Neymotin et al. BMC Neuroscience 2010, 11(Suppl 1):P170
http://www.biomedcentral.com/1471-2202/11/S1/P170

BMC Neuroscience

POSTER PRESENTATION Open Access

Altered information transfer in neuronal networks marks pathology

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From Nineteenth Annual Computational Neuroscience Meeting: CNS*2010
San Antonio, TX, USA. 24-30 July 2010

Understanding how information flowing in neuronal networks is encoded by populations of neurons is a fundamental problem in neuroscience. Brains and neuronal networks must at the same time store information about the world and react to information in the world. We used simulations of neocortical columns to measure how the activity of the network alters information flow and related this to experimental data from animal models of epilepsy and schizophrenia. In our simulations, we found that networks with greater internal connectivity reduced information flow from inputs to outputs, measured by normalized transfer entropy [1]. With no internal connectivity, the feed-forward network *transformed* inputs through nonlinear summation and thresholding. With greater connectivity strength, the recurrent network *translated* activity and information due to contribution of activity from intrinsic network dynamics. This dynamic contribution amounted to added information coming from the network.

Gradual reduction in information flow-through with increased internal weight was asymptotic. At one extreme, the highly interconnected network was epileptic and any input ignited a seizure [2]. Such networks no longer accepted external information and no longer coordinated well with other brain areas. These types of networks also had more stereotyped activity patterns, with lesser information content present in the network and minimal information communicated from outside. EEG data from an animal model of epilepsy showed a similar reduction in entropy, which was also accompanied with a decrease in coordination between different brain areas.

At a lower level of dynamic pathology, abnormalities in power and coordination in gamma bands were seen, as has been noted in schizophrenia [3-5]. In schizophrenia, the core cognitive deficit is believed to be a problem of coordinating external and internal states to provide a coherent view of the world, and the person's relation to the world [6]. This is most evident with hallucinations, a mistaken attribution of internal activity to the external world. EEG data from an animal model of schizophrenia showed increased correlation between the power of oscillations in the gamma and beta bands, suggesting an over-reliance on internal information. This is analogous to the over reliance on internal information in our simulations of networks with overly high internal connectivity.

Our simulations provide a framework for relating aspects of epilepsy and schizophrenia to information processing. By quantifying the competing effects of internal and external information sources in simulation, we can suggest how symptoms of these disorders may be produced.

Acknowledgements
Research supported by National Institute of Mental Health R01MHO84038), National Institute of Neurological Disorders and Stroke R24NSO64474, NIH grants MHO82417 and MHO57068.

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Published: 20 July 2010

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doi:10.1186/1471-2202-11-S1-P170
Cite this article as: Neymotin *et al.*: Altered information transfer in neuronal networks marks pathology. *BMC Neuroscience* 2010 **11**(Suppl 1): P170.