A Digital Realization of Neuroglial Interaction Model and Its Network Structure

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ABSTRACT Astrocyte cells, the most existing abundant cells in central nervous system, play an essential role in modulating the neuronal activities, information processing, and regulating the synaptic plasticity through calcium (Ca2+) fluctuations of ions hemostasis by using a feedback mechanism. The pathophysiological and hypersynchronous neuronal activity can lead to the epileptic seizures, which is known as one of the neurodegenerative disorders in the field of neuroscience. This paper presents a modified neuron-astrocyte interaction (tripartite synapse) model based on leaky and integrate fire neuron and the astrocyte-synapse models using an area-efficient hardware approach called stochastic computing paradigm. The proposed model is synthesized physically on field-programmable gate array as a proof of concept. The implementation results of the presented model can mimic the bidirectional communication in biological minimal network of pre-postsynaptic and Ca2+-based model for astrocyte with considerably lower hardware cost. The influence of astrocytes on neural network behavioral has been investigated by providing a proper feedback mechanism and considering the role of gap junction coupling and the various coefficients on desynchronizing the impaired synchronization of the coupled neurons.

INDEX TERMS Astrocytes, desynchronization, field programmable gate array (FPGA), neural-glial interaction.

I. INTRODUCTION

Epilepsy is one of the neurological malfunction disorders which occurs due to the unusual patterns of neuronal firing and hypersynchronous neuronal activities. Authors in [1] and [2] have investigated the impact of astrocytes during epilepsy in synchronizing the coupled neurons in network level by proposing different functional modeling. One of the fundamental elements which are involved in the development of enormous neuron motor disorders are astrocytic cells. Astrocytes within central nervous system (CNS) are involved for modulating synaptic plasticity and transmission between neurons through hemostasis and metabolic processes [3].

Recently, neuromorphic VLSI implementation of neural networks has been investigated to discover the implementation of the large-scale Spiking Neural networks [4], [5].

Hardware implementation of various biological models can be achieved using mainly two different platforms. First approach is analog implementation which provides an efficient and low-cost design in terms of occupied area, hardware resources, and power consumption but its development time is longer, its vulnerability to noise is higher, and it is an inflexible approach in comparison to digital platforms. In [6] and [7], analog designs are proposed for the biological models. Alternatively, digital implementation, which has recently become a popular approach to bio-inspired computing which can offer a flexible and reconfigurable design. Digital platforms require a shorter development time in comparison to analog platforms. In this sense, FPGA-based designs can provide a higher level of precision and stability than other types of designs [8].

In this paper, a computationally low-cost hardware based tripartite synapse model according to a neuroglial interaction model has been proposed and implemented on FPGA which
is able to mimic the original behavior of the tripartite synapse model. The main contributions of this paper are listed as follows:

- An efficient digital design for a minimal network using Stochastic Computing technique which can mimic the original architecture of neuron-glial interaction.
- Investigating the influence of astroglia network on controlling the neuronal hyperexcitability during epileptic seizures by stabilizing the asynchrony behavioural between the coupled neurons.
- The proposed design is implemented on both hardware and software and has been compared with the original and other similar designs of hardware tripartite synapse models.

The rest of the paper is organized as follows. Section II discovers the related works. A complete tripartite synapse model which is composed of two neurons, an astrocyte, and the synapse model has been discussed in section III. Section IV discusses the proposed structure. Hardware implementation based on the modified model is presented in Section V. Section VI presents a neuroglial network model. The implementation results are shown in Section VII. This article is concluded in Conclusion section.

II. RELATED WORKS

Many neuropathological disorders such as epileptic seizures [9], Parkinson’s disease [10], and sleep-related disturbances [11] are required to be carefully studied in order to discover possible pathways for diagnostic and treatment approaches. In recent years, bidirectional interaction between astrocytic network and neuronal activity have been determined in the area of computational neuroscience. This interaction can be understood in a feedback-based manner to accommodate the intermittent neuronal synchrony.

Different models are proposed for modelling the bidirectional communication for neuron-astrocyte interactions to demonstrate the impact of astrocytes on the dynamics of spiking activity in neural network [12], [13], [14], [15]. In these articles, a mathematical model for neuron-astrocyte interaction system is proposed which can describe the tripartite synapse including pre-postsynaptic neurons, Ca2+ oscillations in astrocyte and synapse model. Calcium elevation in astrocyte can mediate the gliotransmitter release such as gamma aminobutyric acid (GABA), adenosine triphosphate (ATP), glutamate which results neuronal excitability through a feedback mode and can activate postsynaptic neurons at other synaptic terminals. On the other hand, glial cells can communicate with each other via Gap Junctions by transmission of IP3 molecules and Ca2+ waves propagations. Di Garbo and colleagues in [12] proposed a minimal biological neural network for describing the glial-neuron interaction which considered the impact of ATP on modulating neuronal activities. A dressed neuron model by describing the neuron-astrocyte interaction is proposed by Jung and Nadkarni [13]. A mathematical model is proposed for the elements of tripartite synapse in [14] and [15] which can reproduce the dynamical patterns of the biological neuron-astrocyte network.

