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ORIGINAL ARTICLE

Returning from night shift to day life: Beneficial effects of light on sleep

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Abstract

Subjects working a 12 h offshore night shift for 2 weeks normally adapt to the night shift and are out of synchrony when they return home to day life, with consequent problems of poor sleep. The aim of this study was to investigate the effectiveness of timed light treatment to hasten circadian adaptation and improve sleep after the night shift. Ten male shift workers worked 19.00–07.00 h ($n = 4$) or 18.00–06.00 h ($n = 6$) offshore shift schedules. They were assessed for the last 7 days of a 14 or 21 day offshore night shift and for the following 14 days at home. Either timed light treatment/sunglasses or no light treatment/no sunglasses were scheduled in a crossover design during days 1–5 after the nightshift, theoretically timed to advance the circadian system. Subjects completed the Horne Östberg questionnaire. They wore an Actiwatch-L throughout the study to monitor light/activity and completed daily sleep diaries. Actigraphic sleep efficiency after the light/sunglasses treatment was significantly improved (days 1–5), that is, 86.7 ± 5.8% (mean ± SD; light treatment) compared to 79.4 ± 10.3% (no light treatment), $P < 0.05$. Objective sleep duration (days 6–14) was significantly improved in the light treatment leg; actigraphic sleep duration was longer after light treatment (6.75 ± 0.50 h) compared to 5.76 ± 0.73 h, $P < 0.05$. If appropriately timed, light and darkness has beneficial effects on sleep efficiency and sleep duration following a night shift.

Key words: light, melatonin, shift work, sleep.

INTRODUCTION

Shift work is associated with greater health problems than "normal" day work.[1,2] Night-shift workers are required to work and eat at the "wrong" phase of their circadian cycle, resulting in complaints of sleepiness, reduced performance and disturbed sleep due to lack of adjustment of the circadian timing system.[3–5] Attempts to sleep at inappropriate phases of the circadian cycle, for example during the declining phase of melatonin and the rising phase of core body temperature, usually result in shorter sleep episodes and more awakenings.[6–8]

In general, the circadian system adapts either partially or not at all to night-shift work.[9,10] However, previous research in unusual circumstances (e.g. working 18.00–06.00 h on offshore oil rigs and 20.00–08.00 h in Antarctica) has reported that shift workers can fully adapt to the night-shift schedule within a week.[11,12] If shift workers fully adapt to an offshore night shift they will be out of synchrony with their home environment upon returning home, with consequent problems of poor night sleep, reduced daytime alertness and performance

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and possible digestive problems. Factors such as season,[13] length and timing of the shift,[12,14] early or late initial circadian phase,[15] light exposure,[9] and sleep/wake patterns (as these indirectly affect light exposure), may all affect circadian adaptation. The use of light treatment at home, appropriately timed, may alleviate/reduce the physiological and behavioral problems caused by circadian rhythm disturbance experienced following offshore night-shift work.[5,16]

Light has been suggested as a counter measure against night work impairment of sleep and alertness.[10,17] Intense artificial light can shift the phase of the human circadian timing system and has been successfully used to induce phase shifts in circadian rhythms and to improve sleep, performance, and alertness.[18–21] Bright light as a counter measure for circadian desynchrony has been used in field studies of shift workers, though the number of studies is limited.[16,22–24] Recent research has shown the effectiveness of short wavelength blue light to phase-shift human circadian rhythms[25,26] and increase alertness.[21,27,28]

Recently, Bjorvatn *et al.*[16] evaluated the effects of bright white light and melatonin to phase delay the circadian system in offshore shift workers working a "swing shift" schedule. Subjective and objective measures of sleep were obtained. Melatonin reduced sleepiness at work during the day shift and increased sleep by 15–20 min per day, whilst bright light gave values in between those of melatonin and the placebo, but with few statistically significant results. In these studies, the effect of melatonin and light on circadian phase was not investigated.

