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## **ACCEPTED MANUSCRIPT**

# A new bio-inspired stimulator to suppress hypersynchronized neural firing in a cortical network

Masoud Amiri<sup>1</sup>, Mahmood Amiri<sup>1\*</sup>, Soheila Nazari<sup>2,1</sup>, Karim Faez<sup>2</sup>

#### **Abstract**

Hyper-synchronous neural oscillations are the character of several neurological diseases such as epilepsy. On the other hand, glial cells and particularly astrocytes can influence neural synchronization. Therefore, based on the recent researches, a new bio-inspired stimulator is proposed which basically is a dynamical model of the astrocyte biophysical model. The performance of the new stimulator is investigated on a large-scale, cortical network. Both excitatory and inhibitory synapses are also considered in the simulated spiking neural network. The simulation results show that the new stimulator has a good performance and is able to reduce recurrent abnormal excitability which in turn avoids the hyper-synchronous neural firing in the spiking neural network. In this way, the proposed stimulator has a demand controlled characteristic and is a good candidate for deep brain stimulation (DBS) technique to successfully suppress the neural hyper-synchronization.

Keywords: DBS, spiking neural network, bio-inspired stimulator, astrocyte

### 1. Introduction

Neural synchronization in the nervous system is important for normal brain performance and it involves in short- and long-range of the different brain areas [1-2]. On the other hand, *epilepsy* is one of the most common neurological disorders, affecting about 1 % of the world's population. While the accurate mechanism of epilepsy pathophysiological changes in the brain is unclear, it has known as an intense neurological disorder clinically identified by hyper-excitability and/or hyper-synchrony in the cortex and other subcortical regions of the brain [3]. Disorders containing focal hyper-excitability of the brain have frequently seen in psychiatry and neurology [4].

Although hyper-excitability of individual neurons can contribute to the occurrence of epilepsy, it is widely proposed to be a "network disease" driven by improper synaptic interaction between neurons

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