Several hardware implementations for various biological calcium-based neuron-astrocyte models are proposed on FPGA platforms in [16], [17], [18], [19], and [20]. There are some approaches that can be employed to develop digital neuromorphic circuits. One of these approaches is base-2 method in which nonlinear terms of biologically inspired models can be replaced with base-2 functions and physically implemented using logical shift and add operations on hardware [20], [21]. The next approach is CORDIC (coordinate rotation digital computer) structure which is involved in computing complicated nonlinear functions through simple shift and add operations [16], [22]. This technique can cause the simplicity of the hardware implementation for the biologically plausible models; however, the main drawback associated with CORDIC paradigm is the latency issue. Piece-wise linear approximation method can be used to eliminate the nonlinearity of the biologically plausible models by utilizing several linear segments [17], [23]. In order to elevate the accuracy of the design, the number of linear segments are needed to be increased which can result in escalating the hardware cost. The last method is called stochastic computing (SC) approach which is an efficient hardware-based scheme. This method uses simple digital circuits to perform the arithmetic operations and is proposed initially in 1960s [24]. This architecture can be represented in both bipolar and unipolar formats [25], [33]. The main concept of this probabilistic-based computing approach is to process data by utilizing the digitized probabilistic unary stream. In recent years, SC method has gained greater insight in hardware realization of neural networks and neuromorphic computing studies due to its error-tolerant inherent and the optimized performance in terms of power and occupied area [26]. In [27], authors have proposed a hardware based tripartite synapse including Leaky Integrate-and-Fire (LIF) neuron, synapse, and astrocyte using Stochastic Computing (SC) and the Extended Stochastic Logics (ESLs). ESLs has been introduced as new method for SC for the hardware design of neural network applications [28]. In their work [27], astrocyte model is approximated using piece wise linear approximation method. Recently, a digital design for an optimized SC-based Izhikevich spiking neuron model is presented in [29].

The main predominancy of the proposed model over the recently published SC-based neuroglial model in [27] is that a detailed architecture for astroglial model and the hardware device utilization is discussed within this research study.

III. MATERIAL AND METHOD

A. NEURON MODEL

In this paper, leaky integrate-and-fire (LIF) neuron model with adaptation current which is one of the simplest spiking neuron models has been used to describe the spiking behaviour of the presynaptic and postsynaptic neurons. LIF spiking neuron fires when the membrane voltage becomes greater than threshold level. This model can be described
by the following differential equations, respectively [30] and [43]:

\[
\begin{align*}
C \frac{dV_{Pre}}{dt} &= -g_L (V_{Pre} - E_L) - I_{Pre} \\
\frac{dw_{Pre}}{dt} &= -\frac{1}{\tau} (w_{Pre}) \\
C \frac{dV_{Post}}{dt} &= -g_L (V_{Post} - E_L) - I_{Post} \\
\frac{dw_{Post}}{dt} &= -\frac{1}{\tau} (w_{Post})
\end{align*}
\tag{1}
\]

where \( C, V_{Pre}, V_{Post}, w_{Pre}, w_{Post}, g_L, E_L \), and \( \tau \) present the membrane capacitance, membrane potentials and variable of adaptation currents for presynaptic and post synaptic neurons, the conductance of leaky channels, the leak reversal potential, and time constant for adaptation current, respectively. The following listed equations are used to describe the behavior of the presynaptic \( I_{Pre} \) and postsynaptic currents \( I_{Post} \):

\[
\begin{align*}
I_{Pre} &= I_{applied} + I_a \\
I_{Post} &= I_e + I_{Glu} + I_{ATP} + I_a \\
I_{Glu} &= \gamma G_m \\
I_{ATP} &= \eta G_a \\
I_e &= g_e (V - E_e) \\
I_a &= w (V - E_k)
\end{align*}
\tag{2}
\]

where presynaptic LIF neuron model is stimulated only by an external stimulus current and adaptation current represented as \( I_{applied} \) and \( I_a \), respectively.

Postsynaptic current for LIF neuron shown by \( I_{Post} \) which includes astrocytic glutamate release, hydrolysis of ATP, excitatory synaptic, and adaptation currents that have been illustrated by \( I_{Glu}, I_{ATP} \), and \( I_e \), respectively. In synaptic and adaptation currents, \( g_e \) represents the value of excitatory conductance, \( E_e \) and \( E_k \) are the excitatory reversal potential the reversal potential of potassium. \( \gamma \) and \( \eta \) are the coupling coefficients for the variable \( G_m \) and the variable \( G_a \), respectively.

