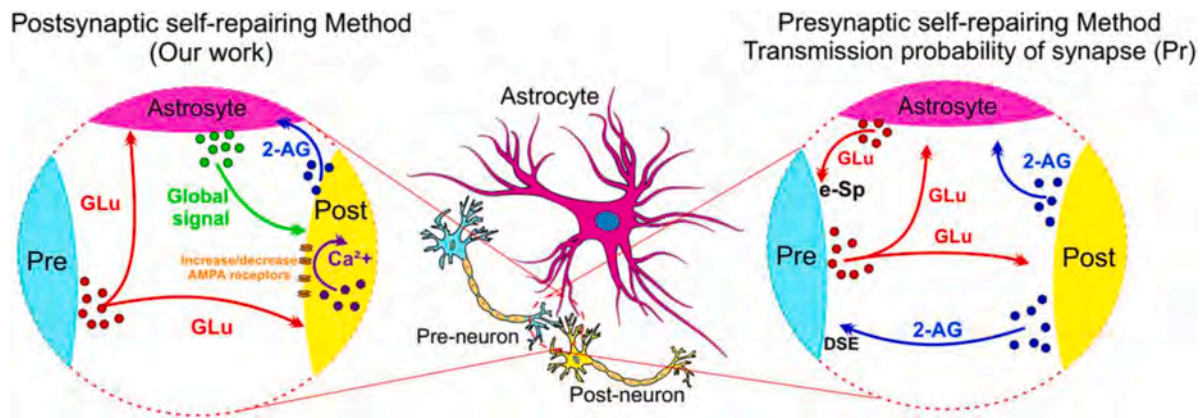


Fig. 1. Biological tripartite synapses consist of two neurons and one astrocyte. Two complementary self-repair methods are considered. (Right) The presynaptic PR mechanism regulates postsynaptic homeostasis. Within the tripartite synapse, in addition to neuron-neuron interactions, Glu and 2-AG release and diffusion in the synaptic space (DSE) are mediated through astrocyte signaling (e-SP). The combination of these processes constitutes a primary driver of self-repair dynamics. (Left) The postsynaptic homeostatic mechanism comprises synaptic scaling and the generation of intercellular Ca^{2+} in the postsynaptic neuron as a set-point controller. Here, astrocyte output acts as a global signal that modulates the postsynaptic intercellular Ca^{2+} to tune the set-point and thereby regulate receptor trafficking, which controls receptor accumulation at synapses.

their own activity, integrate this information over longer time scales, and adjust synaptic properties to maintain activity around a desired set point [17]. Homeostasis typically operates through two complementary processes: regulation of synaptic strength (synaptic scaling) and modulation of intrinsic neuronal excitability [14]. In this framework, self-repair and homeostasis can be viewed as closely related mechanisms that maintain stable neural activity despite perturbations or synaptic degradation [8,18].

Astrocytes also play a critical role in glutamatergic neurotransmission. Glial cells supply metabolic precursors for glutamate signaling, where glutamate taken up by glial cells is converted to glutamine and transported back to presynaptic terminals for reconversion into glutamate [19]. Astrocytes can further participate in presynaptic self-repair processes by generating glutamate-mediated signals that influence presynaptic release probability and synaptic transmission [3,20–22]. Because astrocytes integrate inputs from multiple neurons, they can provide relatively stable reference signals that contribute to network stability.

Recent studies have highlighted the important role of astrocytes in regulating neuronal activity and coordinating neural function. Astrocytes may play a potential role in the processing of noisy neural information by modulating neuronal dynamics and enhancing signal transmission in the presence of noise [24]. Astrocytes have been shown to promote faster learning and support memory formation [23]. Lorenzo et al. [25] proposed an extended tripartite synapse model incorporating perinodal astrocytic processes, demonstrating that local calcium transients in extrasynaptic astrocytic branches can modulate glutamate signaling, increase presynaptic release probability, and transiently enhance synaptic efficacy during short-term plasticity. Astrocyte-mediated modulation can also shape neuronal dynamics, including spike initiation, the emergence of self-sustained oscillations, and both inhibitory and excitatory effects, depending on synaptic strength [26]. These findings highlight the multifaceted role of astrocytes in neural computation and their potential relevance for adaptive neuromorphic systems. Studies have explored the role of astrocytes in unsupervised learning [27], neuromorphic simulations using computational astrocyte models to achieve high-fidelity macro-level behavior [28], and the contribution of astrocytes to self-repair in spike-timing-dependent plasticity (STDP)-based unsupervised learning [29] as well as in retrograde signaling pathways [30–32].

Several neuromorphic implementations have explored hardware mechanisms for homeostatic regulation. In [33], a brain-inspired homeostatic neuron based on phase-change memory (PCM) devices is

proposed. By exploiting the gradual conductance modulation of PCM, the neuron dynamically adjusts its firing threshold, enabling intrinsic homeostasis, spike-frequency adaptation, and active forgetting directly in hardware. When combined with STDP-based synapses, the system demonstrates stable, unsupervised learning and robustness to noise. Similarly, [34] investigates homeostatic regulation in a silicon leaky integrate-and-fire (LIF) neuron, where the firing rate gradually returns to its spontaneous level after prolonged perturbations. The regulation is achieved through two mechanisms: presynaptic activity-dependent synaptic weight adjustment and activity-dependent modification of the synaptic threshold. However, these approaches rely solely on neuronal mechanisms and do not incorporate glial modulation or tripartite synaptic interactions. Neuromorphic architectures capable of self-detecting and self-repairing synaptic faults without conventional fault-detection circuitry were presented in [35]. Despite these advances, designing neuromorphic hardware that remains robust and fault-tolerant over long periods continues to be a significant challenge.

Despite these biological insights, most neuromorphic circuits have primarily focused on presynaptic homeostatic regulation. Both analog implementations [36–38] and digital architectures [35] typically compensate for reduced postsynaptic responses by adjusting neurotransmitter release probability or vesicle size [7,18]. In several analog studies [36,37], simplified astrocyte circuits [30] are used to emulate certain astrocytic functions by generating excitatory presynaptic signals that regulate presynaptic release probability. Other approaches, such as the memristor-based neural network presented in [39], incorporate limited self-repair capabilities but do not include biologically inspired glial-mediated repair mechanisms.

More broadly, previous research has predominantly concentrated on presynaptic homeostatic mechanisms, while comparatively less attention has been given to postsynaptic regulation [33,34,38]. One reason is that postsynaptic homeostasis is more challenging to implement in hardware, even though postsynaptic processes often exert a stronger influence on neuronal output. For instance, the circuit proposed in [38] implements postsynaptic synaptic scaling using an ultra-low-leakage circuit to enable regulation over very long-time scales. Nevertheless, many existing approaches implement only single-layer homeostatic regulation and lack astrocyte-mediated interactions, which may limit biological realism and scalability in larger neuromorphic systems.

In this work, we focus on postsynaptic homeostasis as a mechanism for self-repair in neuromorphic neural networks. Inspired by these biological principles, this study proposes a biologically motivated self-repair strategy that combines postsynaptic homeostasis with

astrocyte-mediated feedback in a tripartite synapse structure. The proposed approach enables fault compensation at the postsynaptic level in a compact analog neural network. To demonstrate this concept, a novel tripartite synapse architecture is introduced in which postsynaptic regulation is guided by a target activity level together with astrocytic feedback signals. Using the astrocyte circuit reported in [40], the proposed design shows that astrocyte-mediated feedback can partially compensate for network faults through a biologically inspired self-repair algorithm. This work, therefore, presents a simple analog circuit framework for implementing postsynaptic self-repair in neuromorphic systems and represents a step toward more stable and scalable neural network-based circuits.

The remainder of this paper is organized as follows. Section 2 reviews the biological self-repair mechanisms in neural systems and presents the implementation of the proposed analog circuit together with astrocytic components. Section 3 presents the simulation results of the proposed circuit. Finally, Section 4 discusses the significance of the proposed approach and outlines potential directions for future research.

2. Self-repairing algorithm

This section discusses two topics. First, the self-repair capability of biological astrocytes within spiking neuron-astrocyte networks is introduced. Second, the implementation of a self-repair mechanism in the proposed circuit is discussed.

2.1. Principle of biological self-repairing

As previously mentioned, biological neural networks possess various self-repair mechanisms, including (a) presynaptic self-repair and (b) postsynaptic self-repair, both illustrated in Fig. 1. The former mechanism (Fig. 1, Right panel) involves the regulation of presynaptic neurotransmitter release. Upon arrival of an action potential at the presynaptic terminal, glutamate is released into the synaptic cleft. This triggers postsynaptic depolarization, which in turn prompts the release of the 2-arachidonoylglycerol (2-AG) signal from the postsynaptic neuron. The released 2-AG can reach the postsynaptic terminal via two pathways: a direct pathway and an indirect pathway. In the direct mode, presynaptic terminal receptors directly receive 2-AG without astrocyte mediation, generating a signal known as DSE signal [3]. This signal can be considered as inhibitory signal in the presynaptic neuron. Indirectly, 2-AG binds to receptors on astrocytes, triggering the release of inositol triphosphate (IP3) and subsequently increasing intracellular Ca^{2+} . This Ca^{2+} elevation leads to glutamate release from astrocytes. The glutamate released by astrocytes then binds to group-1 metabotropic glutamate receptors (mGluRs) on the postsynaptic terminal, referred to as e-SP receptors [22]. This signal can be considered excitatory for the presynaptic neuron. Within the context of this algorithm, the astrocyte is modeled as a glutamate supplier in the tripartite synapse [20].

