An Integrated Astrocyte-Adaptive Exponential (AAdEx) Neuron And Circuit Implementation

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***Abstract*—Reconstruction and implementation of biological models of the brain cells can help to understand brain information processing algorithms through system-level modeling of cellular networks, study the mechanisms underlying neurological and psychiatric disease, simulate drug treatment, designing brain computer interfaces and many useful applications. The simulations of brain require qualitative models describing its components and how they interact with each other. Most of previous effort only consider neurons as active component in processing of information in the brain, but it has proven that the glial cells unlike former belief, are involved in the many brain function. This paper couples an accurate, simple and versatile AdEx neuron model with a concise astrocyte model and investigate their mutual effect pathways. The simulation result of integrated models, reveals the significant effect of the astrocyte on the post-synaptic neuron activation or inactivation. In the last section a circuit for implementation of the silicon astrocyte is presented that can be used alongside with available AdEx neuron circuits for the realization of the astrocyte integrated AdEX neuron (AAdEx).**

Keywords : Adaptive Exponential Integrate and Fire, AdEx , Astrocyte , Neural Network, Circuit Implementation, Neuromorphic.

I. Introduction

Until recently, it was believed that the neuroglia did not play active role in the processing of informations in the brain and only provide physical and chemical support for neurons. A classical synaptic model consist only of two parts, a pre-synaptic and post-synaptic terminal. During the last decade the astrosyte neuron interactions has been widely explored. New data shows glial participation in synaptic transmission, long-term potentiating, synaptic plasticity, and the development of neuronal pathologies [1]-[5]. So for more precise simulation of neural network, presence and activity of glial cells should be taken into account. Real mathematical model must cover all relevant ionic currents, the processes of production and propagation of neurotransmitters and etc. Such model inevitably will be high-dimensional with large number of control parameters and high complexity. Therefore, simplified models can be used for studies in the field of neural information coding, memory and network dynamics.

Following postnov et al. [6] a qualitative model, describing the two connected neurons via synapse and the glial cell has been used. Two mechanisms have been taken into account for neural glial interactions in model (see figure 1,I): fast mechanism and slow mechanism. When an action potential arrives at the pre-synaptic terminal, it releases glutamate into the synaptic cleft. The Released glutamate then, binds to receptors on the post-synaptic terminal and triggers excitatory post-synaptic potential. Glutamate may also partially binds to the glutamate receptors (factor β in figure) of the astrocytes and in consequence, increases IP3 concentration in intracellular space of it. IP3 in turn, binds to the IP3 receptor in the endoplasmic reticulum (ER) and result in Ca^{2+} releases from the ER into the Cytosol. Liberating Ca^{2+} from internal stores is much slower than the time scale of the action potential [7]. Astrocytes with elevated Ca^{2+} concentrations releases Gliotransmitter into the extracellular space (factor δ in figure) that can even propagate through extracellular space into adjacent astrocyte cells [8].

Another pathway for astrosyte neuron interaction is instantly depolarization of astrosyte by K^{+} ions that releases because of action potentials in the post-synaptic neuron (factor α in figure) [6]. Each of pre and post synapti neyrons are described by the FitzHugh-Nagumo model [9]. post-synaptic

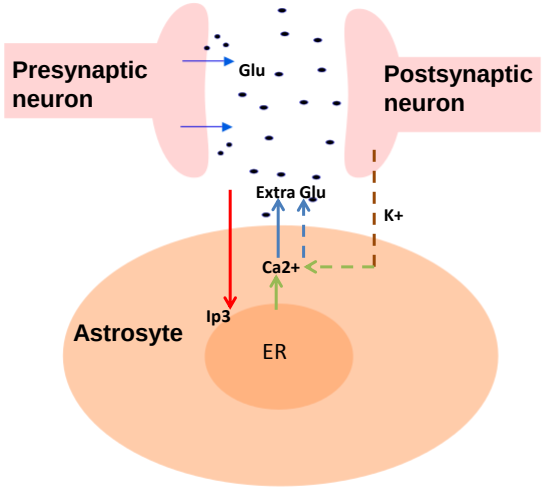

Fig. 1. Pre-synaptic neuron activity releases neurotransmitter (Glutamate) into the synaptic cleft that binds to receptors on the post-synaptic terminal and stimulate excitatory post-synaptic potential. Furthermore, glutamate partially binds to the glutamate receptors of the astrocytes and trigger IP3 release inside cells. IP3 binds to the IP3 receptor in the (ER) and in consequence the Ca $^{2+}$ releases from the ER into the Cytosol. Raised Ca^{2+} concentrations release Gliotransmitter into the extracellular space.

