In the control condition, participants could sleep 8h/night throughout the entire protocol. Blood samples were taken after baseline sleep, after 5 nights of restricted or control sleep, and after 2 nights of recovery sleep. Data were analyzed using generalized linear mixed models.
Results: Sleep restriction was associated with decreased capacity of GCs to inhibit COX-2 expression in monocytes ($p<.01$) and has the expected inflammatory effect on IL-6 production in monocytes ($p<.01$). Moreover, sleep restriction has lasting inflammatory effects as shown in increased inflammation following 2 nights of recovery sleep ($p<.01$).
Conclusion: In conclusion, the present preliminary analysis suggests that in patients treated with GCs, sleep restriction potentially reduces their effectiveness in controlling inflammation, thus contributing to increased inflammation-related morbidity. Sample collection and data analysis is ongoing.
Support (If Any): NIH/NHLBI R01-HL136310; NIH/NCRR UL1-RR02758, M01-RR01032; German Research Foundation (DFG) EN1291/1-1.

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ROLE OF COFILIN AND CALCIUM SIGNALING IN SLEEP-LOSS NEURAL INJURY

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Introduction: Chronic sleep disruption (CSD) in young adult mice leads to phenotypes consistent with early (pre-plaque) Alzheimer's Disease (AD), including increased Aβ and hippocampal neuron loss. Mechanisms underlying this injury are not known. Both acute sleep loss and AD activate cofilin, a regulator of actin dynamics. Activated cofilin (AC) in AD mouse models can impart neural injury, increase Aβ, and cofilin translocation to the mitochondria delays cytosolic Ca2+ clearance. We are critically testing the role of AC in chronic short sleep (CSS) and sleep fragmentation (SF) neural injury.
Methods: Synapse loss was studied using STED confocal microscopy and Imaris in CSS (n=9) and rested (n=10) mice. Synapses were identified as overlaps of pre- and postsynaptic densities. Percent area of cofilin was measured with FIJI. To further understand if and how wake-induced cofilin activation induces sleep-loss synapse and neural injury, we implanted AAV9CAMKII-GCaMP6f and then GRIN lenses, and later studied CAMKII calcium transients in CA1 of WT controls (n=4) and SF mice (n=4) by measuring GCaMP6f calcium transients. We developed a Shiny R application to analyze the frequency of Ca2+ spikes, ΔF/F0, and the rising and clearance patterns of spikes. To directly test cofilin's role in delayed calcium clearance, we studied the calcium transients in hAPP mice (n=2) after injection of AAV-CAMKII-CofilinS3A to express AC and GCaMP6f. All data were analyzed with two-way ANOVA or unpaired t-tests.
Results: Results reveal significant synapse loss in CA1 of CSS mice (CSS=48.8±10.3; Rested=83.4±8.4), $t(16)=2.63$, $p<0.02$, and increased cofilin activation (AC=19.8±3.41; Rested=8.76±1.95), $t(16)=8.43$, $p<0.0001$. SF mice reveal an increase in NREM sleep firing rates, $F(1,1)=22.0$, $p<0.001$. In contrast, hAPP-AC mice show significantly increased ΔF/F0, $F(1,1)=356$, $p<0.0001$, prolonged calcium influx, $F(1,1)=18.6$, $p<0.02$, and prolonged calcium clearance duration, $F(1,1)=23.9$, $p<0.01$, but not increased firing frequencies.
Conclusion: CSS induces CA1 synapse loss and cofilin activation in WT mice. Increased CAMKII calcium ΔF/F0 occurs through different pathways in SF and AC, suggesting additional factors in CSD neural injury.
Support (If Any): NIH AG054104; AG064231

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AFFECT AND AFFECTIVE PERSONALITY DURING A SIMULATED FIRST NIGHT SHIFT

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Introduction: Shiftwork contributes to work-related stress and chronic partial sleep deprivation. A first night shift can be particularly problematical for many workers since they are required to adjust to working at night following a period of sleeping at night. Part of this adjustment involves emotional stability and reactivity including affect. The purpose of this study is to examine how affect and a related construct, affective personality, changes during a simulated first night shift.
Methods: Ninety undergraduate students, 60 males and 30 females (22.1 ± 3.0 years), participated in a simulated first night shift. As part of the night shift, the participants completed the PANAS test (using the cue: how they felt lately) assessing positive and negative affect four times during the simulated shift (6:30 pm, 10:30 pm, 3 am, 7:30 am). The PANAS data were grouped using median split values from the 6:30 pm survey into high and low positive and negative affect groups to represent four categories of affective personality (Self-Actualizing: high positive, low negative; High Affective: high positive, high negative; Self-Destructive: low positive, high negative; Low Affective: low positive, low negative).
Results: A 2x4 ANOVA found significant changes across the night for affect ($p<.0001$), a significant difference between positive and negative affect ($p<.0001$), and a significant interaction ($p<.0001$) with positive affect decreasing during the night but negative affect remaining stable. A Friedman Test found significant changes in affective personality across the night ($p<.0001$) with decreasing occurrences of Self-Actualizing and High Affective but increasing occurrences of Self-Destructive and Low Affective personality types.
Conclusion: These findings suggest that positive affect decreases during a first night shift which could reduce workers' competence resulting in more performance errors and potential safety hazards. The change in affective personality suggests that these groupings are not personality traits but instead could be considered affective personality states. Managers and organizations should anticipate decreases in the positive affective personality states during a first night shift and may find that workers will be less adaptive and efficient.
Support (If Any): This research funded in part by the Creative Inquiry Program at Clemson University.