

significantly alter phase. Hence, researchers could conduct a baseline phase assessment one week before an experimental manipulation to let subjects recover from the sleep deprivation of the phase assessment. Our results suggest that even if subjects sleep an hour later on weekends and nap 12 hours from the midpoint of their nocturnal sleep, their circadian phase will not change as long as they receive morning bright light and continue to follow a fixed weekday sleep schedule.

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0239.E

Alignment of Circadian Rhythms to Daytime Sleep Improves Performance and Alertness in Night Shift Workers

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Introduction: Impaired performance, reduced alertness, and fatigue are common complaints of night shift workers. These problems occur because their circadian clock is not aligned to a night work and daytime sleep schedule. In this study, bright light during the night shift, melatonin before daytime sleep, a regular dark/sleep opportunity, and sunglasses during the commute home were tested to determine their ability to delay the circadian clock. The phase delays produced by various combinations of these interventions are reported in an accompanying abstract (Crowley et al). Here, we report performance and alertness during the night shifts and daytime sleep duration.

Methods: During each of 5 consecutive simulated night shifts (23:00 to 07:00), subjects completed the Neurobehavioral Assessment Battery. Following each night shift, subjects had to stay in bed, in the dark, for a full seven hours (08:30 to 15:30). Subjects recorded bedtimes, sleep onset, and wake times on daily sleep logs (verified by wrist actigraphy). After the 5 night shifts, final circadian phase was assessed using salivary dim light melatonin onset (DLMO). The temperature minimum (Tmin) was estimated by adding 7 hours to the DLMO.

Results: Subjects were divided into 3 groups: those whose final DLMO was <1:30 (n=10), i.e. the Tmin did not occur during the time of the previous daytime dark/sleep period ("non-shifters"); those whose final DLMO was >1:30 and <5:00 (n=16), i.e. the Tmin occurred in the first half of dark/sleep (partial re-entrainment); and those whose final DLMO was >5:00 (n=41), i.e. the Tmin occurred in the second half of dark/sleep (complete re-entrainment). There was no difference among the 3 groups in daytime sleep duration (mean \pm SD): 6.5 ± 0.6 , 6.6 ± 0.3 , and 6.7 ± 0.2 h. There was no difference in performance or alertness between the group who showed partial and the group who showed complete re-entrainment. However, these two groups had significantly better performance and alertness than the non-shifters. For example, the average response time on the Psychomotor Vigilance Task during the last 3 night shifts for the non-shifters was twice as long (794 ± 857 versus 301 ± 40 and 311 ± 79 ms) with twice as many lapses (7.1 ± 3.7 versus 3.5 ± 1.6 and 3.7 ± 2.2). They were also much sleepier (Stanford Sleepiness Scale: 5.1 ± 1.3 versus 3.7 ± 1.1 and 3.6 ± 1.3).

Conclusions: Even though all 3 groups slept almost all of the allotted 7 hours, there were differences during the night shift depending on the amount of phase delay. This shows that a reasonable amount of daytime sleep is not enough to reduce sleepiness or improve performance in night shift workers; some circadian alignment is needed. Furthermore, in young adults, complete re-entrainment is not necessary; partial re-entrainment is enough to increase performance and alertness during the night shift.

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0240.E

To Eastward Jet Lag Sufferers: Not So Fast!

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Introduction: Our previous study (Burgess et al, Sleep. 25: A182, 2002) tested a method to advance circadian rhythms during the 3 days before flight to reduce jet lag after eastward travel. The sleep schedule of the subjects was advanced by 1 h/day and they were exposed to one of three light conditions for 3.5 h upon waking (continuous bright light > 3000 lux, intermittent bright light: 4 pulses, 0.5 h on, 0.5 off, etc, and dim room light < 60 lux). The phase advances (mean \pm SD) produced by the 3 treatments were 2.1 ± 0.5 h, 1.5 ± 0.8 h, and 0.6 ± 0.3 h respectively. There was no significant difference between the phase shifts produced by the continuous and intermittent treatments, and both were significantly greater than the dim light treatment. Since intermittent bright light is more convenient than continuous, the present study aimed to advance the circadian system further using the same intermittent bright light pattern, but coupled with a 2 h/day sleep schedule advance.

Methods: Healthy young adults received morning intermittent bright light with 2 h/day sleep schedule advances (n = 15, ages 22-35) and were compared to the intermittent bright light 1 h/day subjects run previously (n = 11, ages 22-36). Subjects sat at a desk facing one light box. Before and after the 3-day treatments, saliva samples were collected every 30 minutes in very dim light (< 10 lux) to determine the dim light melatonin onset (DLMO).