The aim of this study was to investigate the effects of timed bright light treatment on sleep and circadian adaptation in offshore night-shift workers returning to day life at home after adapting to the offshore night shift. The protocol was based on the 6-sulphatoxymelatonin (aMT6s) data from a previous study by Barnes *et al.*[11] In this study the subjects were working a 2-week 18.00–06.00 h night shift and their average aMT6s peak occurred at 12.98 h at the end of the night shift. A 5 h advance of aMT6s during subsequent day shift or home life should lead to the peak of melatonin itself (which is on average approximately 2 h before the aMT6s peak) occurring within the normal sleep period (before 07.00 h). An 11 h delay would similarly shift the peak of melatonin to within the normal sleep period (after 23.00 h). We hypothesized that adaptation to home life would be faster with a 5 h advance than an 11 h delay and that timed light treatment aimed to phase advance the circadian system would improve night sleep at home and would hasten adaptation of the circadian rhythm of urinary aMT6s compared to a "no light treatment" condition.

METHODS

Pre-study

Ethical permission was obtained from the University of Surrey Ethics Committee (ACE/2002/95SBLS). Before starting the study, written informed consent was obtained from all volunteers. Subjects had to be working a 2–3 week night shift and be free of any medication known to affect the melatonin rhythm (β-blockers, α-blockers, calcium channel blockers, antipsychotics, benzodiazepines, antidepressants, barbiturates, and antiepileptic drugs). Diurnal preference was assessed using the Horne Östberg (HÖ) questionnaire.[29]

Subjects

Eight men (S1–S8) aged 46 ± 11 years (mean ± SD), body mass index (BMI) 28.1 ± 2.5 kg/m^2, HÖ score 57 ± 8, working a 19.00–07.00 h offshore shift schedule on an oilrig platform in the North Sea at 58°N for 14 or 21 consecutive nights were recruited during the summer months (May–August 2005). Six men (S9–S14) aged 49 ± 7 years (mean ± SD), BMI 28.4 ± 2.1 kg/m^2, HÖ score 58 ± 4, working a 18.00–06.00 h offshore shift schedule in the North Sea at 59°N for 14 consecutive nights were recruited during the winter months (October–March 2006). Of these initial recruits, two did not adapt to nights and two completed only one study leg. These four subjects were excluded from further analysis. One subject collected urine on one leg only, two provided sleep diaries but did not wear an Actiwatch-L (see below). Thus, subjective sleep data derives from ten subjects, actigraphic sleep and light data from eight subjects, aMT6s data from nine subjects. All subjects returned home onshore after finishing their night shifts.

Study design

Subjects were studied for 21 days, namely the last 7 days of a 2- or 3-week 19.00–07.00 h offshore night-shift schedule or the last 7 days of a 2-week 18.00–06.00 h offshore night-shift schedule, and for 14 days at home after completion of the night shift. Subjects completed daily sleep diaries. They wore an Actiwatch-L (Cambridge Neurotechnology, Cambridge, UK) for

21 days to record light exposure and activity in 1-min epochs. The subjective sleep parameters recorded were sleep onset (defined as the time the subject went to sleep), sleep offset (defined as the time the subject woke up), sleep latency, number of night awakenings and sleep quality (1 = best ever sleep, 9 = worst ever sleep). In the actigraphy analysis, sleep onset, offset, efficiency (% time spent asleep during desired sleep time), and fragmentation index (a measure of movement during sleep) were analyzed. Light exposure (lux) derived from the Actiwatch-L was analyzed in 1-h bins on days 1–5 at home after the night shift to determine the timing of brightest daily light exposure period for each individual subject.

Subjects collected sequential urine samples to assess circadian adaptation to the night shift and readaptation after the night shift. Samples were collected approximately every 4 h during waking hours and longer over the sleep period during the last 2 days (48 h) of the offshore night shift and the 7 consecutive days at home after the night shift.

6-sulphatoxymelatonin analysis

The volume of each sample was measured and a 5 mL aliquot was frozen and transported to the University of Surrey for analysis. 6-sulphatoxymelatonin was measured by radioimmunoassay,[30] with reagents provided by Stockgrand Ltd, UK. Interassay coefficients of variation at 4.7 ng/mL (n = 14), 14.9 ng/mL (n = 19), and 25.0 ng/mL (n = 15) were 10.0%, 9.9% and 9.4%, respectively. The aMT6s acrophase (individual daily mean peak time for the last 3 days of night shift, and the 7 consecutive days afterwards) was calculated from cosinor analysis of each 24 h period (programme developed and kindly provided by Dr DS Minors, University of Manchester, UK). Acrophase values were only accepted if the cosinor fit was significant at the 95% level or if the fit was significant at >80% level, and the variance (percentage rhythm) accounted for by the cosine curve was greater than 50%. Circadian adaptation offshore was defined as when the aMT6s acrophase occurred during the day sleep period. Acrophase time is given in decimal hours.