**B. ASTROCYTE MODEL**

The neurotransmitter release (ATP, GABA, Glutamate) forms the synaptic cleft can trigger the secondary mediator IP3 production in the glial cells which leads the elevation levels of Ca\(^{2+}\) concentration. This elevation of Ca\(^{2+}\) oscillation can result in activation of postsynaptic neurons. The interaction model between the coupled neurons and astrocyte can be governed by the following nonlinear differential equations which describes the Ca\(^{2+}\) dynamics between extracellular space and cytoplasm [14], [15]:

\[
\begin{align*}
T_c \frac{dc}{dt} &= -c - c_0 f (c, c_e) + r + \beta S_m + \alpha w \\
\varepsilon_c T_c \frac{dc_e}{dt} &= f (c, c_e) \\
T_{Sm} \frac{dS_m}{dt} &= [1 + \tanh (s_{Sm}(z - h_{Sm}))](1 - S_m) - \frac{S_m}{d_{Sm}} \\
T_{Gm} \frac{dG_m}{dt} &= [1 + \tanh (s_{Gm}(c - h_{Gm}))](1 - G_m) - \frac{G_m}{d_{Gm}} \\
T_{Ga} \frac{dG_a}{dt} &= [1 + \tanh (s_{Ga}(c - h_{Ga}))](1 - G_a) - \frac{G_a}{d_{Ga}}
\end{align*}
\tag{4}
\]

where \( c \) and \( c_e \) are the astrocytic calcium concentration in cytoplasm and calcium concentration in endoplasmic reticulum, respectively. \( f (c, c_e) \) is used to describe the dynamic behavior of Ca\(^{2+}\) between cytoplasm and the endoplasmic reticulum, and glutamaters, \( G_m \) and \( G_a \) present astrocytic glutamate release and ATP production, respectively. The secondary messenger production is shown by variable \( S_m \) and controlled by element of \( \beta \) which can be activated by synaptic terminal. \( r \), \( c_a \), and \( \varepsilon_c \) represent the transmembrane current, constant for variable \( c \), and the time separation constant. The component of \( \alpha w \) defines the potassium activation pathway. The threshold values in the listed equations are named as \( h_{Sm}, h_{Ga}, \) and \( h_{Ga} \) used for activation and inactivation states of variable \( z \) and \( c \), respectively. \( T_c, T_{Sm}, T_{Gm}, \) and \( T_{Ga} \) are used to show the controlling time scale for the existing variables. The controlling parameter of deactivation rates for each variable are shown by \( d_{Sm}, d_{Gm}, \) and \( d_{Ga} \). The controlling parameter of steepness of activation for each variable are shown by \( s_{Sm}, s_{Gm}, \) and \( s_{Ga} \). Variable \( z \) belongs to the synapse model. The complete structure for the tripartite synapse model which contains the main elements involved in the slow activation pathway is depicted in Fig. 1. The \( K \) activation pathway term has not been considered for the total calcium flux architecture. Parameter values that are employed for the simulation process are listed as Table. 1.

**C. SYNAPSE MODEL**

In this network, the functions \( z_i(t) \) and \( z_{ei}(t) \) are used to define the time evolution of the inhibitory and excitatory currents between presynaptic and postsynaptic neurons, respectively which have been governed by the following equations [12]:

\[
\begin{align*}
\frac{dz}{dt} &= T_i (1 - z_i) - \frac{z_i}{\tau_i} \\
\frac{dz_e}{dt} &= T_e (1 - z_e) - \frac{z_e}{\tau_e} \\
T_i &= 2 \left[ 1 + \tanh \frac{V_{Pre}}{4} \right] \\
T_e &= 2 \left[ 1 + \tanh \frac{V_{Post}}{4} \right]
\end{align*}
\tag{5}
\]
where \( \tau_e = 2 \text{ ms} \) and \( \tau_i = 10 \text{ ms} \) which are the time delay for the inhibitory and excitatory neurons.

### D. STOCHASTIC COMPUTING

Stochastic computing paradigm is a digital realization approach which is presented in the 1960s [24]. This architecture purposes an extremely low-power and error-tolerant implementation which utilizes digital logics to perform arithmetic operations such as addition, division, and multiplication and relies on using binary bit-streams [24], [32]. In this approach, a sequence of random bit-streams is used to encode the real values which are interpreted as the probability of being either 1 or 0. For instance, the multiplication operation in stochastic computing representation can be performed using AND gate for unipolar format in the range of [0,1] and XNOR logic gate for bipolar format in the range of [-1,1] which has been illustrated in Fig. 2. This approach has been employed in many applications to lower the computational complexity such as image and signal processing related problems, decoding of low-density parity-check (LDPC), and neural networks and bio-inspired approaches [33], [34], [35].

A stochastic computing system is composed of 3 main phases [25]: 1. Randomizer or stochastic number generator is used to convert binary bitstream to stochastic bitstream, 2. The main stochastic circuit design, and 3. De-randomizer is used to convert stochastic bitstream to binary bitstream which can be designed by using a counter. A general schematic diagram for this methodology is depicted in Fig. 3 [36].