TABLE I. COMPARISON OF ADEX AND FITZHUGH-NAGUMU NEURON MODEL

Models	biophysically meaningful	tonic spiking	phasic spiking	tonic bursting	phasic bursting	mixed mode	spike frequency adaptation	class 1 exitable	class 2 exitable	spike latency	subthreshold oscillations	resonator	integrator	rebound spike	rebound burst	threshold variability variability	bistability	DAP	accommodation	inhibition-induced spiking	inhibition-induced bursting	chaos
FHN	-	+	+	-		-	-	+	-	+	+	+	-	+	-	+	+	-	+	+	-	-
AdEx	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+

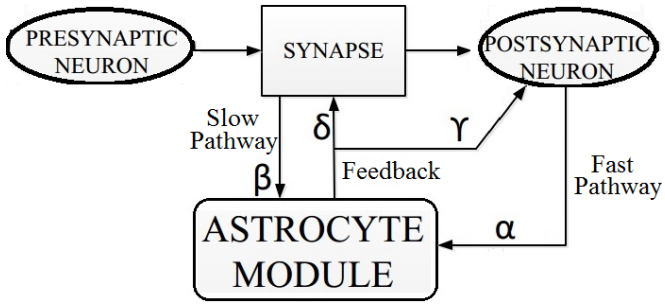

Fig. 2. Two pathway for neuron-glial interaction : 1) slow activation pathway through β and δ 2) fast activation pathway through α and γ.

neuron has an additional term for the feedback from the glial cell A (factor δ in figure). Synapse S is described with first-order nonlinear differential equations as in the [6].

The FHN model is a widely used approximation of the Hodgkin-Huxley model that has significant limitations. It has been noted that the FHN model can not exhibit certain firing pattern like bursting and self-sustained chaotic dynamics and is also difficult to adapt to neurons with specific properties [10]. Table I shows the ability of both models to mimic biological neuron behaviors. As it obvious from table AdEx support all of the biological neuron firing patterns. Furthermore, parameters in FHN model are not biologically meaningful. For this limitations authors encouraged to change the FitzHugh-Nagumo with adaptive exponential integrate and fire neuron (AdEx) model [11]. This model is introduced by Brette and Gerstner in 2005, and is hybrid two-dimensional model with exponential Spike initiation which fits to the experimental results and its parameters could be directly related to biological values [12]. . Beside that, AdEx can generate different types of spiking patterns in contrast to FHN model. The model is also computationally fast Compared to biologically detailed models like Hodgkin-Huxley [13] .

The rest of the paper organized as follows: at section 2 the model for neuron and astrocyte has been reviewed. In the section 3 AdEx neuron model and astrocyte coupled and the mutual effects investigated. Next section present an analog circuit for implementation of astrocyte and the paper is concluded in the last section.

II. NEURONS AND ASTROCYTE MODELS

This section briefly reviews the utilized AdEx model to replace the FHN model used in the [6] instead of (pre-post) synaptic neurons, alongside the synapse and astrocyte model, and explain their parameters.

