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SIMULATED NIGHT WORK ACUTELY IMPAIRS GLUCOSE TOLERANCE

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Introduction: About 8.6 million Americans regularly undertake night work, which is associated with type 2 diabetes (T2D) risk in epidemiologic studies. Such observational studies cannot definitively demonstrate causality or underlying mechanisms. Here, using an experimental design, we tested the hypothesis that simulated night work impairs glucose tolerance.
Methods: Thirteen healthy, non-obese adults (8 men, 20-49 years of age) without significant shift work history completed two, 8-day, in-laboratory protocols in random order, one including day work and the other night work (sleep/wake and fasting/feeding cycle inverted by 12 h). Each condition included 4 baseline days, followed by either day or night shifts. The diet was isocaloric, identical between conditions, and included standardized mixed meals on Days 1 and 3 of day/night work to assess serum glucose and insulin responses. Subjects began eating the meal at 8AM (day work) or 8PM (night work) and were required to finish eating in 20 min. Blood was sampled pre-meal (fasted), then every 10 min for 90 min, then every 30 for 90 min.
Results: Here, we only show results pertaining to mixed meals consumed on Day 1 of day work and night work. Fasting glucose levels were similar between conditions (mean±SEM: day work, 85±2 mg/dl vs. night work, 85±2 mg/dl; P=1.00). During night work, glucose 3-h area-under-the-curve (AUC) was 23% higher (342±17 mg/dl.3 h vs. 419±13 mg/dl.3 h; P<0.0001) and peak glucose levels were 16% higher (166±7 mg/dl vs. 193±4 mg/dl; P<0.0001). There was a significant interaction of condition*time-since-meal (P<0.0001), with slower decline of glucose levels during night work. Fasting insulin levels were 15% lower during night work (5.1±0.8 uIU/mL vs. 4.3±0.7 uIU/mL; P=0.035). Insulin 3-h AUC was not significantly different but peak insulin levels were 13% lower during night work (99±15 uIU/mL vs. 86±13 uIU/mL; P=0.035). For insulin, there was a significant interaction of condition*time-since-meal (P<0.0001); with 33-41% lower levels at min 20 (P=0.002) and min 30 (P=0.002), but 40-50% higher levels at min 80 (P=0.005) and min 90 (P=0.025), possibly due to the elevated glucose values during night work.
Conclusion: Night work per se can impair glucose tolerance. This is associated with lower postprandial early-phase insulin levels. These findings support a causal role of night work in the increased T2D risk among shift workers.
Support (If Any): NHLBI-R01-HL094806; NASA-NCC-9-58; 1-UL1-RR025758-01

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ARMODAFINIL IMPROVES CREATIVITY PERFORMANCE ON THE REMOTE ASSOCIATES TEST IN NIGHT WORKERS WITH SHIFT WORK DISORDER

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Introduction: Armodafinil is a wakefulness promoting agent used for the treatment of excessive sleepiness (ES) in individuals with Shift Work Disorder (SWD). In the present study, we determined the effect of armodafinil on creativity as measured by the Remote Associates Test (RAT), which was developed as an associative evaluation of the process of creative thinking. We further attempted to determine if this improvement was related to changes in alertness as measured by the multiple sleep latency test (MSLT).
Methods: Participants were 10 night workers (age: 44.2±9.78 years, 8F) that complained of ES (≥10 on the Epworth Sleepiness Scale (ESS); mean=13.8±2.9), meeting ICSD-2 criteria for SWD, and having no other sleep or medical disorders. During each 12 hour study (2130-0930), subjects were kept awake in a moderately lit room (500 lux in the angle of gaze) and out of bed except for MSLT (0130, 0330, 0530, and 0730). Wakefulness was monitored using polysomnography (PSG). Armodafinil (150 mg) or placebo was administered (2345) in a randomized, double-blind, crossover design. At 0400, subjects were read instructions and given 40 minutes to complete the RAT.
Results: Armodafinil significantly improved both RAT and MSLT scores: there was a 26% improvement in RAT score with armodafinil administration compared to placebo (8.2±4.6 vs. 10.3±5.6, p=0.03), and a 5 minute increase in MSLT score (4.8±2.6min vs. 9.9±4.2min, p=0.005). A repeated measures ANCOVA controlling for MSLT score difference found that armodafinil's effect on RAT score remains significant [F(1,7)=8.28, p=0.02], suggesting that RAT score improvement is, at least in part, independent of armodafinil's effect on alertness.
Conclusion: Armodafinil improves creativity as measured by the RAT in night workers with SWD marked by ES. Importantly, covarying for alertness improvement did not negate the effect on creativity.
Support (If Any): This study is supported by investigator initiated grant C10953/6308 awarded to C. Drake by Teva Pharmaceutical Industries Ltd.

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ENDOGENOUS CIRCADIAN TEMPERATURE AND MELATONIN PERIOD LENGTHS IN PATIENTS WITH DELAYED SLEEP PHASE DISORDER

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Introduction: The present study aimed to identify the underlying mechanisms of Delayed Sleep Phase Disorder (DSPD) that is characterized as an abnormally delayed sleep period. The currently assumed aetiology is simply a phase-delay in individuals' biological body clocks. However, DSPD cases treated to produce a corrective phase advance are prone to relapse. It has been suggested that this may be due to an abnormally long period length (time taken to complete one cycle of the rhythm) in DSPD. Circadian period lengths of endogenous core body temperature and salivary melatonin were measured to investigate this premise.
Methods: Following rigorous screening procedures, nine healthy controls and nine persons with DSPD were selected for a 80-hour ultradian modified constant routine protocol residing in a dimly lit (<15 lux), time-free environment. They followed "1-hour days" which involved 20-minute sleep opportunities alternating with 40-minutes of enforced wakefulness. Core body temperature and salivary melatonin were recorded hourly and best fit temperature curves and dim light melatonin onsets were determined to derive circadian period length measures during the 80-hours.
Results: Although core temperature period lengths showed a trend in DSPD to be longer than controls, this trend was not statistically significant in this study. However, the melatonin period length was on average 23.4 minutes longer (p = .010) in the DSPD group (M = 24.64 hrs, SD = 0.35) than the healthy control sleepers (M = 24.25 hrs, SD = 0.21).
Conclusion: Together these findings suggest that abnormally long biological circadian rhythms contribute to delayed sleeping patterns of individuals with DSPD. These outcomes may explain patients' persistent tendency to delay and relapse post-treatment. Therefore, continuing treatment with morning bright light stimulation and early evening low dose melatonin administration are recommended to treat DSPD and prevent relapse.
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