Neuronal oscillations as a critical phenomenon and its implications for information processing

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Summary

Information processing in the brain happens at multiple levels, from integrating information at the single neuron level, to communication between neurons and neuronal networks at different spatial and temporal scales. Neuronal oscillations and, more recently, neuronal avalanches have been proposed to play an important role in this communication. Interestingly, both oscillations and neuronal avalanches exhibit scale-free dynamics, a hallmark of critical behavior. Empirical studies have shown that scale-free modulation of oscillations can predict behavioral variability in humans, and is altered in epilepsy, schizophrenia and Alzheimer's disease. Theoretical studies have linked criticality in terms of neuronal avalanches to desired information-processing characteristics such as reactivity, adaptability and robustness to input, and the activation of neuronal representations in the form of meta-stable activity patterns. This led to the questions of whether these different forms of critical-state dynamics are related, and what is the functionality of critical oscillations?

The major aim of this thesis was to understand the mechanism and information processing functionality of scale-free amplitude modulation of oscillations. To accomplish this I created a neuronal network model where scale-free oscillations emerged through the mechanism of balanced excitatory and inhibitory connectivity. I found that networks poised at this balanced state also showed scale-free spreading of activity in the form of neuronal avalanches. By integrating the previously separate fields of neuronal avalanches and oscillations into a new form of network dynamics, multi-level criticality, it raised the possibility that modulations in oscillations may be used as a proxy to predict changes in the neuronal criticality and excitation-inhibition ratio of a network. It also allowed me to investigate the implications of critical oscillations for information processing in neuronal networks.

To understand how critical oscillations affect neuronal network function I stimulated networks which possessed sub-, critical and super-critical dynamics. I found that state-dependent information processing capabilities that had been previously linked to

neuronal oscillations only occurred close to the critical state. In other words, to understand the momentary reactivity of a network, insight into the dynamic state over long time periods is needed, which can be detected by the proximity of the oscillatory dynamics to the critical state. This finding has wide-ranging implications for understanding and detecting behavioral variability, and loss of function, in healthy and diseased neuronal networks.

Biomarkers of disordered neuronal networks are essential for the detection and treatment of disease. Imbalances in the excitation-inhibition ratio (E/I) at the cellular and network level have emerged as a potential biomarker of multiple disorders including autism and schizophrenia. However, it has remained difficult to define E/I at the neuronal network level and detect it using non-invasive recordings in humans. Using the framework of critical-state dynamics and the model of critical oscillations that I had developed, I investigated whether it was possible to estimate E/I ratio from neuronal network oscillations. This led to a new biomarker, $\widehat{E/I}$, that could be applied to magneto- and electroencephalography (M/EEG) recordings. Applying this new biomarker to human subjects performing a threshold stimulus detection task, we found that $\widehat{E/I}$ during the task varied between subjects, and that these differences could explain the neuronal network response to the stimulus. This biomarker has great potential for detecting imbalances in E/I, which would be useful for disordered networks and their response to treatment.

Overall, in this thesis, I show that scale-free neuronal oscillations can be understood through the framework of criticality, and that by creating a neuronal network to model them it is now possible to understand the effects of these dynamics on the information processing capabilities of neuronal networks. This will allow for a greater understanding of how and why the dynamics of neuronal oscillations are altered in healthy and diseased networks.