Results: There was no statistically significant difference between the phase advances produced by the two treatments (1.8 ± 0.5 h for 2 h/day and 1.5 ± 0.8 h for 1 h/day). We estimated the time of the temperature minima (Tmin) by adding 7 h to the DLMOs. There was no difference between groups in the interval from the baseline Tmin to the start of the bright light on the first treatment day (2.0 ± 1.2 h for 1 h/day and 1.7 ± 1.2 h for 2 h/day). The first light exposure started after the Tmin, i.e. on the advance portion of the PRC. On the third treatment day, the light started 1.5 ± 1.1 h after the final Tmin in the 1 h/day group, but 0.5 ± 1.5 h BEFORE the final Tmin in the 2 h/day group.

Conclusions: Increasing the amount of sleep schedule advance from 1 h/day to 2 h/day did not produce a significantly greater phase advance. Many of the 2 h/day subjects probably received some delaying bright light, especially on the third treatment day, possibly reducing their net phase advance. Thus, the 2 h/day sleep schedule advance was "too fast" for the circadian system in some people, even with morning bright light. More slowly advancing schedules (e.g. 1.5 h/day) and adding more days of treatment should be tested.

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Light boxes donated by Enviro-Med, Vancouver, WA

0241.E

The Role of Cholinergic Projections from the Brainstem and Basal Forebrain to the Suprachiasmatic Nucleus: Implications for a Feedback Loop Between the Sleep-Wake and Circadian Systems

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Introduction: Mammalian circadian rhythms are regulated by the suprachiasmatic nucleus (SCN), a set of paired nuclei located in the hypothalamus, directly above the optic chiasm. The circadian clock can be reset by a variety of stimuli in a time-dependent manner. Carbachol, a cholinergic agonist, is capable of advancing the circadian clock of both rats and mice when applied during the night. Cholinergic projections to the SCN arise from both the basal forebrain and brainstem. To understand the behavioral context of cholinergic regulation, we are studying the effects of cholinergic stimulation in vivo on acetylcholine (ACh)

This 2003 Annual Meeting abstract supplement contains all of the abstracts to be presented at the APSS 17th Annual Meeting on June 3-8 in Chicago, Illinois. This year we experienced the largest increase in submitted abstracts that has ever occurred, with 1148 submissions. This represents a 51% increase over that submitted in 2002. This likely reflects the combined meeting with the World Federation of Sleep Research Societies, the 50th anniversary of the discovery of REM sleep being celebrated at the meeting, and the sustained increase in science addressing sleep and its disorders. Thus, more science will be presented in Chicago this year than has been presented at any previous meeting addressing this topic.

Of the 1148 abstracts, 169 will be presented in oral format, 51 in poster symposiums, and the remainder as posters. With this number of abstracts being presented as posters, the Program Committee had to make some hard decisions as to how best to handle this. They elected to:

- 1) Thematically group the posters.
- 2) Posters will be up for only one day during the meeting, but will be up the entire day.
- 3) Most importantly, there will be a 90 minute block of time each day (Thursday, Friday, and Saturday, June 5, 6, and 7) between 1:30pm and 3:15pm that poster viewing will be unopposed by any other scientific presentation.

Each poster will have a unique 4 digit number within the appropriate category (listed below) which should allow for easy identification.

The categories for this year's science have not changed from last year and are listed here:

- A. Basic Neuroscience
- B. General Physiology
- C. Clinical Pharmacology
- D. Dreams
- E. Circadian Rhythms
- F. Phylogeny
- G. Pediatrics
- H. Aging
- I. Sleep Deprivation
- J. Sleep Disorders - Breathing
- K. Sleep Disorders - Narcolepsy
- L. Sleep Disorders - Insomnia
- M. Sleep Disorders - Parasomnias
- N. Sleep Disorders - Movement Disorders
- O. Sleep Disorders - Neurologic Disorders

- P. Sleep in Medical Disorders
- Q. Sleep in Psychiatric Disorders
- R. Instrumentation & Methodology
- S. Sleep Education
- T. Molecular Biology & Genetics
- U. Sleep & Behavior

Despite the troubled times in America and abroad, we expect this will be the largest APSS meeting to date. It is our hope that this year's Annual Meeting will provide the opportunity for the meaningful exchange of both clinical and basic science related to sleep and its disorders.

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