A. Pre-synaptic Neuron

Pre-synaptic neuron is described by AdEx model [14]. The AdEx neuron model is a 2-D dynamical system in which its states is determined by two variables, namely membrane potential (v_1) and adaptive current (w_1) and is described by:

$$\begin{aligned} C\frac{dV_1}{dt} &= -g_L\Delta T exp(\frac{V_1 - V_T}{\Delta T}) + I_{app} - w_1 \\ \tau_w\frac{dw_1}{dt} &= \alpha(V_1 - E_l) - w_1 \end{aligned} \tag{1}$$

with a auxiliary reset equation as:

$$if \quad V_1 > 0 \quad then \quad \begin{cases} V_1 \rightarrow v_r \\ w_1 \rightarrow w_r = w_1 + b. \end{cases} \tag{2}$$

Where the first equation determines the membrane potential V1, and the w1 denotes the adaptation current variable. Also, I_{app} represent the applied current to the neuron.

- C : Total membrane capacitance (pF);
- g_l : Total leak conductance (nS);
- E_l : effective rest potential (mV);
- ΔT : threshold slop factor (mV);
- V_T : effective threshold potential (mV);
- V_r : resetting potential (mV);
- τ_w : adaptation time constant (ms);
- a : subthreshold adaptation (nS);
- b : spike-triggered adaptation (pA);
- I_{app} : applied current (pA)

By changing this parameters the model can produce different firing pattern like bursting, fast spiking, regular spiking, etc [14].

B. Synapse Model

Following postnov et al. [6] the synapse considered only as threshold activation (sigmoid function) and a delay :

$$\tau_s\frac{ds}{dt} = (1 + tanh(s_s(V1 - h_s)))(1 - z) - \frac{z}{d_s} \tag{3}$$

$$I_{syn} = (k_s - k_f\delta G_m)(z - z_0) \tag{4}$$

where, z is a synaptic activation variable and τ_s describes the time delay. h_s is threshold for activation of synapse and s_s is a factor that helps to quicken the transition of hyperbolic tangent. Likewise, d_s is responsible for activation and relaxation of synapse. when v1<h_s, the absolute value of the term inside hyperbolic tangent with multiplication of s_s become large enough, and the hyperbolic tangent can be considered almost as $\simeq$-1, and therefore z$\simeq$0 with a rate proportional to $1/\tau_s$. Likewise, when v1>h_s, then z becomes $\simeq$1. Activation of z produce I_{syn} for post-synaptic neuron. k_s is the conductivity

of synapse and δG_m is astrosyte response through release of gliotransmitter in synaptic cleft. When the pre-synaptic neuron is silent z(t) must be equal to z_0.

C. Postsynaptic Neuron

Same as pre-synaptic neuron the AdEx model has been used for post-synaptic neuron:

$$\begin{aligned} C\frac{dV_2}{dt} &= -g_L\Delta T exp(\frac{V_2 - V_T}{\Delta T}) + I_{app} + I_{glion} - w_2 \\ \tau_w\frac{dw_2}{dt} &= \alpha(V_2 - E_l) - w_2 \\ I_{glion} &= k_f\gamma G_m \end{aligned} \tag{5}$$

and the reset equation

$$if \quad V_1 > 0 \quad then \quad \begin{cases} V_2 \rightarrow v_r \\ w_2 \rightarrow w_r = w_2 + b. \end{cases} \tag{6}$$

The applied current to post-synaptic neuron is sum of synapse current I_{syn} and the current induced by gliotransmitter because of astrosyte activity I_{glion}. So, the two parameters, δ and γ, control the amount of astrosyte influence on synapse and post-synaptic neuron.

D. Astrosyte Model

1) Ca^{2+} Dynamics: Intracellular astrocyte Ca^{2+} dynamics has been modeled using dimensionless equations with additional term describing the extra Ca^{2+} result from the K^+ and Ip_3 as in the [5].

$$\begin{aligned} \tau_c\frac{dc_a}{dt} &= (r + \alpha(w_2 - w_2^*) + \beta S_m) - c_a - c_4 f(c_a, c_e) \\ \epsilon_c\tau_c\frac{dc_e}{dt} &= f(c_a, c_e) \end{aligned} \tag{7}$$