### IV. MODIFIED MODELS

In this section, the tripartite synapse model is modified based on the stochastic computing method to lower the hardware implementation cost and improve the computational efficiency. To achieve to this aim, the stochastic integrator is defined to solve the neural ordinary differential equations which can be designed using a stochastic number generator (SNG), and an n-bit up/down counter [24]. The stochastic integrator requires an n-bit up/down counter which works according to the initial values stored in the counter, and a stochastic number generator which includes a random number generator and a comparator, is required to encode the accumulated values in the counter [37]. To lower the hardware area and power consumption, the shared random number generators can be employed. The two-bit streams of \( a \) and \( b \) for the inputs of A and B can be considered for the up/down counter, respectively. The structure of the up/down counter can be defined as:

\[
I_{i+1} = \begin{cases} 
I_{i+1} & \text{if } a = 1 \text{ and } b = 0 \\
I_i & \text{if } a = b \\
I_{i-1} & \text{if } a = 0 \text{ and } b = 1 
\end{cases}
\]  

(6)

where \( I_{i+1} \) and \( I_i \) represent the values that is saved in the counter at two clock cycles of \( i+1 \) and \( i \), respectively. \( a \) and \( b \) represent the value for both bit-streams of A and B, respectively. Therefore, ordinary differential equations (ODEs) can be approximated using the stochastic integrator based on the Euler method with step size of \( 1/2^n \) [38]:

\[
\frac{dy(t)}{dt} = f(t, y(t))
\]

(7)

\[
\frac{dy(t)}{dt} = \lim_{\Delta t \to 0} \frac{y(t_i + \Delta t) - y(t_i)}{\Delta t} \approx y(t_i + h) - y(t_i) \]

\[
\hat{y}_{i+1} = y_i + hf(t_i, y_i)
\]

(8)

(9)

where \( h \) is the step size, \( \Delta t \) is the time interval and by considering \( t_{i+1} = t_i + h \) based on the Euler’s approach; therefore, \( \hat{y}_{i+1} \) is the numerical simulation for function of \( y(t) \) at \( t_{i+1} \),

### TABLE 1. The parameter values and controlling coefficients of the tripartite synapse models [14].

| Parameter | Value |
|-----------|-------|
| \( c_1 \) | 0.13  |
| \( c_2 \) | 0.9   |
| \( c_3 \) | 0.004 |
| \( c_4 \) | 50    |
| \( \tau_e \) | 8     |
| \( \tau_i \) | 100   |
| \( \tau_{sa} \) | 100  |
| \( \tau_{sb} \) | 100  |
| \( h_{sa} \) | 0.45  |
| \( h_{sb} \) | 0.5   |
| \( d_{sa} \) | 3     |
| \( d_{sb} \) | 3     |
| \( \varepsilon \) | 0.04  |

\( \tau_e = 2 \text{ ms} \) and \( \tau_i = 10 \text{ ms} \) which are the time delay for the inhibitory and excitatory neurons.

### FIGURE 2. Basic stochastic computing elements representation, (a) Unipolar format of stochastic multiplier \( P(x = 1) = P(x = 1)P(y = 1) = xy \), (b) Bipolar format of stochastic multiplier \( 2P(x = 1)-1)(2P(y = 1)-1) = xy \).
i.e., \( y(t+1) \) which is shown in equation (9) [39]. Moreover, the probability of \( a \) and \( b \) at time \( t \) are presented by \( p_a(t) \) and \( p_b(t) \); thus, the numerical simulation of ODE can be estimated as:

\[
\hat{y}_{i+1} = y_i + \frac{1}{2\tau} \left[ p_a \left( \frac{i}{2^n} \right) - p_b \left( \frac{i}{2^n} \right) \right]
\]

(10)

The stochastic integrator has been used for solving differential equation of Ca\(^{2+} \) dynamics and its corresponding differential equation of Ca\(^{2+} \) concentration in ER, has a fast dynamic. These functions can be implemented using stochastic binary projection as follows [40]:

\[
x_b = \begin{cases} 
1, & \text{with probability } p = \sigma(x) \\
0, & \text{with probability } 1 - p 
\end{cases}
\]

(18)

where, \( x_b \) is the binarized variable and \( p \) is the probability illustration of the synaptic transmissions, membrane voltage, and the corresponding variables of the astroglial model. In this expression, \( \sigma(x) \) has been presented by the following expression:

\[
\sigma(x) = \max \left( 0, \min \left( 1, \frac{x + 1}{2} \right) \right)
\]

(19)

\section*{V. HARDWARE DESIGN}

In this section, a hardware implementation is presented for the proposed neuron and astrocyte models which includes the stochastic integrators, Stochastic Number Generators (SNG), and XNOR gates. In this design, the spiking neuron models and the glutamate-induced IP3 production in the glial cells with the astrocytic and synaptic currents are considered as the probabilistic-based models and illustrate the same behavior of the stochastic integrators. The hardware architecture for the proposed neuroglial interaction model with considering the synaptic connection is illustrated as Fig. 4 (A through C). For instance, the spiking neuron model fires when the membrane voltage reaches to its threshold value, similarly the stochastic integrators work based on the stored initial values in the counter.