Light treatment protocol

The study was a randomized crossover design with two study sessions. One session where the subject received timed light treatment and wore sunglasses at appropriate times upon returning home, and a second session where the sample collection protocol was the same but light treatment was not given and sunglasses were not worn. White polychromatic light (~3000 lux, 1000 μW/cm^2) was administered using a light box (Litebook, Alberta, Canada). Previous studies by Barnes *et al.*[11] showed that the peak of the aMT6s rhythm of subjects leaving an oil rig after working 2 weeks of night shifts (18.00–06.00 h) was 12.98 ± 0.50 h. From the findings of this previous study, the light treatment protocol was designed as follows. Day 1 was the day the subjects returned home onshore, and they were asked to wear specialized light blocking sunglasses (Litebook) from the end of their night shift until 13.00 h on day 1. On day 2 subjects wore sunglasses from wake up until 13.00 h and then received light treatment by sitting in front of the light box for 1 h, with the device placed at a 45 degree angle, 30 cm in distance away from their eyes. For the following 3 days (days 3–5) the light was scheduled an hour earlier each day with subjects wearing sunglasses from wake up until the beginning of the light treatment. This protocol was used for those subjects working the 19.00–07.00 h night shift (n = 8). Due to the observed variability of results from the 19.00–07.00 h study, it was decided for the subsequent study for those working a 18.00–06.00 h shift schedule that the light treatment should be individually timed to phase advance the circadian system. This part of the study was thus not randomized as subjects completed the "no light treatment" leg first to allow the offshore aMT6s rhythm to be analyzed, and then in the second study leg the light treatment was individually timed on the basis of their aMT6s results. Subjects whose offshore aMT6s acrophase was between 11.00 and 15.00 h followed the protocol as described above (n = 7). One subject had an offshore aMT6s acrophase between 15.00 and 17.00 h and thus started his light treatment on day 2 at 14.00 h.

Rate and direction of adaptation

Adaptation of the aMT6s rhythm during offshore night shift was considered to be when the acrophase occurred during the daytime sleep period. The rate of adaptation back to home time between the light and no light treatment legs was calculated using the following equation: (aMT6s acrophase on day 5 – mean aMT6s night-shift acrophase for the last 2 days [days −2 and −1] offshore)/number of days (=5). Subjects were then categorized with regard to the direction in which adaptation of the aMT6s rhythm occurred (sequentially earlier, phase

advanced, or sequentially later, phase delayed acrophases) over the 5 days (days 1–5).

Statistical analysis

In the 19.00–07.00 h shift schedule, two subjects (S1 and S2) did not adapt to the night shift, with their mean (±SD) aMT6s acrophases for the last 2 days offshore were 4.3 ± 0.4 h and 5.3 ± 0.5 h for S1 and S2, respectively. As a result these two subjects were excluded from any further analysis. Two subjects (S3 and S7) only completed one leg of the study and were also excluded from further analysis. Two subjects (S4 and S14) completed sleep diaries and these results are included in the analysis but no actigraphy data were obtained and therefore no light exposure analysis could be performed. One subject (S9) collected urine for only one study session; these data were therefore not included in the circadian phase analysis. In total ten subjects were included in the sleep analysis: mean age 46.5 ± 7.8 years, BMI 28. ± 2.0 kg/m^2, and HÖ score 55 ± 5.

Repeated measures ANOVA (PROC MIXED; SAS Version 9.1) with condition and day as factors was used to compare the light and no light treatment legs. Statistical significance was set at $P < 0.05$. All values are mean ± SD.

RESULTS

Age, BMI, and HÖ score

No significant differences were observed in the subjects working the two shift schedules (18.00–06.00 h and 19.00–07.00 h) in terms of age, BMI,and HÖ score.