Where c_a and c_e is Ca^{2+} concentration within the cytoplasm and ER (Endoplasmic reticulum) of astrosyte respectively. ϵ_c and τ_c are responsible for time characteristic of Spontaneous Ca^{2+} Oscillations. r controls the intial state for intracellular Ca^{2+} Oscillations. α and β represent influx from ER to cytoplasm due to extracellular stimulation of glutamate and depolarization by K^+. The exchange of Ca^{2+} between ER and cytoplasm has been described by a nonlinear function [6]:

$$f(c_a, c_e) = c_1\frac{c_a^2}{1 + c_a^2} - \frac{c_e^2}{1 + c_e^2}\frac{c_a^4}{1 + C_a^4} - c_3 c_e \tag{8}$$

2) Gliotransmitter Production: As mentioned before, Liberating Ca^{2+} from ER is much slower than the time scale of the action potentials. So a considerable delay has chosen when the secondary mediator S_m and gliotransmitter (G_m) production has been modeled by a threshold and delay:

$$\tau_{s_m}\frac{dS_m}{dt} = (1 + tanh(s_{s_m}(z - h_{s_m})))(1 - S_m) - \frac{S_m}{d_{s_m}} \tag{9}$$

$$\tau_{G_m}\frac{dG_m}{dt} = (1 + tanh(s_{G_m}(z - h_{G_m})))(1 - G_m) - \frac{G_m}{d_{G_m}} \tag{10}$$

here, h_{s_m} and h_{G_m} are threshold for activation and relaxation of S_m and G_m with rate proportional to τ_{s_m} and τ_{G_m} respectively. So $\tau_{s_m} >> \tau_{G_m} >> \tau_s$.

TABLE II. PARAMETERS USED FOR SIMULATION OF PRE AND POST SYNAPTIC ADEX NEURONS[14].(NOTE THAT THE MODEL IS DIMENSIONLESS.)

C (pF)	200	$g_l(nS)$	10	$E_l(mV)$	-70
$V_T(mV)$	-50	$\Delta T(mV)$	2	$\tau_w(ms)$	30
$a(nS)$	2	$b(pA)$	0	I_{app} (pA)	500
$V_r(ms)$	-58				

III. COUPLING ASTROCYTE AND ADEX NEURON

AdEx is a biological model in comparison to Fitz-Hugh Nagumu model. The membrane action potential generated by AdEx changes between about -70 mV up to 0v while the FHN is between 1 V and -1 V. Therefore, the value of of synapse activation threshold selected as h_s= -55 instead of 0 in the synapse with FHN model so that when I_{app} stimulates neuron and it goes a for a spike, that spike be able to activates the synapse as well. The conductivity factor (k_s), also changed in order to regulate the synapse current in the previous model with post-synaptic AdEx neuron. While astrocyte influence pathways turned off (δ=0 and γ=0) figure 3 shows both neurons and synapse activity, by applying a 500 pA current to the pre-synaptic neuron . The synapse weight selected as this only synapse be able to drive post-synaptic neuron to spiking state. Parameters used for simulation of tonic spiking AdEx neurons are shown in table II.

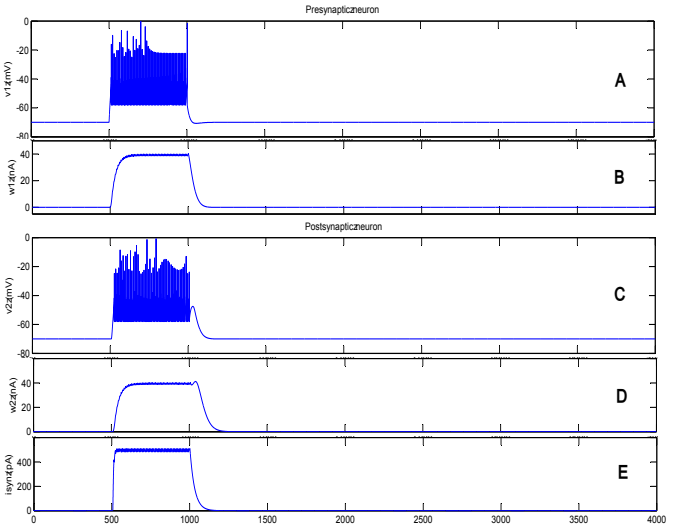

Fig. 3. With all of astrocyte influence pathways turned off (δ=0 and γ=0) the pre-synaptic neuron stimulated with a 500 pA current. v is neuron membrane potential and w is recovery variable. A,B,C,D) pre-synaptic and post-synaptic AdEx neurons. . E) I_{syn} with the impact of pre-synaptic neuron activity.

