BMC Neuroscience

Poster presentation

Open Access

The role of glia in seizures

Ghanim Ullah*[1], John R Cressman Jr[1], Ernest Barreto[2] and Steven J Schiff[1,3]

Address: [1]Center for Neural Engineering, Department of Engineering Science and Mechanics, Pennsylvania State University, University Park, PA 16802, USA, [2]Krasnow Institute for Advanced Study, George Mason University, Fairfax, VA 22030, USA and [3]Department of Neurosurgery, Pennsylvania State University, University Park, PA 16802, USA

Email: Ghanim Ullah* - ghanim@psu.edu

* Corresponding author

from Sixteenth Annual Computational Neuroscience Meeting: CNS*2007
Toronto, Canada. 7–12 July 2007

Published: 6 July 2007

BMC Neuroscience 2007, **8**(Suppl 2):P28 doi:10.1186/1471-2202-8-S2-P28

Summary

We present an ionic current model, composed of Hodgkin-Huxley type neurons and glia designed to investigate the role of potassium in the generation and evolution of neuronal network instabilities leading to seizures. We show that such networks reproduce seizure-like activity if glial cells fail to maintain the proper extracellular K^+ concentration.

Methods

Our neuronal network model combines the Hodgkin-Huxley type of formulism for the neuronal currents with a model for the dynamics of extra and intracellular K^+ concentration controlled by a glial network. The equations for the ionic currents are adopted from the model in ref. [1]. The extra and intracellular K^+ concentrations are calculated based on various K^+ currents.

Results

We investigate the instability in cortical networks by studying two interacting one-dimensional networks consisting of 100 pyramidal cells and 100 interneurons. The network exhibits persistent and spatially confined activity in a parameter range where inhibition is balanced by excitation. We then find various physiologic conditions under which a network displaying a stable persistent activity can switch to seizure like states.

Conclusion

The main finding of our study is that the network activity packet is stable provided that (1) the excitable synaptic strength is not very high; (2) the extracellular potassium concentration is low enough to be well in the physiological range (i.e. the glial network is functioning efficiently); and (3) perturbations to the network are not very strong.

Acknowledgements

Supported by NIH grants ROIMH50006 and K02MH01493.

References

1. Gutkin BS, Laing CR, Colby CL, Chow CC, Ermentrout GB: **Turning on and off with excitation: the role of spike-timing asynchrony and synchrony in sustained neural activity.** *J Comput Neurosci* 2001, **11:**121-134.