Prior sleep history

Sleep (sleep diaries and actigraphy) prior to the light treatment schedule was assessed for 7 days offshore (days −7 to −1), as shown in Table 1. Sleep diary sleep duration was 6.79 ± 0.90 h, whilst actigraphic sleep duration was 5.89 ± 0.65 h.

Circadian phase

No significant differences ($P > 0.05$) were observed in the mean aMT6s acrophase during the last 2 days of their offshore night shift between the no light treatment (14.3 ± 2.5 h) and light treatment leg (15.0 ± 1.5 h; paired Student's *t*-test). No significant differences were observed in the rate of aMT6s adaptation between the light and no light treatment legs for those subjects who had data for both study sessions ($n = 9$). The mean rate of aMT6s adaptation in the no light treatment leg was 2.00 ± 0.45 h/day (mean ± SD) and in the light treatment leg the mean rate of aMT6s adaptation was 2.16 ± 0.86 h/day. In the light treatment session, two subjects adapted by phase advance and seven phase delayed, whereas in the no light treatment session eight subjects adapted by phase delay and one subject phase advanced (Table 2).

Table 1 Sleep diary ($n = 10$) and actigraphy ($n = 8$) data (mean ± SD) for the last 7 days of the first leg of the subjects' night shift, before returning home to day life

Sleep parameter	Sleep diary	Actigraphy
Sleep onset (dec. h)	8.72 ± 1.03	9.06 ± 1.11
Sleep offset (dec. h)	15.93 ± 1.22	15.89 ± 1.29
Sleep duration (dec. h)	6.79 ± 0.90	5.89 ± 0.65
Sleep latency (dec. h)	0.16 ± 0.10	0.22 ± 0.22
Sleep efficiency (%)	N/A	82.7 ± 6.3
No. of night awakenings/day	1.4 ± 0.7	N/A
Duration of night awakenings (dec. h)	0.19 ± 0.13	N/A
Sleep quality	4.5 ± 1.0	N/A

N/A, not applicable; dec., decimal.

Light exposure

No significant differences were observed in the mean timing of the brightest light exposure ($P > 0.05$) between the study legs (days 1–5 on returning onshore; Table 2). In the light treatment leg the brightest light exposure period occurred between 10.0 and 14.0 h (mean 12.9 ± 3.2 h) for all of the subjects and in the no light treatment leg the brightest light exposure period occurred between 11.0 and 16.6 h (mean 13.3 ± 2.7 h) for all of the subjects (Table 2). However significant differences were observed between the light treatment and no light treatment leg ($P = 0.05$) in terms of the amount of bright light exposure that occurred during this time period. In the no light treatment leg, mean light exposure was 2252 ± 1631 lux, whilst in the light treatment leg mean light exposure was increased to 4644 ± 2602 lux. Light exposure, however, was very variable between subjects, with two subjects (S10 and S13) showing little evidence of extra light exposure during the light treatment leg.

Table 2 Time and maximum light exposure on days 1–5 onshore (mean ± SD) and aMT6s acrophase (day 1) for the light and no light treatment legs

	Light treatment					No light treatment	
Subject*	Time of brightest light exposure (dec.h)	Light exposure (lux)	aMT6s acrophase Day 1 (dec.h)	Advance or delay	Time of brightest light exposure (dec.h)	Light exposure (lux)	aMT6s acrophase Day 1 (dec.h)	Advance or delay
S4	14.0 ± 2.5	5701 ± 5692	17.9	Delay	12.8 ± 2.3	4644 ± 4046	N/A	Delay
S5	14.0 ± 2.6	5701 ± 5693	17.2	Delay	13.0 ± 2.2	5771 ± 3875	10.9	Delay
S6	13.4 ± 2.5	10329 ± 3527	N/A	Delay	16.6 ± 2.5	3091 ± 1376	13.7	Advance
S8	13.5 ± 2.4	4560 ± 3284	19.4	Advance	12.3 ± 2.2	215 ± 132	15.1	Delay
S9	12.6 ± 3.0	3783 ± 2354	18.3	Delay	13.5 ± 2.4	678 ± 650	N/A	Delay
S10	10.0 ± 6.2	270 ± 112	N/A	Delay	13.6 ± 4.9	263 ± 101	19.9	Delay
S11	12.3 ± 1.5	6558 ± 3112	18.1	Advance	11.0 ± 2.6	1105 ± 1237	22.9	Delay
S13	13.0 ± 5.1	253 ± 40	N/A	Delay	N/A	N/A	13.1	Delay
Mean ± SD	12.9 ± 3.2	4644 ± 2602	18.2. ± 0.8		13.3 ± 1.7	2252 ± 1631	13.3 ± 2.7	

