

Sleep Fragmentation in 16p11.2 Deletion Mouse Model of Autism

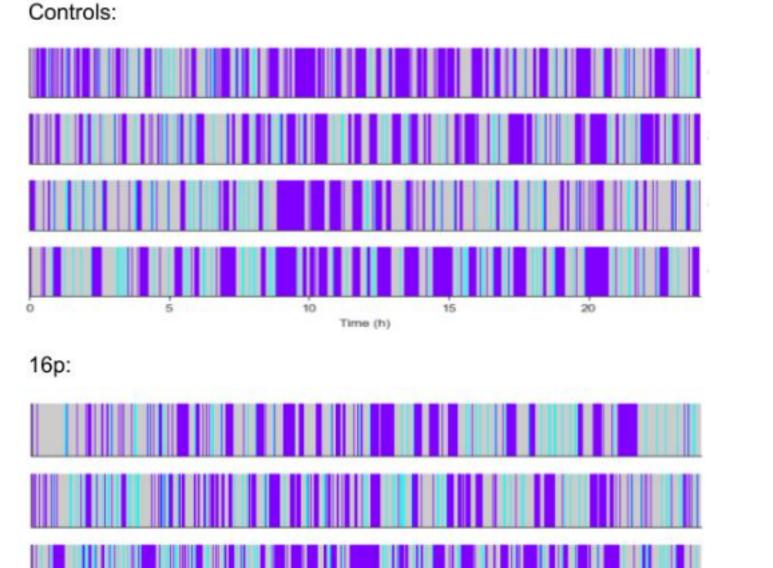
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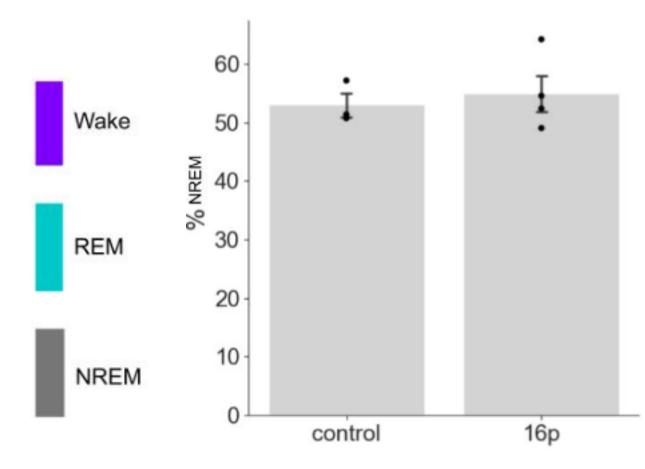
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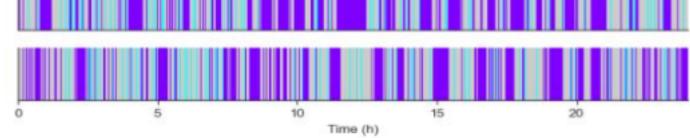
Objective

- Individuals with autism spectrum disorder often suffer sleep disturbances/deficits
- This study aims to determine if the 16p mouse line is a valid ASD mouse model for studying sleep
- These figures show the 16p mouse line exhibits similar sleep disturbances (like sleep fragmentation increased awakenings) to those seen clinically in ASD patients

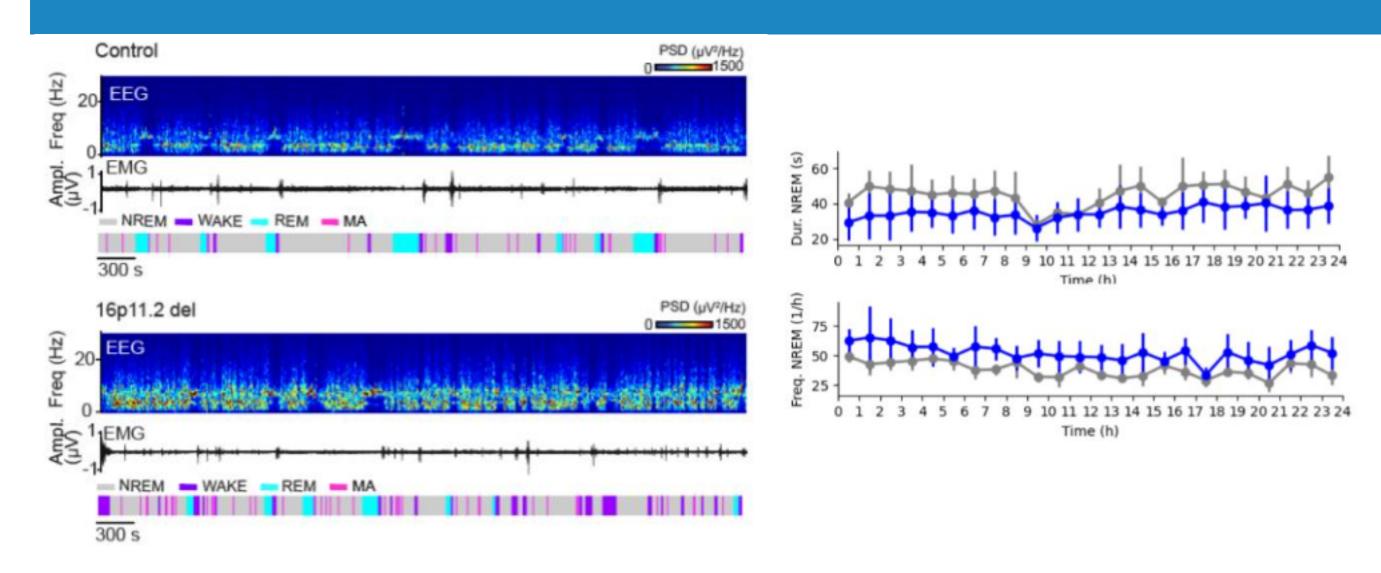
16p mice and controls spend comparable amount of time in Wake, NREM, and REM sleep