For the SNG unit, an \( n \) bit-LFSR (Linear Feedback Shift Register) is used to generate an uncorrelated and pseudo random sequence from 0 to \( 2^n - 1 \). In this paper, an LFSR is shared between two SNGs with two comparators to reduce the occupied hardware overhead and create a low-correlated SNG circuit [41]. The shared-LFSR structure may result in low accuracy of the design; therefore, a bit-rotation scheme can assist to reduce the cross-correlation between SNGs. In order to have a higher correlation in the output of the generated stochastic numbers, a 1-bit randomly shifter used at the end of the LFSR to feed each SNG. This structure has been employed for 6-bit and 8-bit LFSRs and illustrated in Fig. 5 [41]. According to this design, a tradeoff between the computational complexity, latency and time has been considered.

A basic block diagram which is used to explain the whole biochemical processes between the elements by considering the influence of coupling coefficients which exist within the neuroglial network has been depicted as Fig. 6.

\section*{VI. NEUROGLIAL NETWORK}

In this section, the influence of astrocyte on the functionality of neuroglial network, has been investigated to realize
A simple network according to the slow and fast activation pathway for various spiking patterns of a tripartite synapse is presented in Fig. 7 (B), that includes four astrocytes that are connected to their neighboring cells via gap junctions to form the astrocytic network. This network is called astrogial syncytium and a pair of coupled neurons is interacting with an astrocyte.

Thus, gap junction flow for IP3 \((J_{IP3})\) and calcium \((J_{Ca^{2+}})\) between two neighboring cells through GJs, are governed by the following equations respectively [31], [42]:

\[
J_{IP3} = G_{IP3} ([IP3]_i - [IP3]_k)
\]

\[
J_{Ca^{2+}} = G_c ([c]_i - [c]_k)
\]

where the IP3 gradient between two neighboring astrocytes is presented by \(([IP3]_i - [IP3]_k)\), the flow of calcium in the network of cells can be shown by \(([c]_i - [c]_k)\). \(G_{IP3}\) and \(G_c\) are the maximal flux diffusion (coupling strength) between two astrocytes through gap junctions for IP3 and Ca2+, respectively. According to these equations, \(i\) presents glial cell in the astrogial network and is situated to its adjacent cells that is specified by \(k\). The astrocytic current which is shown by \(I_{astro}\) can be added to each neuron in tripartite synapse model also is defined as follow [1], [2]:

\[
I_{astro} = \lambda_i [c], \quad i = 1, 2
\]

where \(\lambda_i\) is the coupling parameter for depolarizing currents from astrocyte to pyramidal neuron and interneuron. Calcium
signalling can have an influence on the synaptic transmission from presynaptic to postsynaptic neuron through coefficient of $\lambda_1$ and $\lambda_2$.

IP3 is known as the second messenger. IP3 and calcium waves which can propagate through GJs from one astrocyte to the adjacent astrocyte are involved in the release of ATP and Glutamate within extracellular space. The gap junction coupling deficiency between glial cells in the network can lead to neuronal hyperexcitability which induces abnormal and periodic seizures. The coupling strength depends on the number of the gap junction coupling in astroglia network [43].

To evaluate the physiological and pathological situation of the glial cells, a measuring concept for the synchronization of neurons has been used by the following Kuramoto order parameter $\Phi$ [44], [45]:

$$\Phi(t) = \frac{1}{N_{osc}} \sum_{j=1}^{N} e^{i\psi_j(t)}$$

(23)

where $\Phi(t)$ sets the mean phase, $\Phi(t)$ is used to measure the synchronization index, $N_{osc}$ measures the number of cells in the simulation, and $\psi_j(t)$ is the phase of neuron $j$ at time $t$ that has been defined as [1]:

$$\psi_j(t) = 2\pi \frac{t - t_n}{t_n+1 - t_n}$$

(24)

where $t \in [t_n, t_{n+1})$ and firing pattern can be included between this timing interval, $t_n$ sets the onset time of the kth burst of jth neuron and is considered as $0 \leq \Phi(t) \leq 1$ for all the time. When $\Phi = 0$, oscillations are desynchronized, and for $\Phi = 1$ phase synchronization will occur. A similar method for desynchronization of two coupled oscillators in [2] has also been employed for the simulation process of the modified and original models which is demonstrated in Fig. 8 (A through E). In this paper, gap junction coupling strength between astrocytes and applied current are also considered as the controlling parameters during the neuronal desynchrony and synchrony analysis. By increasing applied current, coupled neurons start interacting and becoming synchronized due to an elevation of the excitatory coupling coefficient on postsynaptic neuron. The other controlling parameters which are used for the simulation process, are listed as follows: GJ coupling coefficient ($G = 0$ and 1.4) and the rest of the parameters $\lambda_1 = 0.06$, $\lambda_2 = 0.11$, and $g_e = 0.02$. According to the results shown in Fig. 8, by changing the strength of GJ coupling, the mode of coupled neurons will fluctuate from in-phase to anti-phase synchronization mode and the impact of astrocytic synctium can be examined on synchronized neurons. With strengthened coupling coefficient, anti-phase spiking rates can be observed.