*All subjects received light treatment on day 1 at 13.00–14.00 h except S11 who received light treatment on day 1 at 14.00–15.00 h. N/A, not available; dec., decimal.

Analysis of the participants' light exposure revealed that the natural bright light exposure was greater (6105 ± 3444 lux) than the light treatment itself (3000 lux) in six out of eight subjects. In the light treatment leg, on day 1 after the night shift all subjects (n = 5) with an available aMT6s acrophase received the brightest natural light exposure before their peak of melatonin. In the no light treatment leg, three subjects received their brightest natural light exposure before the peak of melatonin on day 1, and two subjects received their brightest natural light exposure after the peak of melatonin (three subjects had an unknown acrophase on day 1; Table 2).

Sleep

Sleep analysis after the night shift was separated into days 1–5 (during the light exposure treatment) and days 6–14, which were chosen as the main descriptive analysis for all sleep diary and actigraphy data. This allowed a direct comparison between the light and no light treatment legs as days 2–5 could have been affected directly by the light treatment.

Actigraphic sleep

Individual actigraphy data for the light and no light treatment legs for days 1–5 and days 6–14 are shown in Table 3a,b, respectively.

During days 1–5 there was a significant increase (P = 0.04) in the sleep efficiency in the light treatment session (86.7 ± 5.8%) compared to the no light treatment session (79.4 ± 10.3%). No other statistical differences in actigraphic sleep were observed between the two study conditions. During days 6–14 the following differences were observed: sleep onset was significantly earlier in the light treatment condition (23.67 ± 0.49 h) compared to the no light treatment condition (24.50 ± 0.92 h; P = 0.04); and mean actigraphic sleep duration was significantly longer (6.75 ± 0.50 h) in the light treatment condition compared to 5.76 ± 0.73 h in the no light condition (P = 0.01). No other statistical differences were observed between the two study conditions.

Subjective sleep (sleep diaries)

Individual sleep parameters (mean ± SD, n = 10) derived from the daily sleep diaries for days 1–5 and days 6–14 are shown in Table 4a,b, respectively.

During days 1–5 no significant differences in subjective sleep were observed between the light and no light conditions. During days 6–14, however, there was a

Table 3 Actigraphic sleep parameters for days 1–5 and days 6–14 onshore (mean ± SD, $n = 8$) for the no light treatment and light treatment legs

Sleep parameter	No light treatment	Light treatment	*P*-value
a) Days 1–5			
Sleep onset (dec.h)	23.45 ± 1.26	22.85 ± 0.94	0.90
Sleep offset (dec. h)	6.19 ± 1.86	5.78 ± 1.36	0.80
Sleep duration (dec. h)	5.95 ± 0.75	6.18 ± 1.06	0.60
Sleep efficiency (%)	79.4 ± 10.3	86.7 ± 5.8	0.04**
Fragmentation index	30.7 ± 8.8	27.2 ± 8.2	0.37
Sleep latency (dec. h)	0.38 ± 0.39	0.21 ± 0.17	0.25
b) Days 1–14			
Sleep onset (dec.h)	24.50 ± 0.92	23.67 ± 0.49	0.04**
Sleep offset (dec. h)	6.93 ± 0.64	6.86 ± 0.99	0.23
Sleep duration (dec. h)	5.76 ± 0.73	6.75 ± 0.50	0.01**
Sleep efficiency (%)	80.6 ± 6.6	84.3 ± 6.3	0.40
Fragmentation index	36.7 ± 10.7	34.0 ± 10.3	0.40
Sleep latency (dec. h)	0.34 ± 0.19	0.26 ± 0.19	0.52

