(08:30 to 15:30). Bedroom windows were darkened with black plastic. While traveling home, subjects wore sunglasses with normal or dark lenses (transmitted 15% or 2% of light respectively). Subjects took a placebo or melatonin (1.8 mg sustained release) pill before daytime sleep. During the night shifts, subjects were exposed to a moving pattern of intermittent bright light (~5000 lux, 20 min on, 40 minutes off, 4-5 light pulse/night) or remained in dim light (~200 lux). There were 6 intervention groups: 1) Dark/Sleep + Normal Sunglasses; 2) Dark/Sleep + Dark Sunglasses; 3) Dark/Sleep + Dark Sunglasses + Melatonin; 4) Dark/Sleep + Normal Sunglasses + Bright Light; 5) Dark/Sleep + Dark Sunglasses + Bright Light; 6) Dark/Sleep + Dark Sunglasses + Melatonin + Bright Light. There was a circadian phase assessment before (baseline) and after (final) the night shifts to determine the salivary dim light melatonin onset (DLMO). Sleep and night shift performance data are reported in an accompanying abstract (Lee et al).

Results: The final DLMO (mean clock time (SD in h)) in groups 1 through 6 was 2:29 (4.0), 4:19 (3.0), 4:42 (1.5), 6:08 (3.1), 6:57 (0.5), and 7:31 (0.6), respectively. Thus, the three bright light groups achieved complete re-entrainment to the daytime sleep schedule (DLMOs were 1-2.5 h before bedtime at 8:30). There was no difference among the three bright light groups and their DLMOs were significantly later than those of group 1. The difference between groups 1 and 2 (which differed only in type of sunglasses) did not reach statistical significance, showing that very dark sunglasses added little to the phase delay. There was no significant difference between groups 2 and 3 (which differed only in that one took melatonin and one took placebo), showing that melatonin does not increase the phase delay given very dark sunglasses.

Conclusions: Given appropriately timed bright light during the night shift and a regular daytime dark period, neither very dark sunglasses nor melatonin had any added benefit. The minimum combination of interventions that produced complete re-entrainment to the daytime sleep and night work schedule was bright light during the night shift, normal sunglasses during the commute home and a regular dark episode for daytime sleep.

Research supported by NIOSH grant R01 OH03954; light boxes donated by Apollo Light Systems, Orem, UT; melatonin donated by Ecological Formulas, Concord, CA

0237.E

Apparent Mechanism of the Afternoon "Nap Zone"

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Introduction: An afternoon period of transitory increase in sleep propensity is well documented for sleep latency; napping/siesta patterns, sleep in temporal isolation, and day sleep/sleepiness in medical sleep disorders. It is maximum about 180° out of phase from the mid-point of the major sleep period. We tested the hypothesis (Broughton, 1999; Webb, 1999)) that the phenomenon represents the period after morning awakening when accumulating process-S has increased to a sufficiently high level to facilitate sleep onset, while a more powerful SCN-dependent circadian arousal process has not yet risen high enough to reverse it. This was done by delaying and advancing the circadian arousal process using evening and morning bright light therapy and recording sensitive waking measures of sleep propensity (simple RT; motivation-free EEG spectral power, SP).

Methods: Eight normal male subjects, aged 20-30 years, balanced for morningness/eveningness (Horne-Osberg) were studied over two continuous 4-5 day periods under low ambient light (150 lux). Night sleep was restricted to 2300-0600h. 24-hr PSG included EEG (C3-A2, O2-A1), rightEOG-M1, leftEOG-M2 and submental EMG using 8-channel Oxford Medilog 9000 ambulatory recorders with concurrent q1min core body temperature recording using rectal probe and a Minilogger Series 2000 system. Each condition was preceded by a 24-hour baseline recording followed by two consecutive days of bright light stimulation (10,000 lux) either in the evening (2000-2200h) or morning (0600-0800h) in a cross-over design (half of Ss first had the evening and half the morning light treatment; then reversed). Daytime snacks were taken q1hr. No time cues were allowed. There was a 30 day re-adaptation period of regular sleep hours (actograph) between treatments. During wakefulness (other than during periods of bright light treatment) the level of waking arousal was assessed hourly by a 5 min sampling of artefact-free EEG for quantified SP analysis followed by a 10 min simple reaction time test for performance (P) vigilance level. QEEG-SP measures were averaged hourly. Stats were by ANOVA.

Results: Baseline prior to evening bright light treatment Ss had a Tmin at 0340h, Pmin (longest RT, most gaps) at 1346h and SPmax (all frequencies; theta) at 1400h. Evening light delayed these by about 1.5h to Tmin at 1540h (p .001), Pmin at 1452h and SPmax at 1600h (p .001). Baseline prior to morning bright light had the Tmin at 0348h, Pmin at 1452h, SPmax at 1500h. Morning bright light phase advanced the measures by about 1hr with Tmin at 0255h (p .01), Pmin at 1352 (.001), and SP max at 1600h (.001).

Conclusions: The phase delay of minimum daytime arousal measures by evening bright light treatment and their phase advance by morning bright light is consistent with the hypothesis.

Research supported by Canadian Institutes for Health Research

0238.E

Weekday DLMOs are Stable in Subjects Who Sleep One Hour Later on Weekends

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Introduction: The time of the dim light melatonin onset (DLMO), the offset (DLMOff), and the midpoint between the two are commonly used markers of circadian phase. This study examined the stability of these markers in subjects who maintained a fixed sleep schedule during the week but had later bed and wake times on weekends. We also wanted to see whether the process of collecting the melatonin profile (keeping subjects awake overnight in dim light followed by a midday nap) would itself shift circadian phase.

Methods: Healthy young adults (5 males, 6 females; 26.8±5.7 yrs) were required to be in bed with lights out from 23:00 to 7:00 on weekdays (regardless of their previous schedule) for 23 days. On weekends they were allowed to wake up and go to bed up to one hour later and were allowed to nap between 13:30 and 16:30 (i.e. 12 hours from nocturnal sleep). Every morning the subjects were required to go outside for a minimum of 15 minutes between 8:00 and 9:00 to receive natural light. To ensure compliance, all subjects wore wrist activity monitors, light sensors around the neck, and completed Daily Sleep Logs and Daily Light Logs. There were two overnight circadian phase assessments in dim light (<5 lux) beginning on Tuesdays (days 15 and 22) in which subjects gave saliva samples every 30 minutes. Following the first phase assessment, subjects napped from ~13:00 to 17:00. The DLMO and DLMOff threshold was 35% of the average of the three maximum levels.

Results: Analysis of the sleep log data in-between the two phase assessments showed that all subjects adhered to the weekday sleep schedule and went to bed and woke up about one hour later on weekends. Seven subjects took naps on weekends. The mean±SD time of the DLMO for the first and second phase assessment was 21:48±1.0h and 21:54±0.7h; the DLMOff was 6:30±1.1h and 6:48±1.0h. The absolute phase change (i.e., the phase shift giving both advances and delays a positive sign) was 0.5±0.2h for the DLMO, 0.7±0.6h for the DLMOff, and 0.4±0.3h for the midpoint.

Conclusions: In this study, all three phase markers remained very stable in phase assessments one week apart. The phase assessment and subsequent nap, and later weekend bed times, wake times, and naps did not

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.

Research supported by NIOSH grant R01 OH 03954

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 DMLO 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 ± SD): 6.5 ± 0.6, 6.6 ± 0.3, and 6.7 ± 0.2h. 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 ± 79ms) 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.

Research supported by NIOSH grant R01 OH03954.

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 ± 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.

Research supported by NINR RO1 NR07677
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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