



Presentation Abstract

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Presentation Title: Altered number of neural population activity patterns in Fragile-X mice

Location: Hall A

Presentation time: Tuesday, Oct 20, 2015, 8:00 AM -12:00 PM

Presenter at
Poster: Tue, Oct. 20, 2015, 10:00 AM - 11:00 AM

Topic: ++C.06.g. Fragile X

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Abstract: Brain disorders such as Fragile X syndrome (FXS), autism and schizophrenia have been associated with different changes in neural function, such as an imbalance in excitation and inhibition, hyperexcitability, and altered synaptic connectivity. Why or how these particular changes would be detrimental for neural coding remains unknown. Here we test the hypothesis that these cellular changes affect information processing

by shifting the typical number of activity patterns used by the circuit. We analyzed spontaneous activity of neural populations in somatosensory cortex recorded in vivo using two-photon calcium imaging in both wild-type and Fmr1 knockout mice, a model for FXS. We developed a new statistical model for the probability distribution of all 2^N possible neural population activity patterns that required only N^2 parameters, where N is the number of neurons. Using this analysis method, we found that even subtle alterations in neural firing rates and correlations in Fmr1 knockout animals, relative to wild-type, across development lead to dramatic shifts in the typical number of circuit activity patterns. Adolescent Fmr1 knockout mice showed fewer neural activity patterns than wild-type, while the opposite was true in adult mice. Using a computational model of mouse layer 2/3 somatosensory cortex, we explored the contributions of the underlying circuit components, leading to two main conclusions: first, there are multiple non-exclusive neurobiological routes to altered circuit dimensionality. Second, the type of parameter changes needed to reverse the FXS deficits is qualitatively different in adolescent versus adult mice. If also true in humans, this result suggests that a pharmaceutical intervention that can successfully reverse symptoms in adults with FXS might prove ineffective or possibly even exacerbate symptoms in children with this disorder. Altogether, our findings show how relatively small changes in neural circuit parameters in brain disorders can have dramatic consequences for information processing.

Disclosures:

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