It is assumed that each time the postsynaptic neuron generates a spike (fires), 2-AG is released. Subsequently, the released 2-AG is modeled using the following equation [30,31].

$$\frac{d(\text{AG})}{dt} = \frac{-\text{AG}}{\tau_{\text{AG}}} + r_{\text{AG}}\delta(t - t_{\text{sp}}), \quad (1)$$

where AG denotes the amount of 2-AG released by the postsynaptic neuron, τ_{AG} and r_{AG} are the decay and generation rates of 2-AG, respectively, and t_{sp} is the time of the postsynaptic spike.

In the next self-repair mechanism (Fig. 1, Left panel), two algorithms are involved: (1) synaptic scaling and (2) homeostatic regulation. Astrocytes again play a critical role, similar to the presynaptic mechanism. Specifically, astrocytes integrate 2-AG signals from neighboring neurons and generate a global signal that modulates postsynaptic neurons and thereby regulates neurotransmitters and neuromodulators, including Glu, D-serine, adenosine triphosphate (ATP), and other molecules.

Global astrocyte Ca^{2+} events modulate cortical up-states and contribute to maintaining stable network excitability [41]. This global signal is referred to as the excitatory signal. It provides a reference for neighboring neurons by reflecting the average spike activity and establishing a baseline for frequency regulation. In the proposed algorithm, this reference is detected using the astrocytic output signal. When the presynaptic neuron fires with sufficient intensity, it triggers postsynaptic spiking. The resulting postsynaptic activity regulates the intercellular Ca^{2+} dynamics of the postsynaptic neuron to achieve homeostasis. Accordingly, this signal can be considered as inhibitory for the postsynaptic neuron.

When a fault occurs in the synapse, the firing frequency of postsynaptic neuron decreases, leading to a reduced intercellular Ca^{2+} level (i.e. the negative-feedback signal). This decrease enables the neuron to detect the fault, and the hemostasis is restored. Postsynaptic neurons sense changes in activity through Ca^{2+} -dependent sensors and adjust receptor trafficking to regulate receptor accumulation at synapses [14, 11]. This negative-feedback mechanism is similar to the DSE signal in the presynaptic self-repair mechanism.

Both presynaptic and postsynaptic self-repair mechanisms operate simultaneously in biological neural systems. In the present framework, the astrocytic output signal directly regulates synaptic scaling while indirectly modulating postsynaptic homeostasis regulation mechanisms. In this work, only the postsynaptic self-repair algorithm is employed, while the presynaptic alternative is mentioned solely for comparison and is not implemented in our model.

2.2. Self-repairing mechanism in an analog circuit

In this model, each neuron is connected to 10 synapses. Synaptic inputs are represented as currents injected into the neuron, and the neuron outputs are neural spikes. If an error occurs in some synapses connected to a given neuron, the resulting input current to the postsynaptic neuron decreases, which reduces its firing frequency. As the firing decreases, the postsynaptic intercellular Ca^{2+} level also decreases. This reduction enables fault detection and activates the postsynaptic negative-feedback mechanism.

Because an astrocyte is included in the network and generates a global signal, small network errors do not significantly alter its output. Therefore, the astrocyte output remains approximately constant during an error and acts as a reference signal in the tripartite synaptic space. Under this stable global reference and the reduction in postsynaptic intercellular Ca^{2+} , healthy tripartite synapses detect the error in the system and compensate for the reduced current. Subsequently, healthy tripartite synapses detect the fault and compensate for the reduced input current. In the following, each circuit is explained separately.

2.2.1. Neuron

This paper adopts the circuit design proposed by Wijekoon [42], which is based on the numerical model introduced by Izhikevich [43]. In this model, neuronal dynamics are described using two state variables, corresponding to the voltages across two capacitors: V (membrane voltage) and U (slow variable). The circuit behavior is approximated by the following equations:

$$\frac{dV}{dt} = \begin{cases} a_1 V^2 - a_2 V - a_3 U^2 + a_4 U + a_5 + i_1 & V > U - V_T \\ a_6 V^2 - a_7 V - a_8 UV + i_2 & \text{otherwise} \end{cases} \quad (2)$$

$$\frac{dU}{dt} = a_9 V^2 - a_{10} V - a_{11} U^2 + a_{12} U + a_{13} \quad (3)$$

$$\text{If } V > V_{\text{th}} \text{ then } \begin{cases} V \leftarrow c \\ U \leftarrow U + d \end{cases} \quad (4)$$

where a_i (for $i=\{1, \dots, 13\}$) V_T , V_{th} , i_1 , c , and d are constants defined in

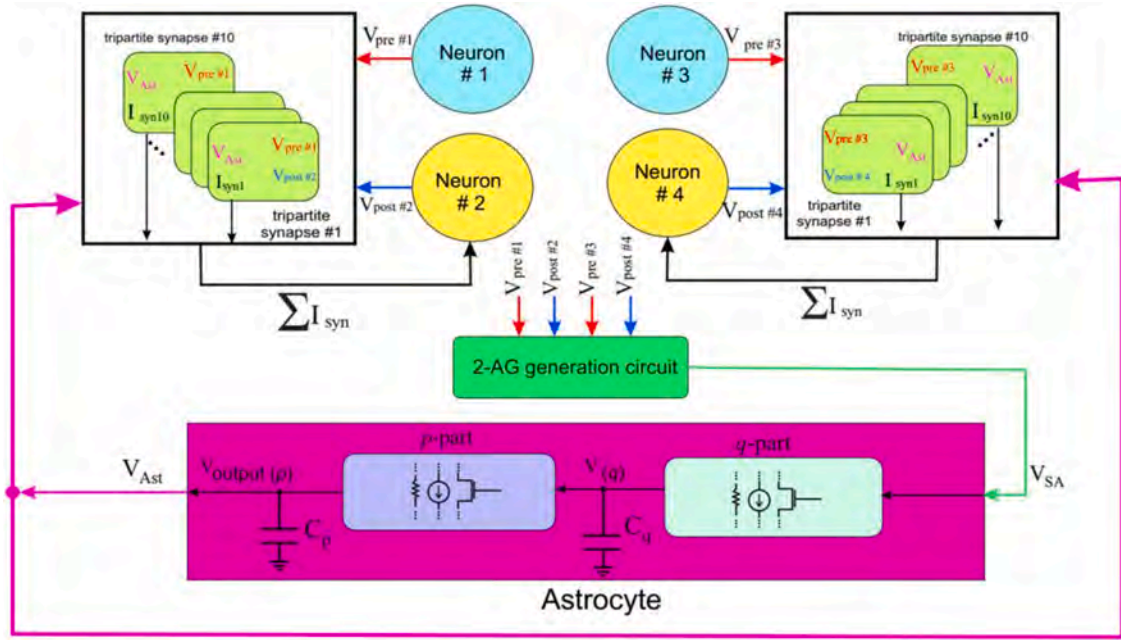


Fig. 2. Block diagram of the proposed self-repairing network. In this scheme, when the presynaptic neurons spike, Glu in the synaptic cleft is released. This Glu causes a synaptic current ($I_{syn,i}$). The sum of these currents is applied to the post-synaptic neuron. The magnitude of this current depends on the set-point value from the postsynaptic neuron and the astrocyte output (V_{Ast}). The astrocyte input is also controlled by 2-AG produced by all four neurons connected to the astrocyte.

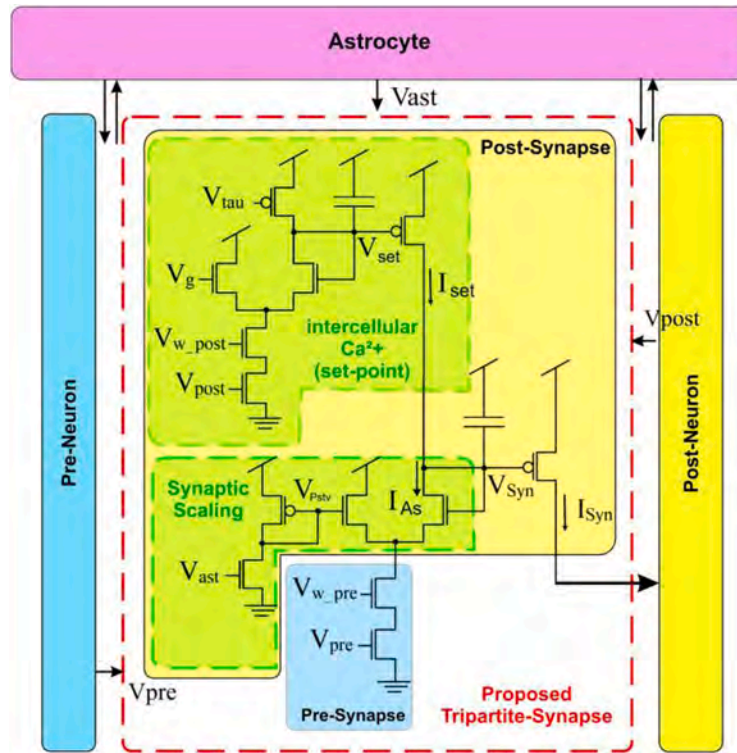


Fig. 3. Proposed tripartite synapse circuit in the postsynaptic self-repairing algorithm. The negative feedback is obtained through the process of intercellular Ca^{2+} generation, which sets the set-point value. Signal scaling, produced by receiving 2-AG and producing IP₃ and Ca^{2+} in the astrocyte circuit, causes the generation of a global signal in the synaptic space. This signal is received by the postsynaptic neuron, which is effective in regulating the output current (I_{syn}).

