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BRIGHT LIGHT DURING WAKEFULNESS IMPROVES OBJECTIVE AND SUBJECTIVE SLEEP QUALITY: A FORCED DESYNCHRONY STUDY

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Introduction: Under real life conditions, increased light exposure during wakefulness seems associated with improved sleep quality, quantified as reduced time awake during bed time, increased time spent in non-REM (NREM) sleep or increased power in the EEG delta band (0.5–4 Hz). The causality of these important relationships and their dependency on circadian clock phase and/or time awake has not been studied in depth. To establish causality of light effects during wake time on subsequent sleep, and to disentangle possible circadian and homeostatic interactions, we employed a forced desynchrony (FD) protocol under dim light (6.5 lux) and bright light (1307 lux) during wakefulness.

Methods: The protocol consisted of a fast cycling sleep-wake schedule (13h wakefulness – 5h sleep; 4 cycles), followed by 3h recovery sleep in a within subject cross-over design. Individuals (7 men) were equipped with 10 polysomnography electrodes. Subjective sleep quality was measured immediately after wakening.

Results: Results indicated that circadian variation in delta power was only detected under dim light. Circadian variation in time in rapid eye movement (REM) sleep and wakefulness were uninfluenced by light. Prior light exposure increased accumulation of delta power and time in NREM sleep, while decreasing wakefulness, especially during the circadian wake phase. Subjective sleep quality scores showed that participants were only able to assess light induced improvement of sleep quality correctly when the circadian system promoted wakefulness.

Conclusion: This study presents significant effects of bright light exposure on sleep architecture, leading to sleep pressure related changes in objective sleep quality. At the end of the scheduled sleep phase after increased light exposure, more delta power and NREM sleep were detected, especially when sleep occurred outside the normal sleep phase. Subjective sleep quality scores showed light-induced improvements coinciding with increased delta power and time spend in NREM sleep, suggesting that light during wakefulness may improve subsequent sleep quality. These findings may have important implications for insomnia treatment and clinical applications of light therapy.

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HUMAN ACTIVITY LEVELS REFLECT CIRCADIAN INFLUENCES INDEPENDENT OF SLEEP/WAKE BEHAVIOR

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Introduction: Actigraphy is a non-invasive method that allows long-term recordings of activity, light, and other variables in diverse environments. In real-world settings, activity usually has a 24-hour rhythm that may arise from sleep/wake-associated behavior and/or circadian rhythmicity. We tested for an independent circadian component using data from people living on non-24 hours "days" in the laboratory.

Methods: Data are from five inpatient studies with tightly-controlled forced desynchrony (FD) conditions. Participants (19–34 yo) were healthy by history, physical exam, laboratory tests of blood and urine, and clinical polysomnography, and did not report using prescription medicines. Caffeine-containing substances were prohibited during the study. Protocol 1: 7 participants (3 F) T-cycle (i.e., FD sleep-wake cycle duration) = 42.85h; Rest:Activity ratio 1:3.3. Protocol 2: 8 participants (3 F) T cycle =42.85h; Rest:Activity 1:2. Protocol 3: 9 participants (3 F) T cycle =28.0h; Rest:Activity 1:2. Protocol 4: 7 participants (3 F) T cycle =20.0h; Rest:Activity ratio 1:3.3. Protocol 5: 7 participants (5 F) T cycle =20.0h; Rest:Activity 1:2. At all times except during showers, participants wore an actiwatch that measured activity levels and light. Melatonin period and phase 0 (i.e., fit maximum) were computed using non-orthogonal spectral analyses. Data were analyzed relative to 3-hr Circadian Phase bins (1/8 of computed circadian period for each individual) and 3-hr Wake Duration bins. Activity data were summarized using Zero-Inflated-Poison-based statistics for each Circadian*Wake Duration bin for each individual and then across individuals within each study. Repeated measures ANOVA were conducted. Statistics were performed using SAS.

Results: For all protocols, there were significant differences (all $p<0.007$) by individual participant, by Circadian Phase, and by Wake Duration bin, but not by the interaction term (Circadian Phase* Wake Duration). Highest levels of activity were at Circadian Phase 7.5–10.5 (~10am–1pm) and lowest values at Circadian Phase -1.5–1.5 (~midnight–3 am). Activity values were lowest at scheduled sleep times.

Conclusion: Circadian rhythms independent of sleep/wake behaviors influence activity levels and may be an important component of analyses. In individuals living on non-24-hr days (e.g., some blind people and some sighted people with Non-24-hr Sleep Disorder), it may be possible to derive circadian-based metrics.

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CHRONIC SLEEP AND CIRCADIAN DISRUPTION DIFFERENTIALLY AFFECTS BLOOD PRESSURE, RENAL SODIUM RETENTION, AND ALDOSTERONE SECRETION

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Introduction: Chronic sleep restriction (CSR) and recurrent circadian disruption (RCD; e.g., rotating shiftwork) can increase an individual's risk of cardiovascular and kidney disease. However, no study has assessed whether CSR and RCD together increase blood pressure (BP) and alter renal function (RF). We tested the hypotheses that the combination of CSR and RCD would increase blood pressure, renal sodium retention, and aldosterone secretion in individuals living for 3 weeks on an imposed non-24-h sleep-wake (SW) schedule (induces RCD) and controlled diet with or without CSR.

Methods: Seventeen (9M) healthy participants (aged 26.1±4.5y [mean±SD]) were scheduled to twenty-four 20-h Forced Desynchrony days and were randomized to either Control (1:2 sleep:wake, 6.67h sleep:13.33h wake; n=8) or CSR (1:3.3 sleep:wake, 4.67h sleep: 5.33h wake; n=9) SW conditions during a 32-day inpatient protocol. BP was measured following ~80–90 min in constant seated posture after scheduled waketime. All urine voids were collected, combined