The elevation of calcium ions in astrocyte and diffusion of Ca2+ waves and IP3 molecules via gap junctions in glial synctium can assist in regulating the synaptic transmission between neurons over long distances. Therefore, the strength of astrocytic coupling can improve the spread of calcium signalling among astrocytic synctium and respond to the neuronal activities. According to tripartite synapse model, gliotransmitter release such as ATP and Glutamate using coupling coefficients (shown by $\gamma$, $\eta$) can control the excessive excitatory current on postsynaptic neurons. In order to investigate the role of astrocytic synctium in coordinating the synaptic plasticity, different values of coupling coefficients for tripartite synapse model ($\gamma$, $\eta$) and also maximal flux diffusion have been considered for analyzing the behavior of postsynaptic activity. The absence of astrocyte between presynaptic and postsynaptic neurons can be depicted in Fig. 9. An applied constant current will generate the action potential in presynaptic neurons which results a rise in excitatory synaptic conductance and activation of postsynaptic current on interneurons. In this case, the spiking rates of postsynaptic neurons uncontrollably will enhance, where the postsynaptic current cannot be regulated properly, and this can cause the pathophysiological conditions for the brain. The gap junction coupling strength between glial cells, astrocytic current due to glutamate release which causes depolarization of postsynaptic neuron and, the excitatory current on postsynaptic neuron can be modulated properly and this can prevent from unwanted neuronal hypersynchrony. The influence of the astrocytes between coupled neurons has been illustrated as Fig. 10. According to the results which have been shown in this figure, by increasing $\gamma$ and $\eta$ (Glutamate and ATP effects) and decreasing the GJ coefficient the rate of spiking activities among neurons also can alter and switch to epileptic conditions [46]. Therefore, the presence of astroglia network can prevent from excess extracellular potassium concentration and glutamate accumulation. This abnormal amount of potassium and glutamate can be eliminated by strong gap junctions coupling between astrocytes [47]. In this work,
astrocytes are connected in a squared shape. Gap junction in astrocytic syncytium can redistribute the potassium (K⁺) ion and regulate neuronal activity. Uptake of glutamate and K⁺ can prevent from neurotoxicity. The balance of extracellular Potassium-glial concentration equation can be written as the following equation:

$$W \frac{d[K]}{dt} = \frac{1}{F} \sum_{i=1}^{N} I_{i,K} + G ([K]_O - [K])$$  (25)

where $W$ is the measure of average distance between the glial cells, $F$ is the Faraday’s constant, $I_{i,K}$ shows the electrical current of potassium, and finally $G([K]_O - [K])$ is the diffusion of the potassium. $G$ can be used as the controlling parameters for describing the neuronal synchrony between the coupled neuron [48]. The concentration of potassium in bath is shown by $[K]_O$.

The model of astrocyte can be extended as below for the glial network with considering coupled gap junctions:

$$\tau \frac{dc}{dt} = -c + r + \beta \cdot S_m + \sum_{i=1}^{n} \alpha_i w_i + \sum_{j=1}^{m} G_C [c_j] - [c]$$  (26)

where $i$ is the number of neurons and $j$ is the number of astrocytic cells which are existing in the neuroglial network.

Different behavior of neuronal spiking activities using frequency spectrum (as Fast Fourier Transform and their corresponding histograms) are observed in [50]. On the other hand, FFT approach is used on the spiking activity of the coupled neurons according to increasing or decreasing coupling coefficients within the astroglia network. The results of this analysis for the original and the modified model have been demonstrated in Fig. 11. By selecting the appropriate coupling coefficients, the impact of astroglia network (as a regulator) in desynchronizing the hypersynchronous neurons has been evaluated. Synaptic coupling strength between neurons can activate potassium (K⁺) current after neuronal depolarization; therefore, this fast activation pathway can be controlled by parameter $\alpha$ (potassium activation pathway). In [49], the slow activation pathway in tripartite synapse and its impact on long term potentiation (LTP) of the postsynaptic spiking activity has been investigated to realize the closed loop behavior of neuroglial interaction model. In this paper, the impact of short-term potentiation (STP) mechanism is also considered on postsynaptic neurons which can be evaluated for adaptation (potassium) currents. By controlling parameter of $\alpha$, extracellular K⁺ ions and STP can be regulated. The fast activation pathway causes glial cells depolarization due to rise of extracellular K⁺, and slow activation pathway activates astrocytes by IP3 production.
through synaptic strength. Fig. 12 illustrates that the firing rates unexpectedly can increase due to the rise of extracellular K+ ions when the gap junction rate between astrocytes is less than 0.4. Therefore, by controlling coupling parameters of the neuroglial interaction model, the frequency of firing rates can be managed.