[42], determined by transistor dimensions (i.e. W/L ratio), operating region, bias voltages, and input current. This circuit operates in an accelerated time mode and exhibits low power consumption across various neuronal firing patterns [42].

2.2.2. Astrocyte

For simplicity, an astrocyte model previously reported in [40] is adopted in this work. This circuit generates the global signal and has been utilized for frequency adaptation [40] and synchronization [44]. Owing to its self-repair compatible characteristics, it is employed in the

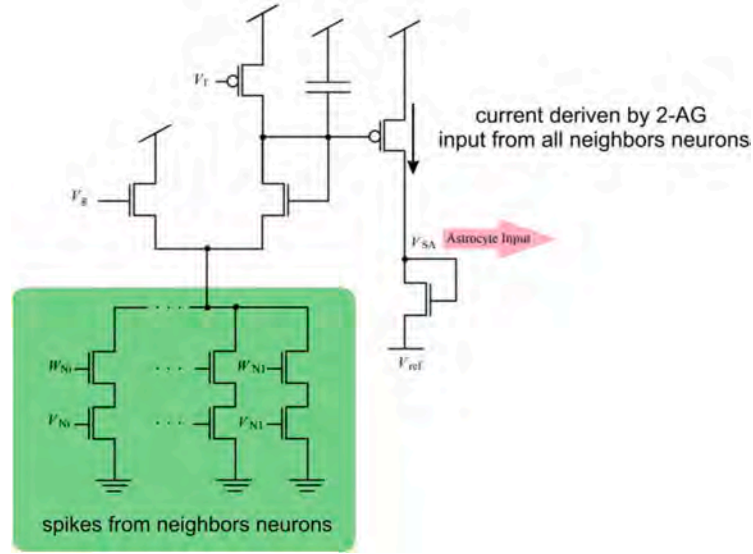


Fig. 4. The circuit involves neighboring neurons connected to an astrocyte, which produce 2-AG. Each astrocyte receives 2-AG from its connected neighboring neurons. The 2-AG is then transported to the astrocyte's input (V_{SA}).

proposed network. The simplified astrocyte dynamics are expressed as:

$$\begin{cases} \dot{q} = (1 + \tanh k_1 [Z - K_2])(1 - q) - k_3 q \\ \dot{p} = -K_4 p + K_5 + K_6 q \end{cases} \quad (5)$$

where q represents the internal astrocyte state, k_i ($i = \{1, \dots, 6\}$) are constants defined in [45], and Z and p denote the input and output signals of the model, respectively. The state variables p and q are physically realized by the across capacitors C_p and C_q respectively [40]. These dynamics, as illustrated in Fig. 2, correspond to the physiological process of the astrocyte. Upon receiving the 2-AG input (represented by Z in Eq. (5)), the astrocyte triggers the release of IP_3 to the endoplasmic reticulum (ER). The binding of IP_3 to its receptors (IP_3Rs) facilitates the opening of Ca^{2+} channels, leading to an increase in intercellular Ca^{2+} concentration and the subsequent release of glutamate. Finally, the astrocyte output voltage (V_{Ast}) is derived from the parameter p and functions as a global reference signal for the network.

2.2.3. Proposed tripartite synapse

The synapse, as the third fundamental component of this neural network, is implemented using the proposed tripartite synapse circuit detailed in Fig. 3. This circuit comprises two primary functional blocks: presynaptic and postsynaptic self-repair mechanisms. The postsynaptic section, which is the central focus of this study, is further subdivided into

two key sub-circuits: synaptic scaling and a set-point controller. At the core of the tripartite synapse architecture lies an above-threshold DPI [46] circuit, engineered to emulate fundamental synaptic integration dynamics. To extend these dynamics toward biologically inspired feedback modulation, a dedicated 2-AG generation circuit and an intercellular calcium-regulation module for the postsynaptic neuron have been integrated. These modules interface directly with the DPI core, thereby establishing functional analogs of astrocyte-mediated and postsynaptic signaling pathways within the circuit.

The input signals of the proposed tripartite synapse include the presynaptic neuron's spikes (V_{pre}), the postsynaptic neuron's activity (V_{post}), the postsynaptic intercellular Ca^{2+} current (used to generate the set-point value), and the astrocyte's output (V_{ast}). When the postsynaptic neuron fires, a 2-AG current is produced and applied to the astrocyte circuit. If the postsynaptic firing frequency decreases, the intercellular Ca^{2+} level also decreases proportionally. This reduction leads to an increase in the synaptic current (I_{syn}).

As long as there is no error in the network, after a short period, the synapse output current (I_{syn}) is regulated to balance with the target set current (I_{set}), based on the set-point voltage (V_{set}) and the astrocyte's output signal (V_{ast} and its associated current). After this equilibrium, if an error occurs in the synapses of one neuron, the astrocyte output does not change significantly because it integrates inputs from all the connected neurons (neurons #1 to 4). With the astrocyte output signal remaining approximately constant, any changes in the postsynaptic firing frequency strongly affect the intercellular Ca^{2+} . As the postsynaptic frequency decreases, the feedback current (I_{set}) decreases, and consequently, the synapse output current (I_{syn}) increases. Therefore, the increased output of healthy synapses partially compensates for the reduced current caused by faulty synapses.

2.2.4. Proposed 2-AG transfer from synapse to astrocyte

The astrocyte receives 2-AG from neighboring postsynaptic neurons. When each neighboring neuron spikes, it releases 2-AG into the synaptic space, which is detected and integrated by the astrocyte. The proposed circuit is illustrated in Fig. 4. Specifically, postsynaptic spikes trigger 2-AG release. The astrocyte senses this 2-AG signal, integrates it with other inputs (from all four neighboring neurons), and produces a coordinated global modulatory output (V_{ast}) delivered to the tripartite synapse. The dynamics of the 2-AG released signal are described by Eq. (1).

In the proposed circuit (Fig. 4), 2-AG signals released by postsynaptic neurons are integrated by the astrocyte. This integration enables the

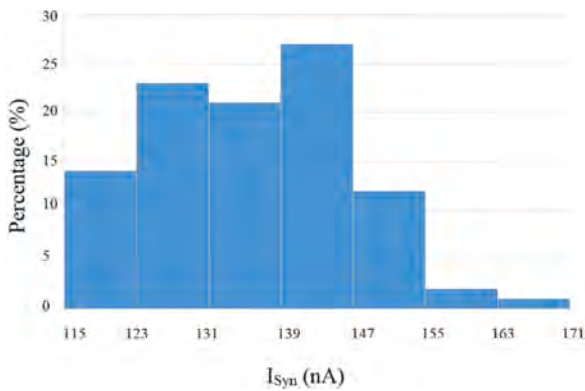


Fig. 5. Histogram of the mismatch effect on the output synaptic current of the proposed synapse circuit. Median = 135 nA, variance = 0.11 fA, standard deviation = 0.105 nA with $V_{ast} = 0.4$ V.

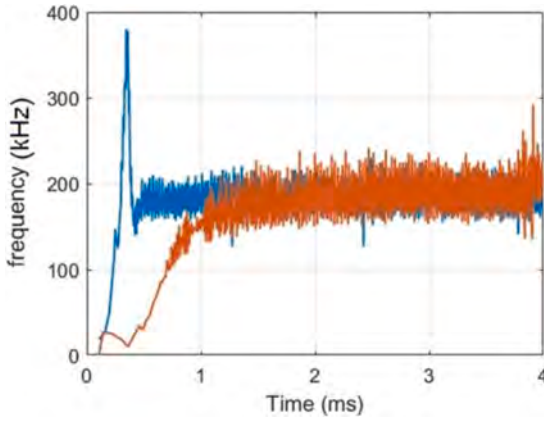


Fig. 6. Output firing frequency of neuron #2. (Blue) Proposed self-repairing circuit without any fault. (Red) Conventional neural network without a self-repair mechanism.

astrocyte to sense the overall network activity and generate a coordinated modulatory output. In the circuit implementation, the total amount of 2-AG produced by the postsynaptic neurons, as described in Eq. (1), is represented by the voltage v_{sa} in the proposed 2-AG generation circuit. This voltage is applied as the input to the astrocyte circuit. The time constant of this process is determined by the capacitor present in the circuit together with the threshold voltage V_T . In addition, the generation rates defined in the mathematical model are implemented in the circuit through the control voltages V_g and W_{Ni} .

