



NEUROSCIENCE AND PHYSIOLOGY SEMINAR SERIES

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"Sleep Architecture in Fmr1 Knockout Rats."

Children with neurodevelopmental disorders, including autism, frequently present with both sensorimotor deficits and poor sleep. Infancy is a period of heightened neural plasticity and extensive sleep, especially rapid eye movement (REM) sleep, during which sensorimotor circuits undergo substantial refinement. However, whether early sleep disruptions are linked to altered sensory processing in autism remains unclear. To investigate this link, we turned to Fragile X Syndrome (FXS), the most common monogenic cause of autism. This study aims to characterize sleep differences between Fmr1 knockout rats, an animal model of FXS, and wild-type controls. Using in vivo extracellular electrophysiology, we recorded neural activity from male wild-type and Fmr1 knockout rats on postnatal days 16 and 20 as they cycled between sleep and wake. Although we see no difference in the overall amount of REM sleep, our preliminary findings show that P16 Fmr1 knockouts have shorter REM sleep bouts, cycle more rapidly between sleep and wake, and exhibit slower theta oscillations during REM sleep. Together, these results are consistent with a developmentally delayed sleep architecture compared with that of the wild-type controls and may offer insights into early cortical development and sleep architecture in autism.

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