In order to investigate the accuracy between the proposed model and the original biological neuroglial interaction models, Root Mean Square Error (RMSE) is computed and defined as follow:

\[
RMSE (V_{org}, V_{modified}) = \sqrt{\frac{\sum_{i=1}^{n} (V_{org} - V_{modified})^2}{n}}
\]  

(A) membrane voltage for two coupled neurons by considering the role of the original astrocyte model (B) spike trains (membrane voltage) for two coupled neurons by considering the role of the modified astrocyte model. (C) Synchronization index for the original astrocyte model (black) and for the proposed astrocyte model (red). (D) (the output of original (black) and the modified (red) astrocyte model). (E) the effect of gap junction coupling between glial cells.

FIGURE 8. Effect of varying gap junction coupling strength between glial cells and the influence of astrocyte between two neurons during synchrony and desynchrony conditions for the original and proposed models. (A) membrane voltage for two coupled neurons by considering the role of the original astrocyte model (B) spike trains (membrane voltage) for two coupled neurons by considering the role of the modified astrocyte model. (C) Synchronization index for the original astrocyte model (black) and for the proposed astrocyte model (red). (D) (the output of original (black) and the modified (red) astrocyte model). (E) the effect of gap junction coupling between glial cells.

FIGURE 9. Membrane potential for the presynaptic and postsynaptic neurons without considering the influence of astrocytic network and its corresponding coupling coefficients. By increasing the excitation current on postsynaptic neuron, the frequency of the spiking activity has been enhanced. This can lead to the hyperexcitability of the neurons. (A) Spiking activity of presynaptic and postsynaptic neuron for the original model. (B) spiking activity of presynaptic and postsynaptic neurons for the modified model.

FIGURE 10. Influence of astroglial network on the spiking activity of postsynaptic neuron for the original and the modified models. In this case, by considering the neuron-astrocytic coupling coefficients, \(\gamma\), \(\eta\), and gap junction coupling between glial cells, the spiking activity of the postsynaptic neurons can be regulated and controlled. (A) Spiking activity (membrane potential) for the original model. (B) Spiking activity (membrane potential) for the modified model.
FIGURE 11. The effects of astrocyte in desynchronization of neurons firing. Raster plot representing the spiking activity neurons astrocytes (each neuron is randomly connected to other neurons through synapses and astrocytes), histograms for each state, and Fast Fourier Transform (FFT) for corresponding histograms. (A) spiking activity of neurons without considering the influence of astroglial syncytium for the original model. (B) spiking activity of neurons with considering the influence of astroglial syncytium in desynchronizing neurons for the original model. (C) spiking activity of neurons without considering the influence of astroglial syncytium for the modified model. (D) spiking activity of neurons with considering the influence of astroglial syncytium in desynchronizing neurons for the modified model.

TABLE 2. RMSE calculations for the neural spiking patterns using different time steps.

| \( \text{I}_{\text{applied}} \) (\( \mu \text{A} \)) | RMSE | \text{NRMSE}(|\%|) \( dt = 1/2^6 \) | RMSE | \text{NRMSE}(|\%|) \( dt = 1/2^8 \) |
|---|---|---|---|---|
| 1.50 | 1.316 | 2.465 | 0.674 | 1.238 |
| 2.50 | 2.537 | 3.013 | 0.898 | 1.411 |
| 3.50 | 3.791 | 4.986 | 1.806 | 2.032 |
| 4.50 | 5.106 | 6.008 | 2.930 | 3.001 |

FIGURE 12. Demonstration of firing rates based on various coupling coefficients. Increment of parameters \( \gamma \) and \( \eta \), can result in increasing spiking rates and by adjusting these two parameters spiking rates can be controlled. Strong gap junction coupling between astrocytes can regulate spiking rates; however, reduced gap junction strength cannot maintain spike rates.

where \( V_{\text{org}} \) and \( V_{\text{modified}} \) represent the original values for the corresponding function and the modified values based on the SC-based values, respectively. The data length for the error measurement is shown by \( n \). The Normalized Root Mean Square Error (NRMSE) can be obtained by the following equation:

\[
\text{NRMSE} = \frac{\text{RMSE}}{V_{\text{max}} - V_{\text{min}}} \tag{28}
\]

where \( V_{\text{max}} \) and \( V_{\text{min}} \) are the maximum and minimum values of membrane voltage.

The \( \text{RMSE} \) and \( \text{NRMSE} \) values according to the different excitation currents under various time steps are calculated and reported as Table 2.