3. Simulation results

This section presents the simulation results of the proposed circuit. These results demonstrate that the designed astrocyte and tripartite synapse properties enable a self-repair mechanism in the network's postsynaptic neurons. To examine the impact of device mismatch on the circuit's performance (Fig. 3), we first considered simultaneous dimensional mismatch for all transistors. Fig. 5 shows the Monte Carlo analysis histogram of the output synaptic current (I_{syn}) with mismatch applied to all transistors. This simulation assumed a k -CMOS process variation (affecting both all L and W parameters) for all transistors. We analyzed 100 Monte Carlo samples with a $\pm 5\%$ variation. The obtained results include: median = 135 nA, variance = 0.11 fA, standard deviation = 0.105 nA, and $V_{ast} = 0.4$ V.

Next, a small neural network was designed to demonstrate the performance of the self-repair algorithm. This network, as shown in Fig. 2, consists of four neurons, one astrocyte, and 20 synapses. Each postsynaptic neuron is connected to 10 synapses, which generate its input signals. Each postsynaptic neuron releases a 2-AG signal into the presynaptic space upon spiking. Due to the astrocyte's connection to multiple neurons across neighboring synaptic spaces (Neuron #1 – 4), the 2-AG signals from all neurons converge on the astrocyte. Therefore, the signal received by the astrocyte represents a global signal that reflects the average activity of the neighboring neurons. Consequently, accumulated errors in some synapses have minimal impact on the astrocyte's output.

The focus of this study is the postsynaptic self-repair behavior implemented by the proposed circuit. In this configuration, the presynaptic neurons (neuron #1 and neuron #3) are stimulated by a constant input current representing presynaptic activity ($I_{in} = 100$ nA). Postsynaptic neurons (neuron #2 and neuron #4) are stimulated solely by the combined output of the proposed tripartite synapses (ΣI_{syn}), without any direct external current injection.

Initially, to demonstrate the accuracy of the proposed algorithm under error-free conditions, the self-repair network (Fig. 2) is compared with a simple neural network without self-repair (comprising four

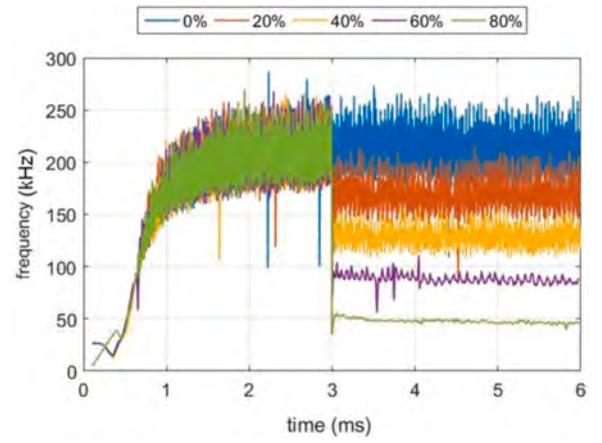


Fig. 7. Firing frequency of neuron #2 without the self-repair mechanism under different fault conditions. Here, 0% to 80% of the synapses connected to neuron #2 become defective at $t > 3$ ms.

neurons and 20 synapses without any additional circuitry). All synapses are assumed to be healthy, and the tripartite synapse circuit emits Glu signals (I_{syn}). The self-repair circuit enables adaptation by injecting this current into the corresponding postsynaptic neuron. In this simulation, all synapse parameters are identical. When I_{syn} is injected into the associated neuron, its firing frequency changes and produces a 2-AG signal (green blocks in Fig. 2). The astrocyte receives the 2-AG signals from four neurons through the circuit shown in Fig. 4 and generates a positive signal (V_{ast}) in the tripartite synapse circuits. As the postsynaptic neuron's firing frequency changes, its internal calcium level detects the variation, and the negative feedback circuit shown in Fig. 3 applies the corresponding error compensation according to the global astrocyte signal. In the network without self-repair circuitry, astrocytic 2-AG and tripartite synapse signals are not utilized. Instead, simple synapses without negative feedback or astrocytes are employed. In this configuration, fixed voltages are applied to V_{set} and V_{pstv} to implement synapses without self-repair. The simulation results are shown in Fig. 6, where V_{set} and V_{pstv} in the synapse circuit are fixed at 2.7 V and 0.4 V, respectively. As shown, the firing frequencies of both circuits are identical. The initial input frequency difference is due to the non-fixed V_{set} and V_{pstv} in the tripartite synapses, highlighting the astrocyte's role in frequency adaptation.

Next, the synapses associated with neuron #2 are assumed to be defective. In this study, the proportion of defective synapses ranges from 0% to 80%. The total simulation time is 6 ms, and defects are introduced at $t > 3$ ms.

In the neural network without a self-repair mechanism, four neurons and 20 simple synapses are used instead of tripartite synapses (i.e., without additional self-repair circuitry). A defective synapse is defined as being disconnected from its associated postsynaptic neuron. When a synapse becomes defective, it can no longer inject current into its neuron. As a result, the total input current to the neuron decreases, leading to a reduction in its firing rate.

The simulation results are shown in Fig. 7. This simulation illustrates the network behavior in the absence of the self-repairing circuit. As expected, increasing the percentage of defective synapses associated with neuron #2 results in an abrupt decrease in its firing rate.

To evaluate the impact of defective synapses on the network equipped with the proposed self-repair mechanism, synapses connected to neuron #2 were systematically impaired from 0% to 80%. The corresponding simulation outcomes are presented in Fig. 8. The total simulation duration is 6 ms, with defects introduced at $t > 3$ ms. Following the onset of synaptic impairment, the network promptly attempts to detect and compensate for the resulting error. The results clearly demonstrate that the network is capable of performing effective

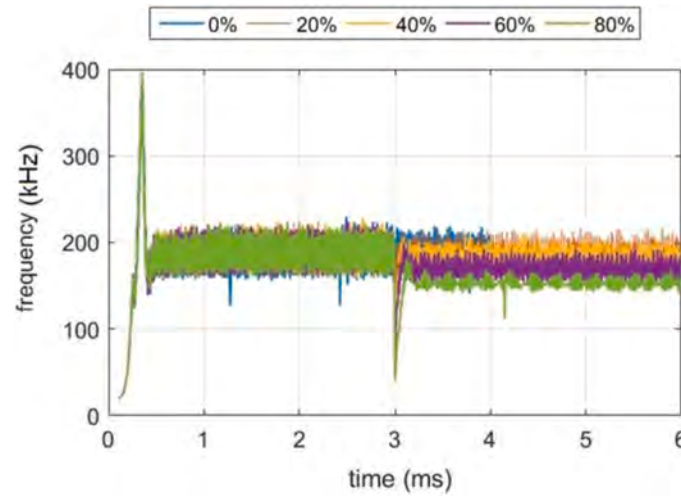


Fig. 8. Frequency of neuron # 2 under varying fault levels (0 – 80% fault). Synaptic defects are introduced at $t > 3$ ms for all cases.

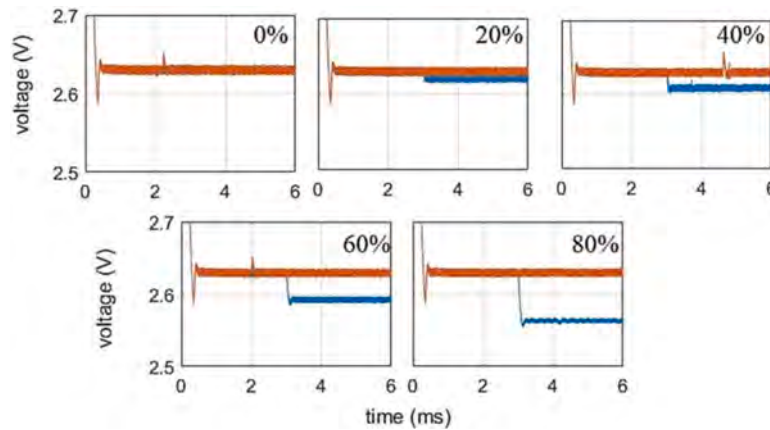


Fig. 9. Simulation results of V_{set} with the self-repair mechanism enabled for 0 – 80% faults in the synapses connected to neuron #2. The red curve shows V_{set} of the healthy synapses of neuron #4, while the blue curve represents V_{set} of the healthy synapses of neuron #2. Synaptic defects are introduced at $t > 3$ ms.

postsynaptic self-repair in response to synaptic degradation. Moreover, as the proportion of defective synapses increases, the compensatory capability of the remaining functional synapses gradually diminishes.

As shown by the comparison between Figs. 7 and 8, the neural network without the proposed self-repair mechanism fails to recover its firing frequency following the introduction of synaptic errors ($t > 3$ ms), revealing its inherent vulnerability to synaptic disruption. In contrast, the neural network incorporating the self-repair circuit exhibits a robust recovery: the postsynaptic neuron successfully restores its firing frequency to a satisfactory level even under high fault conditions. These results clearly highlight the effectiveness of the proposed self-repair mechanism in preserving both network stability and functional reliability.

