quality on a regular (predictable) basis especially when the ability to sleep is compromised either by increasing age with its attendant illnesses and possible depletion of psychosocial resources, or by physical and/or mental ill-health.

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0234.E

Do We Practice What We Preach? Shiftwork and Sleepiness in the Association of Polysomnographic Technologists (APT)

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Introduction: Shiftwork is known to contribute to increased sleepiness and reduced sleep quality and quantity. This study examined the prevalence of these problems in APT members working on the frontline of sleep research and medicine. We hypothesized that these problems would persist, despite specialized knowledge and the use of countermeasures.
Methods: A randomly started, systematic survey of the APT was conducted by mail. The response rate was 62%. For preliminary analysis, night shift responses from individuals who worked ³ 13 night shifts (N-group)(n=133) were compared with day shift responses from those who worked <13 night shifts (D-group)(n=95)for the past year.
Results: Results revealed no significant differences between groups in age, gender, weekly hours worked or existing sleep disorders. Ds were more likely to work in a supervisory role (N 38.5%; D 61.5%). Ns worked longer shifts (N mean=11.68 hours, SD=1.5; D mean=9.27, SD=1.39; p<0.05). Stanford sleepiness scores, modified for typical lowest level of alertness at work, were higher for Ns (N median=4, IQR=2-5; D median=2, IQR=2-3, p<0.05). Ns reported significantly more sleepiness driving to and from work, with several admitting to falling asleep driving in the past year (N 10.0%; D 5.0%). Epworth sleepiness scores were not significantly different, with both groups having a high percentage of scores >10 (N 34.8%, D 30.6%). Ns reported fewer hours of sleep between shifts (N mean=6.6, SD=1.5; D mean=7.4, SD=0.9; p<0.05). Ns scored higher on the sleep disturbance scale of the Standard Shiftwork Inventory (N mean=15.6, SD=3.9; D mean=12.5, SD=3.3; p<0.05). Countermeasures were quantified by frequency using a 5-point scale. Combined responses of 'sometimes, frequently, and almost always' are reported. The only countermeasure that differed significantly between groups was the use of bright light for synchronization to work schedule (N 48.1%; D 15.0%). Many used caffeine (N 64.3%; D 58.6%); while few used other stimulants (N 1.5%; D 1.1%), melatonin (N 8.6%; D 7.2%) or hypnotics (N 8.8%; D 7.2%).
Conclusions: These findings support our hypotheses and replicate those of existing studies, that night shiftwork was associated with less sleep between shifts, higher sleep disturbance and increased sleepiness. Regardless of work schedule, roughly 1/3 of APT members were excessively sleepy by conventional scoring standards, and several fell asleep while driving to and from work. While many APT members utilize bright light and caffeine to cope with shiftwork, nearly all work long night shifts, which are known to cause coping problems. Excessive sleepiness and other problems associated with shiftwork appear prevalent in the APT. We propose the inclusion of education and management policies to address these problems in accreditation standards for sleep laboratories.

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0235.E

How Well Do Fixed And Free Sleep Schedules Predict The Dim Light Melatonin Onset In Young Healthy Subjects?

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Introduction: The endogenous melatonin onset in dim light (DLMO) is a commonly used marker of circadian phase, and can be used to determine the time for the administration of light or exogenous melatonin in order to elicit desired circadian phase shifts. In some settings determining the DLMO is not feasible or is prohibitively costly and time-consuming. In this study we examined how accurately the DLMO could be estimated from subjects with free or fixed sleep schedules.
Methods: We analyzed baseline data from 100 young healthy subjects who participated in various studies in our lab. Some subjects slept at times of their own choosing ("free sleepers", N=60, mean age 27.3 y) and others slept on a fixed sleep schedule that closely matched their typical sleep schedule ("fixed sleepers", N=40, mean age 24.2 y). Subjects recorded bedtimes and wake times on sleep logs (verified with wrist actigraphy recordings). Each subject then participated in a phase assessment session, where they remained awake but reclined in dim light (<10 lux). Saliva samples were taken every 30 mins and were later assayed to determine the DLMO (20% of maximum method). We analyzed the sleep log data from the 6 days before the phase assessment session.
Results: The average (± SD) bedtime and wake times for the fixed and free groups were 23:05 ± 0.8 h, 6:38 ± 0.7 h and 00:44 ± 1.2 h, 9:13 ± 1.4 h respectively, and were significantly later in the free group (both p<0.01). As expected, the within subject variance in bed and wake times was significantly higher in the free sleepers than in the fixed sleepers (p<0.05). However, there was no difference between the groups in the interval from the DLMO to average bedtime (2.3 ± 1.3 h for fixed sleepers; 2.2 ± 1.1 for free sleepers). The correlations between bedtime and the DLMO and between wake time and the DLMO were stronger in the free group than the fixed group (free: r=0.65 and r=0.78; fixed: r=0.56 and r=0.47). We derived a regression equation from the relationship between the DLMO and wake time in the free sleepers: DLMO (dec. time) = 0.83 x wake time (dec. time) + 14.90. Using this equation we were able to predict the DLMO to within 1.5 h of when it actually occurred in 96% of subjects from an independent sample of free sleepers (n=26, Martin and Eastman, Chronobiol. Int., 2002).
Conclusions: Our results indicate that when measuring circadian phase is too costly or impractical, wake times (verified with wrist actigraphy) in young healthy subjects who sleep at times of their own choosing, can be used to the predict the DLMO. Requiring subjects to maintain a fixed baseline sleep schedule does not appear to improve the ability to predict the DLMO.

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0236.E

Comparing Bright Light, Dark Sunglasses, and Melatonin to Phase Delay Circadian Rhythms for Night Shift Work

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Introduction: Melatonin and various patterns of bright light and dark have been tested to help phase shift the circadian clock and promote adjustment to night work and day sleep schedules. This study tested various combinations of these interventions to determine their ability to produce phase delays.
Methods: Healthy young adults (32 males, 35 females, 23.9 ± 6.2 yrs) participated in the summer months of 3 years. They worked 5 consecutive simulated night shifts (23:00 to 07:00) followed by sleep at home

(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.

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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.

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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

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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Editor-in-Chief