VII. IMPLEMENTATION RESULTS

This part presents the results of the hardware design for a neuroglial interaction model. As a proof of concept, the
proposed circuit and the original models have been implemented on a XILINX Virtex-7 platform using ISE tools in order to evaluate the hardware performance. The output of the coupled neurons by considering the effect of glial cell on neuronal regulation is depicted in Fig. 13. Fig. 14. demonstrates the digital oscilloscope photographs of the proposed model for the membrane potential, calcium signalling, and IP3 production on the FPGA board. Low level device utilization details of the implemented original and the proposed models for the slow pathway activation mode have been summarized in Table. 3. The power consumption analysis of the modified model is achieved by 138 mW in comparison to the original model which is realized by 316 mW.

The comparison results of the hardware realization in terms of the number of resources and maximum speed have been performed between the modified models and the previous proposed tripartite synapse models in [17], [27], [43], [50], [51], and [52]. These results are reported in Table. 4. In our proposed design, DSP block which is an expensive resource has not been employed within the implementation in comparison to the design which is proposed in [43]; therefore, the proposed architecture can provide less hardware complexity rather to the original models and the previously published research works. In [27], a hardware implementation using stochastic approach on the tripartite synapse is performed for 16-bit and 12-bit LFSRs; however, the detail of hardware resources for the astrocyte model has not been included in their work. The hardware resources of the neuron model have not been provided in [17] and the number of resources which is reported for the astrocyte model are greater than the hardware resources that are presented in this research article. Furthermore, the frequency of the design in [51] is reported 139MHz. A neuromorphic

| TABLE 3. Low level device utilization and the used percentage of the elements of the modified astrocyte and neuron models for the fast activation pathway. |
|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|
| Logic Utilization | Modified Neuron Model | Modified Astrocyte Model | Original Neuron Model | Original Astrocyte Model |
| Number of Slices | 96              | 185             | 112             | 217             |
| Number of LUTs   | 122             | 251             | 238             | 341             |
| Number of DSPs   | 0               | 0               | 1               | 7               |
| Number of GCLKs  | 2               | 2               | 2               | 2               |

*(N/A) presents the results of hardware resources for Neuron and Astrocyte, respectively.

| TABLE 4. Hardware resources comparison between the proposed model in this work and the previously published works. |
|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|
| Resources       | Modified Models | Model in [17]   | Model in [27]   | Model in [43]   | Model in [50]   | Model in [51]   | Model in [52]   |
| Registers       | (96/185)        | (–/3647)        | (214/–)         | (10383/11666)   | (385/145)       | (–/1065)        | (35/262)        |
| LUTs            | (122/251)       | (5813/–)        | (140/–)         | (9865/11394)    | (441/273)       | (–/1928)        | (180/3052)      |
| DSPs            | (0/0)           | (–/–)           | (0/0)           | (45/42)         | (0/0)           | (–/–)           | (–/–)           |
| Frequency (MHz) | 192.53          | 251.196         | –               | –               | –               | 139MHz          | –               |

*TABLE 5. Hardware resources comparison between the proposed astrocyte model in this work and the recently published works for astrocyte models.*

| Resources | This work | Design in [53] | Design in [54] |
|-----------|-----------|----------------|----------------|
| Registers | 185       | 670            | 675            |
| LUTs      | 251       | 1345           | 1290           |
| DSPs      | 0         | 4              | 8              |
| BRAM      | 0         | 4              | –              |
digital design for neuroglial interaction model by employing the linear approximation method is realized in [52]. Based on their research work, 5 multipliers are reported in the high-level utilization table.

A hardware cost comparison has been performed between the proposed astrocyte model in this work and two recently published articles according to different calcium-based astrocytic models in [53] and [54]. A digital design for an astrocyte model is implemented on FPGA platform which uses four DSP blocks and four block RAMs [53]. In [54], a set of piecewise linear approximation is presented for an astrocytic-based calcium signalling with the maximum speed of 81 MHz. The result of this comparison is summarized in Table 5.

VIII. CONCLUSION

In this paper, a hardware implementation according to stochastic computing method for the neuron-glial interaction (tripartite synaptic model) based on the spiking neuron model and the Postnov astrocyte model [14], [15] has been presented. The impact of glial syncytium and its bidirectional communication between neurons and astrocytes for desynchronizing the abnormal and paroxysmal synchronized neurons within epileptic seizures in the neural network by considering appropriate coupling parameters is also investigated. The comparison results for the hardware and software implementation between the original biological models and the proposed models illustrate that the proposed models have lower hardware costs. The hardware implementation results of the proposed model are compared with previously published studies. The main aim of this study is to investigate the influence of astroglial network on different level of neural hyperexcitability during epileptic seizures. This work illustrates that the proposed design can be a good candidate for large scale bio-inspired neuroglial network hardware implementations on FPGA platforms.

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