When defects arise, the input current to the corresponding postsynaptic neuron decreases. This error is partially detected postsynaptically, triggering a compensatory response through a negative feedback circuit that reduces intercellular Ca^{2+} concentration. This resulting decrease in inhibitory current subsequently enhances I_{syn} in the remaining healthy synapses. Because the astrocyte output remains constant, the increase in I_{syn} among intact synapses effectively compensates for the induced effects.

Fig. 9 presents the simulation results, where V_{set} for the synapses of neuron #4 is shown in red, and V_{set} for the healthy synapses compensating for neuron #2 is depicted in blue. As illustrated, V_{set} in neuron #2's tripartite synapse progressively decreases as the defect percentage

increases, while the synapse associated with neuron #4 remains unaffected (red curve). This adaptive response constitutes the core principle of the proposed self-repair algorithm.

The results demonstrate that the proposed tripartite synapses play a pivotal role in enabling postsynaptic self-repair. This study shows that novel autonomous computational systems can be developed by leveraging biologically inspired mechanisms. The proposed astrocyte-neuron algorithm is implemented as an analog circuit that relies exclusively on astrocytes and the newly introduced tripartite synapses to realize self-repair functionality, without requiring additional auxiliary components. In this framework, the tripartite synapse constitutes the fundamental element that enables adaptive compensation. Furthermore, integrating this approach with presynaptic self-repair strategies could further enhance accuracy and improve biological plausibility.

To validate the scalability of the proposed design, its performance was evaluated in a larger three-layer neural network. The input layer consists of five neurons, the hidden layer comprises 15 neurons, and the output layer includes two neurons. Input currents are directly applied to the neurons in the first layer. Each hidden-layer neuron is randomly connected to three of the five input-layer neurons through 10 synapses (i.e., 10 synapses per hidden-layer neuron).

In addition, three astrocytes are positioned between the input and hidden layers, and two astrocytes are placed between the hidden and output layers. Each astrocyte in the first stage receives 2-AG input from a randomly selected subset of two input-layer neurons and five hidden-

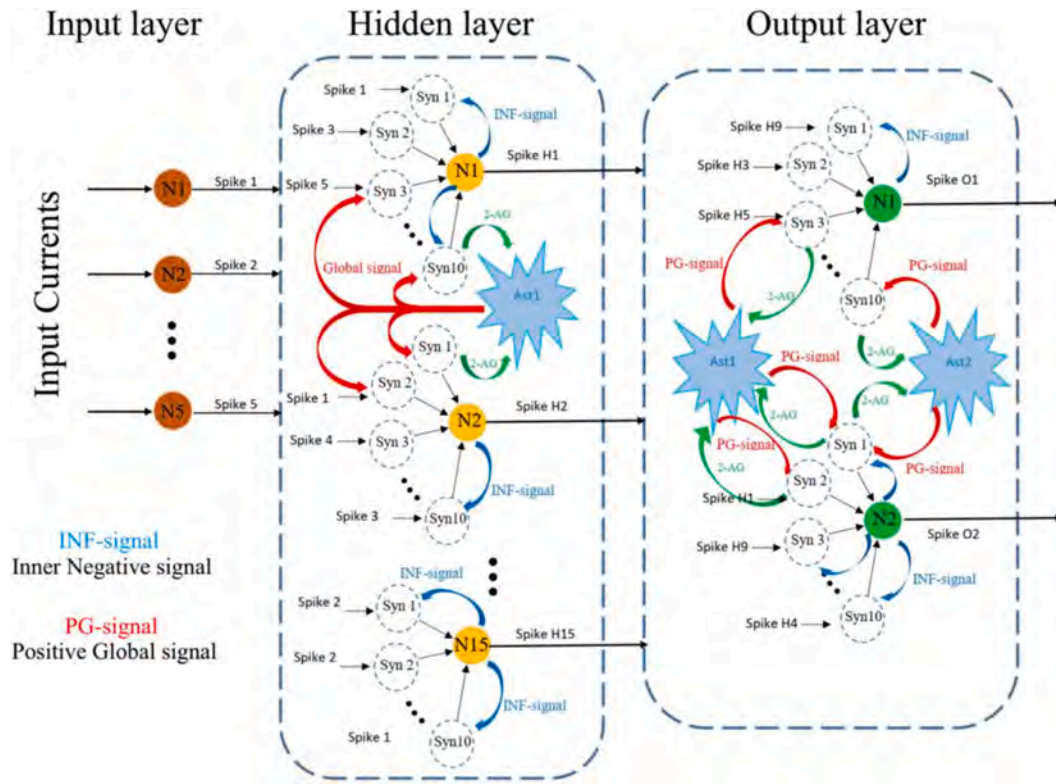


Fig. 10. Neural network incorporating the proposed self-repairing algorithm. The network consists of three layers, 17 neurons, five astrocytes, and 170 tripartite synapses. Each neuron includes 10 synapses that are randomly connected to neurons in the preceding layer. The INF-signal denotes the intercellular Ca^{2+} -mediated inhibitory signal, while the PG-signal represents the astrocyte-mediated global excitatory signal.

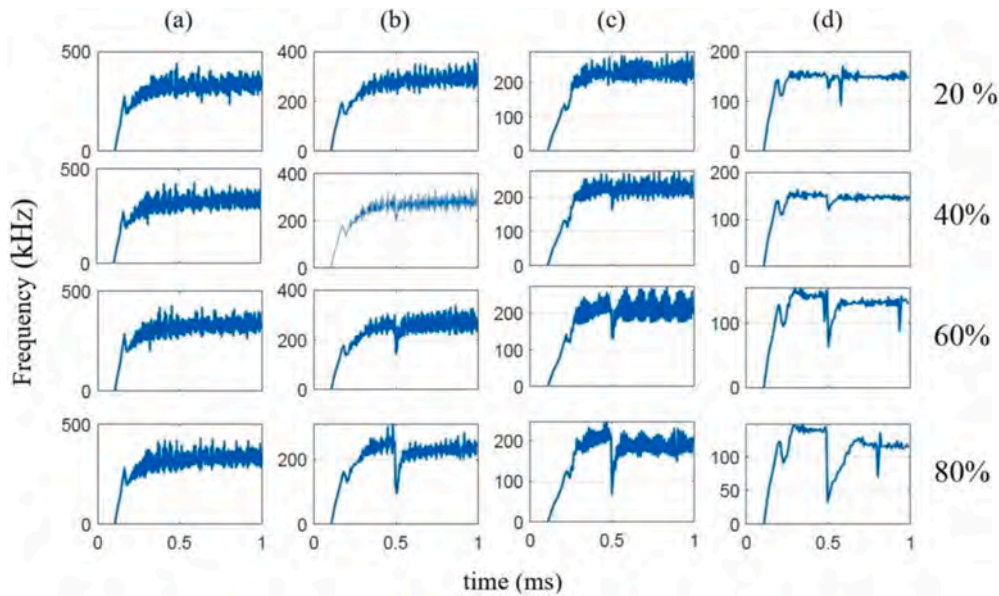


Fig. 11. Firing frequency of hidden layer neurons under 20% – 80% synaptic faults applied to three neurons (neurons #2, #7, #12). (a) Neuron #1 (no defects), (b) neuron #2, (c) Neuron #7 (d) Neuron #12. All neurons belong to the hidden layer. For each neuron, firing frequency is shown for 20%, 40%, 60% and 80% defective synapses (from top to bottom). Synaptic faults are introduced at $t > 0.5$ ms.

layer neurons. Astrocytes in the subsequent stage receive inputs from a random subset of six hidden-layer neurons and one output neuron. The overall network architecture is illustrated in Fig. 10.

All neurons in the network employ tripartite synapses, as illustrated in Fig. 3. When multiple synapses connected to a hidden-layer neuron become defective, the neuron's firing rate decreases due to reduced

glutamate input. Because the neighboring astrocyte receives 2-AG from seven surrounding neurons, the glutamate-dependent global signal delivered to other neurons remains approximately constant, keeping V_{SA} stable. The reduction in firing rate lowers intracellular calcium release in the postsynaptic neuron. As a result, the inhibitory currents at its associated synapses decrease, leading to an increase in excitatory

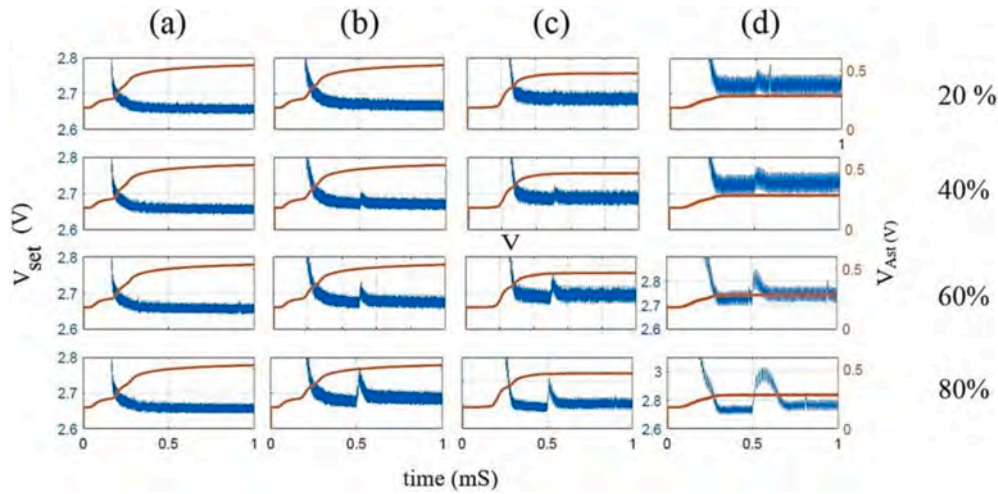


Fig. 12. Activity of selected neurons in the three-layer neural network under 20% – 80% synaptic faults. (a) Neuron #1 (no defects) (b) Neuron #2, (c) Neuron #7, and (d) Neuron #12. Red traces represent the astrocyte output voltage (V_{Ast}), while blue traces indicate the set-point voltage V_{set} generated by the postsynaptic intercellular negative feedback signal. Synaptic faults are applied to the selected neurons at different level (20% – 80%).

currents. This enhancement provides partial compensation for the reduced firing rate.