A. Synapse And Neurons Effects On Astrocyte

First, by assigning the $\beta = 0$, glutamate influence on astrocyte was disabled and then the fast activation pathway strength (through K^+) increased from $\alpha = 0$ to relatively large value. It starts with sub-threshold dumped oscillation at α=0.00056 (Fig. 4 (A)). α=0.00059 arises a single spike of calcium at the beginning of spike train (Fig. 4 (B)). Subsequently, the fast activation pathway blocked by change $\alpha = 0$ and then calcium production by secondary messenger

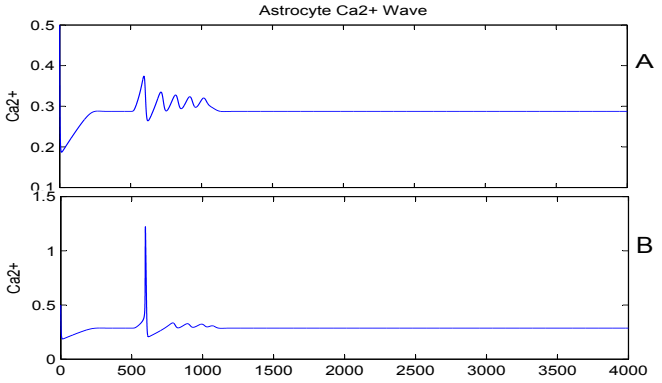

Fig. 4. Calcium concentration waves in cytoplasm of Astrocyte through slow activation pathway: A) dumped oscillation at α=0.00056 and B) single spike at α=0.00059

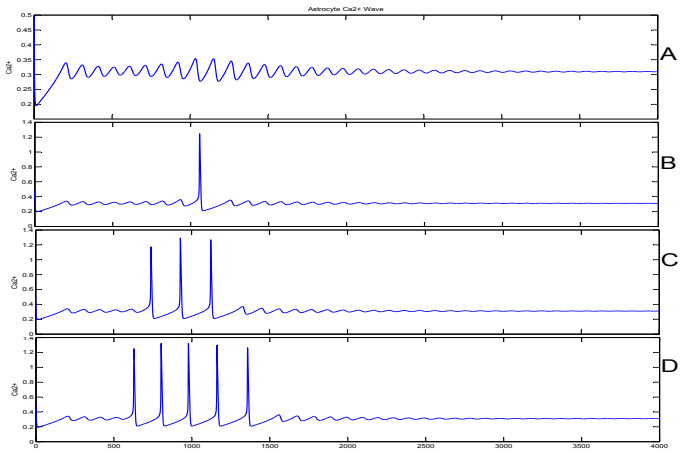

Fig. 5. Calcium concentration waves in cytoplasm of astrocyte by slow activation pathway: A) dumped oscillation at β=0.002 B) single spike at end of neuron activity at β=0.00059 C,D) calcium spikes get closer to beginning of neuron activity($\beta = .004$,$\beta = .005$)

via glutamate release in the synaptic cleft was activated with increasing β. As mentioned before $\tau_{G_m} >> \tau_s$, so the process of releasing gliotransmitter is much slower than duration of action potential. Therefore, both factors β and duration of neuron activation can change the concentration and frequency of gliotransmitter production of astrocyte [15]. At $\beta = 0.0022$ there is sub-threshold calcium oscillation in astrocyte (see Fig. 5 (A)). By increasing beta (at $\beta = 0.0025$) a single spike appears with decline of pre-synaptic stimulation current (Fig. 5 (B)). Ca^2+ oscillation in the case of further increase of β has been shown in Fig. 5 (C,D). The number of spikes increases with the value of β.

