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Presentation Abstract

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Presentation Title: Systematic *in vivo* electroencephalographic characterization of spatial phase coupling in mouse models of schizophrenia

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Abstract: It is well known that certain phenotypes of neural circuit dynamics, such as anomalous levels of power and/or spatial phase coupling in the gamma and theta frequency ranges, are characteristic of mental disorders like schizophrenia. In order to uncover the underlying neurobiological mechanisms of these spatio-temporal network abnormalities, it is important to model the disorder in model species such as mice. However, work of this kind is currently faced with two long-standing challenges. First, due to the 3 to 4 orders of magnitude difference in brain volume between mouse and human, conventional techniques capable of probing spatial interactions among distinct brain areas in human subjects, such as electroencephalography (EEG), cannot be directly translated to the mouse without properly standardized miniaturization, leaving a blank in the work of EEG characterization of schizophrenia-related phase coupling across distinct cortical loci in freely behaving mice. Secondly, the existence of numerous and hugely diverse mouse models of schizophrenia, as a result of the complexity in etiology of the disease, calls for a reliable means to compare and contrast specific neural circuit mechanisms consequent of a wide range of genetic, pharmacological and environmental perturbations that result in schizophrenia-like behavioral phenotypes. Although neurochemical commonalities, such as NMDA receptor hypofunction and deficient inhibitory neurons, have been reported in

multiple mouse models, a systematic search of converging circuit level characteristics across different models has been so far lacking.
We have approached these problems by devising a new method of multi-channel epidural EEG recording in freely moving mice. The method proved to be highly accurate, efficient, reliable and inexpensive, ideally suited for large-scale in vivo studies using mouse models of mental disorders. We first applied this method to a well-documented genetic model, and discovered unique global patterns of theta and gamma phase coupling across distinct cortical recording sites, which had been impossible to identify by using conventional EEG methods in mice. We further attempted to make lateral comparisons among multiple mouse models of schizophrenia, models that involved diverse mechanisms of induction including pharmacological, genetic, as well as behavioral interventions. In sum, we managed to establish a convenient platform on which spatio-temporal cortical dynamics can be detected with high fidelity and compared for commonality and distinctiveness among different mouse models of the same mental disorder.

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