Input-layer neurons receive random external current ranging from 5 nA to 200 nA. In the hidden-layer, three neurons simultaneously contain defective synapses. Each of these neurons receives a distinct global signal from its corresponding astrocytes. These astrocytes (located between the input and hidden layers) receive randomized 2-AG inputs from different subsets of neurons, producing varying positive global signals. This configuration enables evaluation of the astrocyte's influence on the compensation process. The proportion of defective synapses per neuron ranges from 20% to 80%. The corresponding simulation results are presented in Fig. 11. At low defect levels, healthy synapses effectively compensate for the loss; however, at higher defect levels, the compensation becomes only partial.

In this figure, the first column shows the firing frequency of hidden-layer neuron 1, which has no defective synapses. Because the neurons examined in the simulation are connected to different astrocytes (each generating its own global signal), their firing frequencies differ. For hidden layer neuron 1, the firing rate remains stable unless its associated

astrocyte is significantly influenced by neighboring neurons. In this simulation, the number of defective synapses is small relative to the total synapses connected to each neuron, so astrocyte activity remains largely unaffected.

In this network, the healthy synapses of a neuron compensate for synaptic errors to the extent permitted by their current-generation capacity; however, complete (100%) error compensation is not achievable. The degree of compensation is fundamentally constrained by the maximum output current that the remaining functional synapses can provide. As illustrated in Fig. 11, when defects occur in a subset of synapses, the healthy synapses increase their output currents in an attempt to maintain the neuron's firing frequency near its original level. Consequently, the achievable maximum firing frequency depends on the magnitude of the global astrocyte-mediated signal. Because this global excitatory signal remains constant, the operational firing frequency of each neuron is primarily determined by the astrocyte to which it is connected. Due to the randomized network connectivity, neurons receive different global signals, resulting in variability in their firing rates.

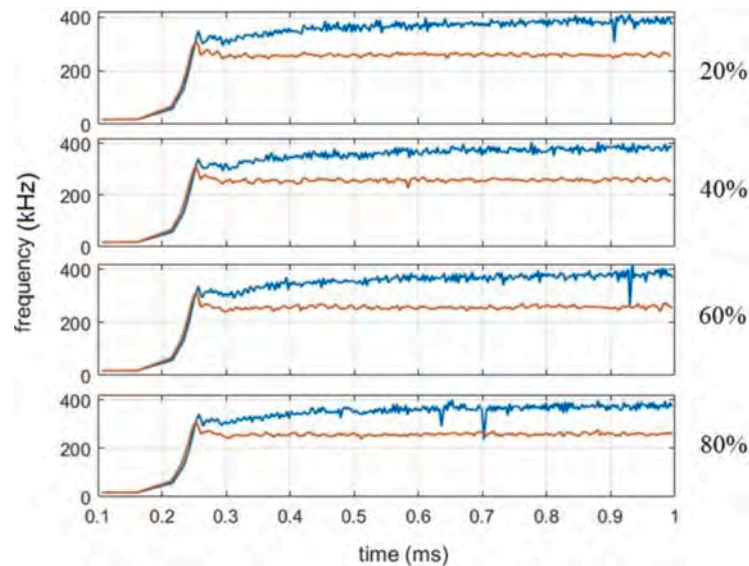


Fig. 13. Firing frequency of output-layer neurons under different levels of synaptic faults in the hidden layer. Faults affecting 20% – 80% of the synapses of hidden-layer neurons are compensated by the remaining healthy synapses. (Blue trace) Output-layer neuron #1, (Red trace) Output-layer neuron #2.

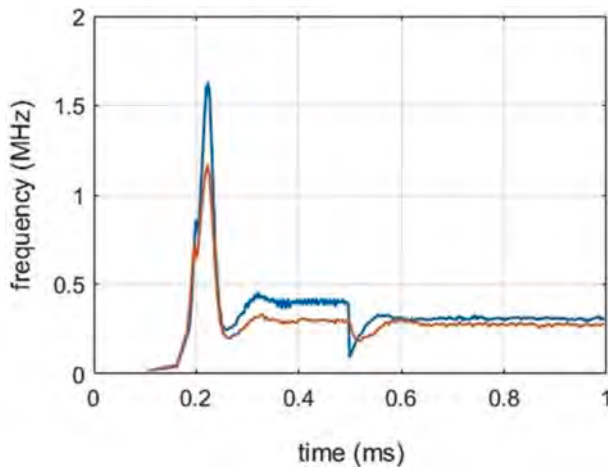


Fig. 14. Firing frequency of output layer neurons under a large propagation of defective synapses. (Blue trace) Output-layer neuron #1, (Red trace) Output-layer neuron #2.

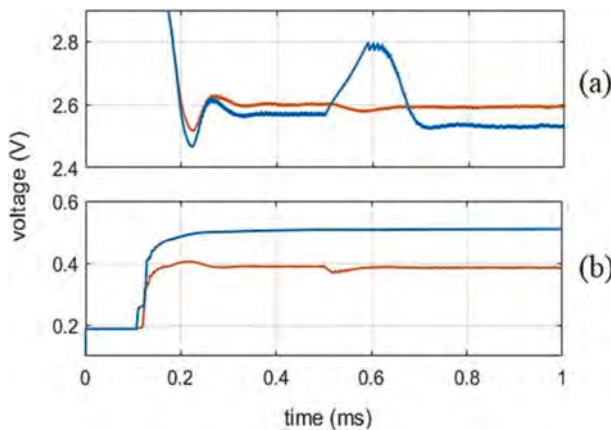


Fig. 15. (a) Set-point voltage V_{set} and (b) astrocyte output voltage V_{Ast} corresponding to the healthy tripartite synapses of the output-layer neurons. (Blue trace) Output-layer neuron #1 (Red trace), Output-layer neuron #2.

The results indicate that the effectiveness of synaptic compensation is directly linked to the activity level of the associated astrocyte. Fig. 12 illustrates the behavior of V_{set} (blue traces) and V_{Ast} (red traces) in the self-repairing compensation circuits. As the percentage of defective synapses increases, the efficiency of compensation decreases. When the astrocyte output (V_{Ast}) is lower, the feedback loop requires a longer time to achieve partial compensation, as demonstrated in the last column of Fig. 12.

As noted, the network connectivity is fully random. All tripartite synapses are assumed to have identical and fixed weights. The parameters V_g and V_T associated with the set-point controller in the tripartite synapses are kept constant, whereas the corresponding V_g and V_T values for the 2-AG signaling toward astrocytes are randomly selected within the ranges of 2 – 3.1 V and 2.75–3 V, respectively.

The effectiveness of synaptic error compensation depends on the output of the associated astrocyte. When an astrocyte generates a sufficiently strong positive global signal, it enhances the capability of the tripartite synapses to compensate for synaptic faults. The functional capacity of an astrocyte is therefore influenced by both the number and the activity of the neurons connected to it. As illustrated in Fig. 13, compensation of synaptic errors in the hidden layer does not significantly affect the activity of the output neurons.

In this simulation scenario, the outputs of both neurons in the output layer are presented. As clearly observed, the firing frequencies of both

neurons remain unchanged despite faults affecting 20% – 80% of the synapses of a hidden-layer neuron. These defects are effectively compensated by the remaining healthy synapses within the network.

Due to the random network connectivity, the impact of synaptic faults strongly depends on the specific neuron-astrocyte interconnections. When faults occur, successful compensation requires a transient period whose duration is proportional to the percentage of defective synapses. Once the astrocyte output is perturbed, extending the simulation time does not significantly improve the compensation process.

As a representative case, a scenario was generated in which 50% of the synapses in the second-layer were defective (equivalent to 25% of the total network synapses). The corresponding results are shown in Figs. 14 and 15. In this case, 50% of the synapses connected to output neuron #1 were defective. Astrocytes associated with neurons containing faulty synapses were more strongly affected, particularly the second astrocyte. The degradation of astrocyte output reduces the effectiveness of synaptic compensation.

For $t < 5$ ms, the firing rate of neuron #1 is noticeably affected; however, the remaining healthy synapses provide partial compensation. In contrast, the synapses connected to output neuron #2 remain intact, and its associated astrocyte initially remains stable. Although hidden-layer faults indirectly influence the astrocyte connected to neuron #2, the output astrocytes gradually recover, leading to the restoration of neuron #2's firing rate.