TABLE III. PARAMETERS USED FOR COUPLING ASTROCYTE AND SYNAPSE WITH ADEX NEURON.(MODEL IS DIMENSIONLESS.)

c_1	0.13	c_2	0.9	c_3	0.004
c_4	$2\backslash\epsilon_c$	ϵ_c	0.04	r	.31
τ_c	8	τ_{s_m}	100	τ_{G_m}	50
s_s	1	s_{s_m}	100	s_{G_m}	100
h_s	-55	h_{s_m}	0.45	h_{G_m}	0.5
k_s	600	z_0	0	d_{G_m}	3
k_f	1000	$w^*{}_2$	40	h_{G_m}	0.5

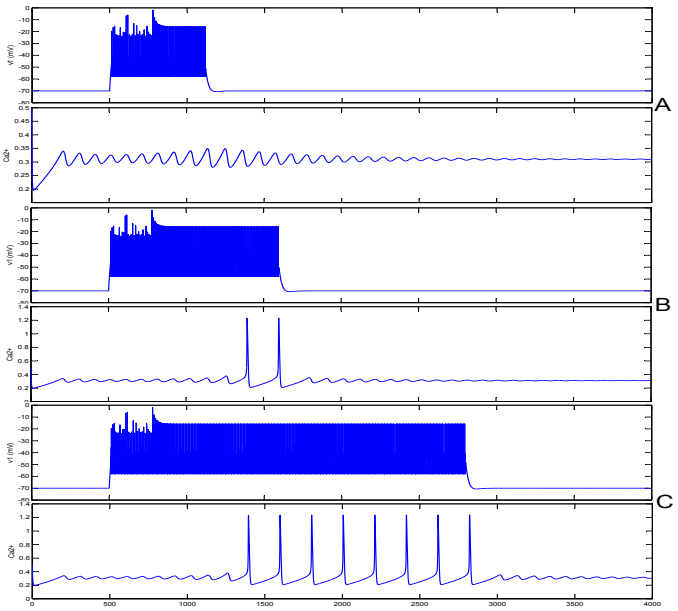

Fig. 6. β has been fixed in β=0.002 A: sub-threshold oscillation at ΔT=700 B,C: Ca^{2+} spikes at ΔT=1200 and ΔT=2400

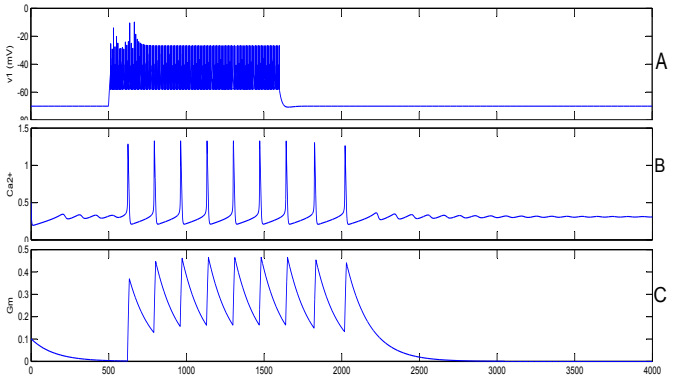

Fig. 7. A) pre-synaptic neuron activity (β=.008 , α=0 and δ=12000) B) calcium wave in astrocyte C) gliotransmitter production by astrocyte

As beta increases astrosyte activates faster and calcium spikes get closer to the starting time of stimulation current in pre-synaptic neuron. In the figure 6, beta has been fixed in 0.002 and then the duration of neuron stimulation and firing increases. At ΔT=700, sub-threshold oscillation can be observed. There is Ca^{2+} spikes at ΔT=1200 and ΔT=2400. But then they stop as soon as neuron stops firing due to small value of β.