Fragmented sleep in 16p mice:



Increased number of microarousals in 16p mice vs. controls

NREM bouts are more frequent but shorter in 16p mice compared to controls

Approach

- The 16p knockout mouse has a deletion at chromosomal region 16p11.2, and studies have shown they display hyperactivity and sexrelated bias of sleep symptoms
- Used electroencephalogram (EEG) and electromyography (EMG) recordings of 16pxB129 mice and control

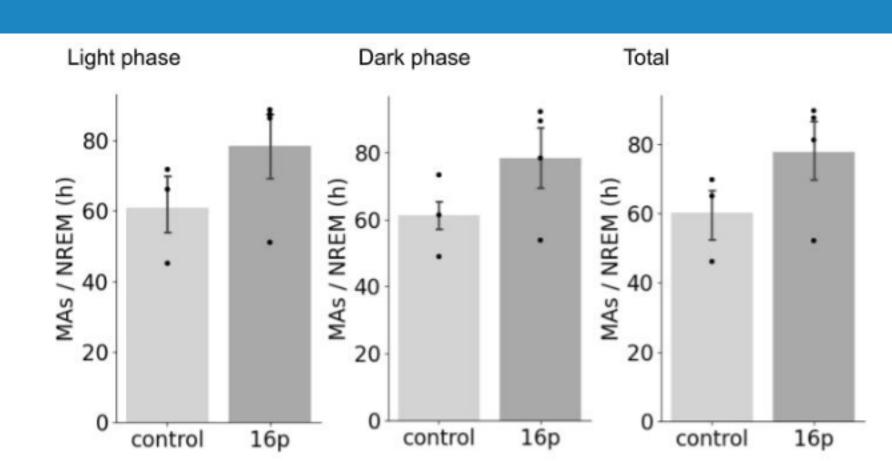
• 24 hour baseline recordings

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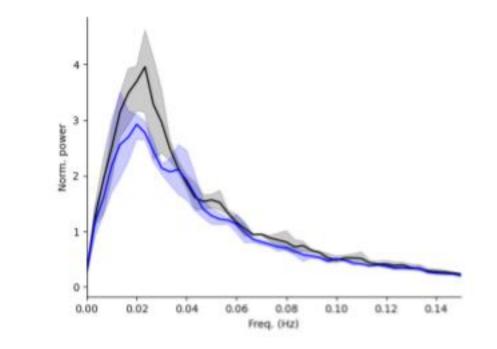
Conclusions

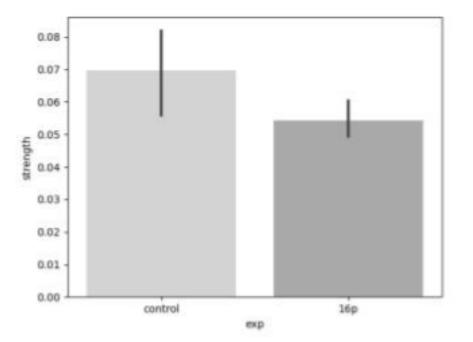
Microarousals and infraslow rhythm

- There was no difference in NREM percentage between 16p mice and controls
- While there is no difference in NREM percentage, microarchitecture of sleep in 16p mice is more fragmented compared to controls
- Exploring neural circuit mechanisms contributing to this sleep fragmentation is a future direction

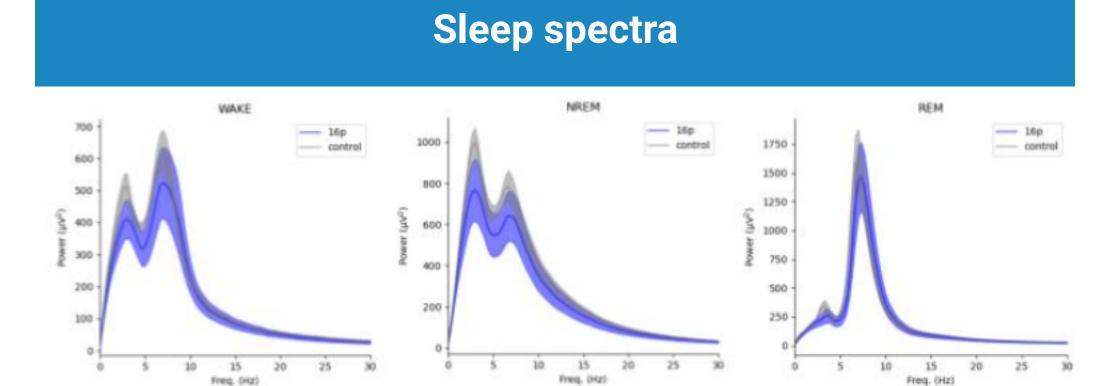


More microarousals in 16p mice during light and dark phase





Infraslow rhythm is weaker in 16p mice



Similar delta, theta, and sigma power during Wake, NREM, and REM sleep

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