Thus, the compensation capability of the network is determined by the type, number, and effective weights of neuron-astrocyte connections across different layers. Notably, the proposed mechanism can compensate for both presynaptic and postsynaptic faults, providing a significant advantage over presynaptic-only self-repair circuits (PR method).

4. Discussion

Self-repair is a fundamental phenomenon in biological neural networks, emerging from the cooperative interaction between neurons and astrocytes. Astrocytes play a central role in maintaining neural stability by modulating neurotransmission and supporting synaptic regulation through distinct signaling pathways that influence both presynaptic and postsynaptic compartments.

The proposed tripartite synapse framework introduces a postsynaptic self-repair mechanism mediated by astrocyte signaling. In this model, the repair process is implemented exclusively in the postsynaptic pathway, where intracellular calcium-dependent inhibitory feedback and astrocyte-driven global excitatory signaling jointly regulate synaptic activity. This mechanism stabilizes neuronal firing and preserves network functionality even when multiple synaptic faults occur.

A key advantage of the proposed approach is that, although the repair mechanism operates solely at the postsynaptic side, it can effectively compensate for faults originating from both presynaptic and postsynaptic sources. This compensation arises from the regulation of postsynaptic activity and synaptic current through astrocyte-mediated feedback, which stabilizes neuronal firing despite disturbances in presynaptic neurotransmission. Unlike presynaptic self-repair circuits (which directly adjust neurotransmitter release), the proposed architecture does not implement a presynaptic repair loop. Instead, it mitigates the functional impact of such faults through postsynaptic regulation within the tripartite synapse. This property provides a robust and biologically plausible strategy for maintaining network stability under substantial synaptic perturbations.

Simulation results further highlight the importance of astrocyte-mediated global feedback in achieving efficient fault compensation. Strengthening the astrocytic global signal (V_{Ast}) significantly enhances the network's ability to maintain stable firing patterns across a wide range of fault densities. Consequently, the effectiveness of error compensation depends on the type, number, and effective weights of neuron-astrocyte connections across different network layers. As long as

Table 1

Comparison of the proposed work with previous studies.

Ref.	Pre/Post-synaptic self-repairing	Type homeostatic	With astrocyte or Glia cell	Analog/Digital (A/D)	Fabrication/Simulation
[28]	Pre	Weight scaling (PR method)	Yes	Simulation	Sim.
[29]	Pre	Weight scaling (PR method)	Yes	Simulation	Sim.
[33]	Post	Internal threshold	No	A/D	Fab.
[34]	Pre/Post	Weight scaling/internal threshold	No	A/D	Fab.
[36]	Pre	Weight scaling (PR method)	Yes	A	Sim.
[37]	pre	Weight scaling (PR method)	Yes	A	Sim.
[39]	-	Weight scaling (PR method)	No	memristive	Sim.
[47]	Pre	Weight scaling (PR method)	Yes	Simulation	Sim.
[48]	pre	Weight scaling (PR method)	No	memristive	Sim.
Our work	post	Intercellular Ca^{2+} / synaptic scaling	Yes	A	Sim.

astrocytic functionality remains intact, the network can sustain stable output despite the presence of faults in various synaptic compartments.

Compared with presynaptic self-repair circuits (which compensate for presynaptic faults primarily by increasing glutamate release) the proposed postsynaptic mechanism offers a more flexible compensation strategy capable of mitigating the effects of faults in either synaptic compartment. Table 1 summarizes the main contributions of this work relative to previous studies. Most existing approaches have focused on presynaptic repair mechanisms, likely due to their simpler implementation. In contrast, prior investigations of postsynaptic regulation have often excluded astrocytic participation and therefore lacked the feedback structure required for effective biologically inspired self-repair. Although some studies have reported beneficial roles for astrocytes in postsynaptic processes related to learning and memory [10,23] the absence of explicit negative-feedback mechanisms (such as depolarization-induced suppression of excitation (DSE) or the calcium-dependent inhibitory regulation adopted in this work) limits their ability to achieve true fault compensation. These studies nevertheless offer an interesting direction for future extensions: incorporating the proposed hardware tripartite synapse into similar neuron-astrocyte network frameworks to examine its system-level impact. Given the regulatory role provided by the explicit inhibitory feedback in our model, such integration is likely to yield more stable dynamics and potentially improved computational performance, making it a promising avenue for future research.

The modular architecture of the proposed astrocyte-neuron unit inherently supports scalability without increasing the intrinsic complexity of the underlying circuit design. This feature is particularly advantageous for large-scale neuromorphic implementations. However, a critical consideration in such scaling is the proper constraint of both the number and the effective synaptic weights connected to each astrocyte. Without these constraints, the astrocyte output may reach saturation, thereby limiting overall system performance and reliability.

It should be noted that the present model focuses specifically on postsynaptic repair dynamics and does not explicitly model presynaptic repair circuitry. Exploring hybrid architectures that integrate both presynaptic and postsynaptic repair processes may provide a valuable direction for future research. Further work may also investigate the scalability of the proposed framework in larger and more complex neural architectures, as well as its potential application in neuromorphic and artificial intelligence systems requiring enhanced reliability and fault tolerance. Considering the conceptual relevance between the present study and the findings reported in [24], future research could explore the proposed tripartite synapse architecture in the context of noisy neural information processing to further examine astrocyte-mediated effects. Ref. [49] introduces an auxiliary neuron with a memristor-based synapse to provide an alternative error-compensation pathway in a small network without astrocytes. As future work, this architecture could be extended using the proposed tripartite synapses.

Funding source

DA gratefully acknowledges funding from the Australian Research Council (FL240100217).

CRediT authorship contribution statement

Mahnaz Ranjbar: Writing – original draft, Validation, Software, Resources, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Gholamreza Karimi:** Writing – review & editing, Validation, Supervision, Project administration, Methodology, Investigation, Conceptualization. **Ali Khosravi:** Validation, Software, Investigation. **Derek Abbott:** Writing – review & editing, Validation, Funding acquisition.

Declaration of competing interest

The authors declare no competing interests.

Data availability

Data generated during the current study will be made available on reasonable request.

References

- [1] S.T. Carmichael, Cellular and molecular mechanisms of neural repair after stroke: making waves, *Ann. Neurol.: Off. J. Am. Neurol. Assoc. Child Neurol. Soc.* 59 (5) (2006) 735–742.
- [2] E.S. Akdemir, A.Y.S. Huang, B. Deneen, Astrocytogenesis: where, when, and how, *Front. Neurosci.* 9 (2020) 233, <https://doi.org/10.12688/f1000research.22405.1>.
- [3] B.E. Alger, Retrograde signaling in the regulation of synaptic transmission: focus on endocannabinoids, *Prog. Neurobiol.* 68 (4) (2002) 247–286, [https://doi.org/10.1016/S0304-0082\(02\)00080-1](https://doi.org/10.1016/S0304-0082(02)00080-1).
- [4] E.C. Kugler, J. Greenwood, R.B. MacDonald, The “neuro-glial-vascular” unit: the role of glia in neurovascular unit formation and dysfunction, *Front. Cell Dev. Biol.* 9 (2021) 732820, <https://doi.org/10.3389/fcell.2021.732820>.
- [5] S. Sun, D. Levenstein, B. Li, N. Mandelberg, N. Chenouard, B.S. Suutari, S. Sanchez, R.W. Tsien, Synaptic homeostasis transiently leverages Hebbian mechanisms for a multiphasic response to inactivity, *Cell Rep.* 43 (4) (2024) 113839.
- [6] Y. Wang, X. Chen, C. Zhang, S. Gao, Multi-scale neural homeostasis mechanisms: insights into neurodegenerative diseases and therapeutic approaches, including exercise, *Adv. Exerc. Heal. Sci.* 2 (2025) 1–15.
- [7] P.H. Chipman, U. Lee, B.O. Orr, R.D. Fetter, G.W. Davis, A unifying mechanism for presynaptic homeostatic plasticity at mammalian peripheral and central synapses, *Neuron* 113 (2025) 2945–2961.e6.
- [8] G. Turrigiano, Homeostatic synaptic plasticity: local and global mechanisms for stabilizing neuronal function, *Cold. Spring. Harb. Perspect. Biol.* 4 (1) (2012) a005736.
- [9] C.A. Frank, T.D. James, M. Müller, Homeostatic control of Drosophila neuromuscular junction function, *Synapse* 74 (1) (2020) e22133.
- [10] R. Vuillaume, J. Lorenzo, S. Binczak, S. Jacquir, A computational study on synaptic plasticity regulation and information processing in neuron-astrocyte networks, *Neural Comput.* 33 (7) (2021) 1970–1992.
- [11] G.G. Turrigiano, The self-tuning neuron: synaptic scaling of excitatory synapses, *Cell* 135 (3) (2008) 422–435.
- [12] T.J. Sullivan, V.R. de Sa, A self-organizing map with homeostatic synaptic scaling, *Neurocomputing* 69 (10–12) (2006) 1183–1186.
- [13] K.D. Miller, Synaptic economics: competition and cooperation in synaptic plasticity, *Neuron* 17 (3) (1996) 371–374.