B. Closing Astrocyte Feedback Pathway

The control parameters has been set at fixed point (α=0 , δ=0 and β=0.008) so that the fast activation pathway and the astrocyte-induced inactivation of synapse become blocked. Fig. 7 shows the gliotransmitter production by slow activation pathway. By increasing of gamma, (at γ=1) the burst was observed in post-synaptic neuron while the pre-synaptic neuron is already in resting state (Fig. 8 A). By further increasing of gamma the burst duration is decreased and almost continued firing appears at post-synaptic neuron after the rest of the pre-synaptic neuron (Fig. 8 B). In the (Fig. 8 C) the astrocyte-

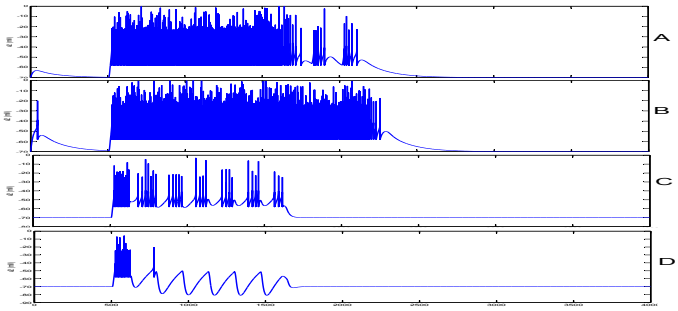

Fig. 8. Closing astrocyte feedback pathways : A,B) astrocyte-induced inactivation of synapse blocked (δ=0). Burst observed in post-synaptic neuron at γ=1 and duration of bursts decreased with further increase of gamma (γ=3). D,C) astrocyte-induced activation of synapse pathway blocked (γ=0). Suppression of post-synaptic neuron activity at (δ=1).

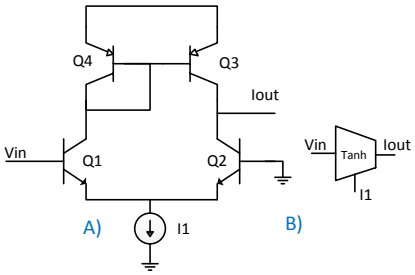

Fig. 9. Hyperbolic Tangent cell A) schematic B) library symbol

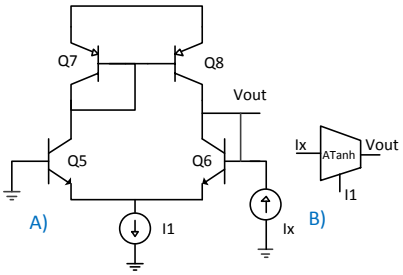

Fig. 10. Hyperbolic Arctangent cell A) schematic B) library symbol

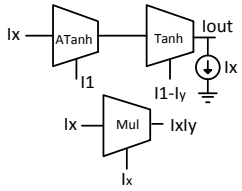

Fig. 11. Analog current multiplier circuit

induced inactivation of synapse pathway closed ($\delta \neq 0$) and the activation pathway blocked (γ=0). (Fig. 8 D) shows that the suppression of synaptic strength due to astrocyte feedback can completely block a stimulus transmission at γ=3 .

IV. Implementation of Models

This section presents a analog circuit for implementation of astrocyte model. There are several analog circuit implementation of AdEx neuron (see [16], [17]) that can be used alongside with the proposed astrocyte circuit.

There are two challenges for implementation of the astrocyte analog circuit. One is the hyperbolic tangent and the other is the analog multiplier. The threshold and delay functions in Eq. 9, 10 have hyperbolic tangent term. Hyperbolic tangents and tangent inverses can be implemented by differential amplifiers (Fig.9 and Fig.10) [18]. For schematic in the figure 9 we have

$$I_{out} = I_1 tanh(\frac{V_{in}}{2V_T}) \tag{11}$$

And for circuit in figure 10

$$V_{out} = 2V_T tanh^{-1}(\frac{I_x}{I}) \tag{12}$$

Adding a bidirectional current to the bias current of the tangent cell and considering the same bias currents in both cells the

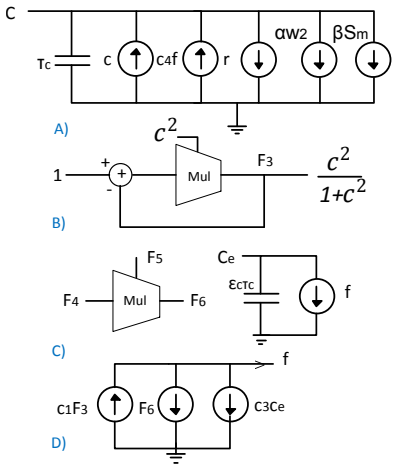

Fig. 12. Proposed circuits for implementation of synapse equations. Circuit in A, and B can be used for secondary mediator and gliotransmitter production too.

