Category G—Sleep Deprivation

(+/- 3.7) in REM, while during the fragmented night, participants spent 14.8 minutes (+/-6.8) in stage 1, 16.0 minutes (+/- 6.6) in stages III and IV, and 15.6 minutes (+/- 3.2) in REM. The arousal index was 8.0 (+/-2.9) during the baseline night 16.0 (+/- 4.4) during the fragmented night. Fragmentation altered functional activation as follows: Both sleep deprivation and sleep fragmentation reduced activation in the executive network, including in the anterior cingulated cortex. Fragmented sleep resulted in greater activation reductions than sleep deprivation. ANOVA revealed significant effects for baseline non-response, baseline-fast, and task non-response. Contrast analyses revealed that these effects were due to differences between rested and deprived conditions for task non-response and baseline-fast, and between rested and deprived and rested and fragmented for baseline non-response. Performance differences between fragmented and deprived conditions were not significant, while activation was.

Conclusion: Sleep fragmentation and deprivation may have differential effects on components of complex cognitive tasks. The results have implications for sleep apnea and conditions associated with acute sleep fragmentation, such as environmental noise.

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0484

IMPACT OF ACUTE AND CHRONIC SLEEP RESTRICTION ON PVT PERFORMANCE: A STUDY OF MEDICAL RESIDENTS

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Introduction: Under controlled laboratory conditions, chronic sleep restriction of 3 to 6 hrs/night for 7-14 nights gradually degrades Psychomotor Vigilance Task (PVT) performance. Less is known about whether this accumulative decrement occurs outside the laboratory. US medical residents work 24- to 30-hr extended shifts twice per week, resulting in both acute and chronic sleep loss. Here, we address the impact of working repeated extended duration work shifts on PVT performance.

Methods: Seventeen PGY-1 medicine residents (26-32y, 7F) worked 30-hour shifts twice per week in an intensive care unit (ICU) for three weeks, such that they worked at least six extended duration shifts; this followed 3 weeks of non-extended shifts. Subjects completed 10-min PVTs 3-6 times at regular intervals across each extended duration shift. Mean log_reaction time (RT) and transformed lapses greater than 500ms $(\sqrt{n}+\sqrt{(n+1)})$ were assessed for change over time (extended shifts block 1-6). Controlling for time of day, we assessed differences in performance at the start and end of each shift as a function of the number of extended shifts.

Results: Mean RT and lapses worsened with increasing number of extended shifts (one-way repeated measures ANOVA, p<0.05, p<0.02, respectively), with post hoc comparisons showing performance to be worse during the last two extended shifts as compared to the first two (p<0.008). For overnight performance (23:00-08:00h), there was a significant effect of extended shift number for mean RT (p<0.05) but not lapses. For day-time performance (7:00h-14:00h), both mean RT and lapses showed a significant worsening with increasing number of extended shifts (p<0.009, p<0.003, respectively) and time on shift (p<0.002, p<0.0005, respectively).

Conclusion: Residents working traditional on-call schedules exhibit poorer performance in the second half of each 24-30-hr extended shift as a result of acute sleep deprivation, and show a chronic deterioration in performance with increasing number of extended shifts.

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0485

CHRONIC SLEEP RESTRICTION AUGMENTS THE ACQUISITION OF DRUG-SEEKING AND DRUG-TAKING BEHAVIORS IN RATS

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Introduction: Substance abuse is a major concern within the United States, compounded by the propensity of many addicted individuals to relapse. The clinical literature suggests sleep deprivation is a factor that can induce relapse in humans, and, in fact, our society is plagued by chronic sleep deprivation. Previously, our lab demonstrated that acute sleep deprivation reliably increases the rate and efficiency of drugseeking and drug-taking behaviors in otherwise unresponsive, low drugtaking rats. The current study utilized a chronic sleep restriction model to further elucidate the effects of sleep deprivation on drug addiction in a model more similar to that experienced by the human population.

Methods: Naïve male Sprague-Dawley rats were chronically deprived of approximately 30% of their baseline sleep (CSD; n=20) or served as non-sleep-deprived controls (NSD; n=15). During the same time period, all rats were trained to self-administer cocaine on a fixed ratio (FR) schedule of reinforcement. In addition, progressive ratio (PR) testing was used to assess the willingness of the rats to work for cocaine.

Results: In accordance with our acute sleep deprivation data, CSD rats self-administered more cocaine infusions during FR training, compared to NSD controls. Also, CSD rats exhibited higher break points (i.e., worked harder for cocaine), shorter inter-infusion intervals (i.e., self-administered cocaine more quickly), and more goal-directed behavior (i.e., focused more exclusively on the drug-associated operandum) than NSD controls during PR testing.

Conclusion: These data have profound clinical implications, and highlight the importance of the awareness of potential relapse-inducing factors, such as sleep deprivation, in the treatment of drug addiction.

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0486

GENERAL INTELLECTUAL FUNCTIONING DOES NOT PREDICT PERFORMANCE IMPAIRMENT ON THE PSYCHOMOTOR VIGILANCE TEST DURING TOTAL SLEEP DEPRIVATION

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Introduction: There are substantial, trait-like individual differences in performance impairment on the psychomotor vigilance test (PVT) during total sleep deprivation (TSD). Based on theories of cognitive reserve and compensatory brain mechanisms, we hypothesized that overall cognitive ability might predict individual differences in PVT impairment due to TSD. In the present study, we aimed to test this hypothesis using the Shipley Institute of Living Scale (SILS), a validated measure of general intellectual functioning.

Methods: As part of a larger study, 12 healthy young adults (age 27.4±4.5; years of education 14.3±1.9; 5 females) spent 7 consecutive days in a laboratory. Following two baseline days with 10h time in bed, subjects underwent 62h of TSD. Performance on a 10min PVT was tested at 2h intervals throughout most scheduled wakefulness. PVT number of lapses (RT>500ms) was averaged across a baseline period from 0h to 14h awake; across the 24h TSD period from 14h to 38h awake; and across the subsequent 24h TSD period from 38h to 62h awake. The SILS was administered during baseline. Raw SILS scores were converted to

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