- [14] G. Turrigiano, Too many cooks? Intrinsic and synaptic homeostatic mechanisms in cortical circuit refinement, *Annu. Rev. Neurosci.* 34 (1) (2011) 89–103.
- [15] G.G. Turrigiano, K.R. Leslie, N.S. Desai, L.C. Rutherford, S.B. Nelson, Activity-dependent scaling of quantal amplitude in neocortical neurons, *Nature* 391 (6670) (1998) 892–896.
- [16] W.B. Cannon, C.E. Rosenberg, Homeostasis, *Wisdom Body* (1932) 263–286.
- [17] D. Stellwagen, R.C. Malenka, Synaptic scaling mediated by glial TNF- α , *Nature* 440 (7087) (2006) 1054–1059.
- [18] G.W. Davis, M. Müller, Homeostatic control of presynaptic neurotransmitter release, *Annu. Rev. Physiol.* 77 (1) (2015) 251–270.
- [19] D.S. Auld, R. Robitaille, Glial cells and neurotransmission: an inclusive view of synaptic function, *Neuron* 40 (2) (2003) 389–400.
- [20] L. Hertz, R. Dringen, A. Schousboe, S.R. Robinson, Astrocytes: glutamate producers for neurons, *J. Neurosci. Res.* 57 (4) (1999) 417–428, [https://doi.org/10.1002/\(SICI\)1097-4547\(19990815\)57:4%3C417::AID-JNR1%3E3.0.CO;2-N](https://doi.org/10.1002/(SICI)1097-4547(19990815)57:4%3C417::AID-JNR1%3E3.0.CO;2-N).
- [21] L. Hertz, H.R. Zielke, Astrocytic control of glutamatergic activity: astrocytes as stars of the show, *Trends. Neurosci.* 27 (12) (2004) 735–743, <https://doi.org/10.1016/j.tins.2004.10.008>.
- [22] M. Navarrete, A. Araque, Endocannabinoids potentiate synaptic transmission through stimulation of astrocytes, *Neuron* 68 (1) (2010) 113–126, <https://doi.org/10.1016/j.neuron.2010.08.043>.
- [23] J. Lorenzo, J.A. Rico-Gallego, S. Binczak, S. Jacquir, Spiking neuron-astrocyte networks for image recognition, *Neural. Comput.* 37 (4) (2025) 635–665.
- [24] T. Palabas, J.J. Torres, M. Perc, M. Uzuntarla, Double stochastic resonance in neuronal dynamics due to astrocytes, *Chaos Solit. Fractals* 168 (2023) 113140.
- [25] J. Lorenzo, R. Vuillaume, S. Binczak, S. Jacquir, Spatiotemporal model of tripartite synapse with perinodal astrocytic process, *J. Comput. Neurosci.* 48 (1) (2020) 1–20.
- [26] D.W. Garcia, S. Jacquir, Astrocyte-mediated neuronal irregularities and dynamics: the complexity of the tripartite synapse, *Biol. Cybern.* 118 (5) (2024) 249–266.
- [27] D.W. Garcia, S. Jacquir, From quiescence to self-sustained activity: how astrocytes reshape neural dynamics, *Neuroscience* 576 (2025) 182–198.
- [28] Z. Han, A.N. Islam, A. Sengupta, Astromorphic self-repair of neuromorphic hardware systems, *Proc. AAAI Conf. Artif. Intell.* 37 (6) (2023, June) 7821–7829.
- [29] M. Rastogi, S. Lu, N. Islam, A. Sengupta, On the self-repair role of astrocytes in STDP enabled unsupervised SNNs, *Front. Neurosci.* 14 (2021) 603796, <https://doi.org/10.3389/fnins.2020.603796>.
- [30] J. Wade, L.J. McDaid, J. Harkin, V. Crunelli, S. Kelso, Self-repair in a bidirectionally coupled astrocyte-neuron (AN) system based on retrograde signaling, *Front. Comput. Neurosci.* 6 (2012) 26578, <https://doi.org/10.3389/fncom.2012.00076>.
- [31] M. Naeem, L.J. McDaid, J. Harkin, J.J. Wade, J. Marsland, On the role of astroglial syncytia in self-repairing spiking neural networks, *IEEE Trans. Neural. Netw. Learn. Syst.* 26 (10) (2015) 2370–2380, <https://doi.org/10.1109/TNNLS.2014.2382334>.
- [32] P. Naghieh, A. Delavar, M. Amiri, H. Peremans, Astrocyte's self-repairing characteristics improve working memory in spiking neuronal networks, *iScience* 26 (12) (2023) 108241, <https://doi.org/10.1016/j.isci.2023.108241>.
- [33] I. Muñoz-Martin, S. Bianchi, S. Hashemkhani, G. Pedretti, O. Melnic, D. Ielmini, A brain-inspired homeostatic neuron based on phase-change memories for efficient neuromorphic computing, *Front. Neurosci.* 15 (2021) 709053.
- [34] S.C. Liu, B. Minch, Homeostasis in a silicon integrate and fire neuron, *Adv. Neural. Inf. Process. Syst.* 13 (2000).
- [35] J. Liu, J. Harkin, L.P. Maguire, L.J. McDaid, J.J. Wade, SPANNER: a self-repairing spiking neural network hardware architecture, *IEEE Trans. Neural. Netw. Learn. Syst.* 29 (4) (2017) 1287–1300, <https://doi.org/10.1109/TNNLS.2017.2673021>.
- [36] N. Veisi, G. Karimi, M. Ranjbar, D. Abbott, Role of astrocytes in the self-repairing characteristics of analog neural networks, *Neurocomputing* 496 (2022) 158–165, <https://doi.org/10.1016/j.neucom.2022.01.077>.
- [37] E. Rahiminejad, F. Azad, A. Parvizi-Fard, M. Amiri, B. Linares-Barranco, A neuromorphic CMOS circuit with self-repairing capability, *IEEE Trans. Neural. Netw. Learn. Syst.* 33 (5) (2021) 2246–2258, <https://doi.org/10.1109/TNNLS.2020.3045019>.
- [38] N. Qiao, C. Bartolozzi, G. Indiveri, An ultralow leakage synaptic scaling homeostatic plasticity circuit with configurable time scales up to 100 ks, *IEEE Trans. Biomed. Circuits. Syst.* 11 (6) (2017) 1271–1277.
- [39] Q. Wan, K. Sun, T. Liu, P. Qin, Memristive neural network circuit of negative emotion inhibition with self-repair and memory, *Int. J. Circuit. Theory Appl.* 53 (8) (2025) 4890–4903.
- [40] M. Ranjbar, M. Amiri, On the role of astrocyte analog circuit in neural frequency adaptation, *Neural Comput. Appl.* 28 (2017) 1109–1121, <https://doi.org/10.1007/s00521-015-2112-8>.
- [41] K.E. Poskanzer, R. Yuste, Astrocytes regulate cortical state switching in vivo, *Proc. Natl. Acad. Sci. U.S.A.* 113 (19) (2016) E2675–E2684.
- [42] J.H. Wijekoon, P. Dudek, Compact silicon neuron circuit with spiking and bursting behaviour, *Neural Netw.* 21 (2–3) (2008) 524–534, <https://doi.org/10.1016/j.neunet.2007.12.037>.
- [43] E.M. Izhikevich, Simple model of spiking neurons, *IEEE Trans. Neural Netw.* 14 (6) (2003) 1569–1572, <https://doi.org/10.1109/TNN.2003.820440>.
- [44] M. Ranjbar, M. Amiri, Analog implementation of neuron–astrocyte interaction in tripartite synapse, *J. Comput. Electron.* 15 (2016) 311–323, <https://doi.org/10.1007/s10825-015-0727-8>.
- [45] G. Montaseri, M.J. Yazdanpanah, M. Amiri, Astrocyte-inspired controller design for desynchronization of two coupled limit-cycle oscillators, in: *Third World Congress on Nature and Biologically Inspired Computing*, IEEE, 2011, October, pp. 195–200, <https://doi.org/10.1109/NaBIC.2011.6089458>.
- [46] C. Bartolozzi, G. Indiveri, Synaptic dynamics in analog VLSI, *Neural Comput.* 19 (10) (2007) 2581–2603, <https://doi.org/10.1162/neco.2007.19.10.2581>.
- [47] J. Liu, L.J. McDaid, J. Harkin, S. Karim, A.P. Johnson, A.G. Millard, J. Hilder, D. M. Halliday, A.M. Tyrrell, J. Timmis, Exploring self-repair in a coupled spiking astrocyte neural network, *IEEE Trans. Neural Netw. Learn. Syst.* 30 (3) (2018) 865–875, <https://doi.org/10.1109/TNNLS.2018.2854291>.
- [48] Q. Hong, H. Chen, J. Sun, C. Wang, Memristive circuit implementation of a self-repairing network based on biological astrocytes in robot application, *IEEE Trans. Neural Netw. Learn. Syst.* 33 (5) (2020) 2106–2120, <https://doi.org/10.1109/TNNLS.2020.3041624>.
- [49] L. Chen, L.I. Ch, A forgetting memristive spiking neural network for Pavlov experiment, *Int. J. Bifurc. Chaos* 28 (96) (2018) 1850080.