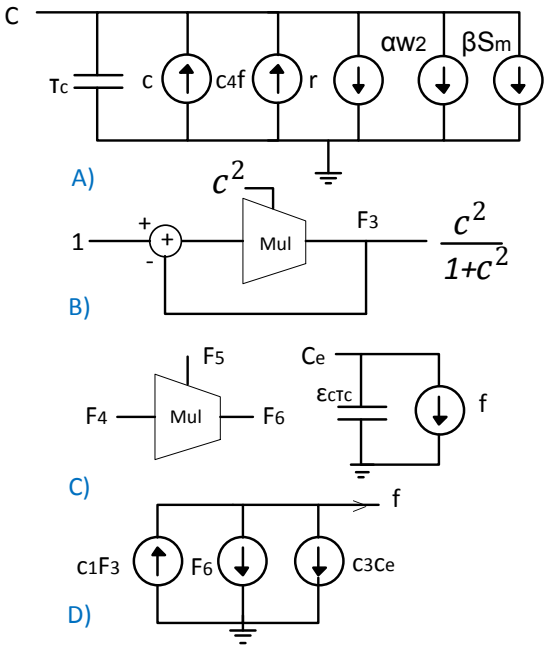

Fig. 13. Proposed circuit for Ca^{2+} and Ce concentration in astrocyte.

output of the Fig. 11 circuit will be:

$$I_{out1} = I_x + \frac{I_x I_y}{I1} \tag{13}$$

Subtracting the current I_x from equation 11 the output current becomes:

$$I_{out} = I_x \frac{I_x I_y}{I1} \tag{14}$$

Which is analog current multiplier. Fig.12 shows proposed circuit for implementation of synapse equations. In this circuit $v_i = V_1 - h_s$ that with Coefficient s_s by voltage dependent, voltage source is input of hyperbolic tangent circuit (Fig.12 A). Therefore, $F_1 = tanh(s_s(v_i - h_s))$. F_2 function is multiplication of F_1 and -z, $F_2 = -ztanh(s_s(v_i - h_s))$. Fig.12 B is implementation of equation 3 and I_{syn} can be obtained from circuit in Fig.12 C. Note that the equation for secondary mediator and gliotransmitter production (Equation 9,10) can be implemented by circuits in the Fig.12 A,B by changing

$$\begin{aligned} &h_s \rightarrow h_{s_m}, h_{G_m} \qquad s_s \rightarrow s_{s_m}, s_{G_m} \\ &h_s \rightarrow h_{s_m}, h_{G_m} \qquad v_1 \rightarrow z, c_a \\ &\tau_s \rightarrow \tau_{s_m}, \tau_{G_m} \end{aligned} \tag{15}$$

respectively. Fig. 13 shows proposed circuit for implementation of calcium oscillation and concentration within ER of astrocyte (Eq.7). The term $c^2 \backslash 1 + c^2$ is implemented by negative feedback loop while the open loop gain is c^2. The term $c_e^2 \backslash 1 + c_e^2$ and $c^4 \backslash 1 + c^4$ can be implemented with similar circuits by changing open the loop gain to c_e^2 and c^4 and presented by F_4, F_5 respectively (see Fig.13 C). Circuit for "f" function also shown in figure 13(D).

V. Conclusion

In this paper the AdEx neuron model coupled with a astrocyte model and mutual effects pathways investigated. AdEx is a biological relevant neuron model, which can produce different types of firing pattern in comparison to originally coupled FitzHugh-Nagumu model. The models describe neuron and asrocyte as a system of nonlinear differential equations. This models solved and simulated by numerical methods and the results have been shown. In the last section a circuit implementation for astrocyte differential equations presented.

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