Early sleep loss accelerates Alzheimer's pathology in mice
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(A) Schematic of the CSS paradigm, where total sleep deprivation (SD, red bars) occurred at the onset of the first three lights-on (light blue) periods of the week (L1, L2 and L3) for 8 consecutive hours. Mice were returned to home cages for the last 4 hrs of the L1, L2 and L3 periods and the ensuing lights off (dark blue bars, D1, D2 and D3) periods. Mice were left undisturbed in home cages for days 4-7 each week. The pattern was repeated weekly for 4 consecutive weeks. (B) Following CSS and Rest control conditions, mice recovered 3 mos before behavioral tests at ages 5 and 7 mos and then an additional 2-3 mos before undergoing the specified protein analyses at ages 9-10 months. Credit: Zhu et al. Fig. 1, JNeurosci (2018)

onset of the motor impairments that develop in this mouse model and increased tau pathology in the locus coeruleus—the site of some of the earliest degeneration in AD—and portions of the amygdala. CSS also exacerbated loss of neurons in both regions.

Together with previous findings that sleep loss contributes to accumulation of amyloid plaques—the other main culprit in AD—these results demonstrate the influence of sleep on neurodegeneration in aging.


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Lack of sleep during adolescence and early adulthood accelerates Alzheimer's disease (AD)-related tau pathology, finds a study of male and female mice published in JNeurosci. These results support the importance of establishing healthy sleep habits in early adult life to help stave off progression of neurodegenerative diseases.

Using a mouse model of tauopathy, Sigrid Veasey and colleagues investigated two forms of sleep disruption increasingly common in modern society: chronic short sleep (CSS) and chronic fragmentation of sleep (CFS).

Both forms of disrupted sleep led to an earlier