**$P < 0.05$. dec., decimal.

Table 4 Subjective sleep parameters for days 1–5 and 6–14 onshore (mean ± SD, $n = 10$) for the no light treatment and light treatment legs

Sleep parameter	No light treatment	Light treatment	*P* value
a) Days 1–5			
Sleep onset (dec. h)	22.91 ± 0.76	22.92 ± 1.47	0.49
Sleep offset (dec. h)	6.66 ± 1.64	6.51 ± 1.22	0.61
Sleep duration (dec. h)	7.26 ± 1.50	6.99 ± 0.96	0.27
Sleep latency (dec. h)	0.13 ± 0.10	0.14 ± 0.11	0.87
No. of night awakenings/day	1.5 ± 0.9	1.3 ± 0.8	0.27
Duration of night awakenings (dec. h)	0.32 ± 0.27	0.31 ± 0.38	0.88
Sleep quality	5.1 ± 1.2	4.6 ± 1.2	0.15
b) Days 6–14			
Sleep onset (dec. h)	23.45 ± 0.55	23.15 ± 0.41	0.58
Sleep offset (dec. h)	7.12 ± 0.72	7.43 ± 1.19	0.45
Sleep duration (dec. h)	7.32 ± 0.70	8.05 ± 0.87	0.55
Sleep latency (dec. h)	0.16 ± 0.10	0.14 ± 0.09	0.85
No. of night awakenings/day	0.60 ± 0.69	0.25 ± 0.31	0.11
Duration of night awakenings (dec. h)	0.19 ± 0.18	0.09 ± 0.06	0.12
Sleep quality	4.1 ± 1.1	4.6 ± 1.6	0.03**

**$P < 0.05$. dec., decimal.

significant decrease in sleep quality (4.6 ± 1.6 h) in the light treatment condition compared to 4.1 ± 1.1 h in the no light treatment condition ($P = 0.03$). No statistical differences were observed in the other parameters analyzed (sleep onset, sleep offset, sleep duration, sleep latency, number and duration of night awakenings) between the light and no light treatment legs for days 6–14 after the night shift.

DISCUSSION

The results of this study are that light treatment has varying effects on the sleep of offshore shift workers. During the first 5 days after the night shift, significant differences were only observed in actigraphic sleep efficiency, which was substantially improved by light treatment (79% compared to 87%). During days 6–14 (after

the light exposure period) measurements of actigraphic sleep showed there was a significant improvement in sleep duration, though unexpectedly a decrease in sleep quality in the light treatment leg compared to the no light treatment leg. Similar results with regards to sleep duration have been reported by Dawson and Campbell,[31] who investigated the use of timed light treatment for those working night shifts. Workers who were exposed to a 4 h light pulse averaged 62 min more sleep measured by polysomnography than the control group who received no light. Other studies have reported different findings. Ross *et al*.[24] assessed circadian phase (aMT6s) tested light treatment in a field study in subjects who were working night shifts in Antarctica. They reported improvement in sleep latency with timed bright light treatment. Our study, along with Ross *et al*.,[24] attempted to investigate, in the field, if these beneficial findings on sleep were related to circadian phase, which this study failed to demonstrate.

Lastly Bjorvatn *et al*.[5] investigated offshore shift workers and reported the effects of 30 min of bright light treatment (~10 000 lux) given for 4 days upon returning home after completion of the 2 week night shift. Light exposure was scheduled individually to phase delay the circadian system, based on the assumption that the circadian nadir was located 2 h before the subject's habitual time of awakening. Light treatment reduced self-rated sleepiness, though in this study circadian phase was not assessed.

The shift workers studied here slept on average less than a cohort studied by Vorona *et al*.[32] Vorona and colleagues reported that men (n = 288) aged 18–91 years with a BMI of 25–29 kg/m^{2,} assessed with a sleep questionnaire, slept on average 7.57 h. In the no light treatment leg, subjects' subjective sleep duration was on average (days 6–14) slightly less (7.32 ± 0.55 h) than that studied by Vorona *et al*.[32]

This study illustrates the complexity of readaptation to day life after working a 2 or 3 week night shift. Light treatment in our study was conducted onshore in subjects own homes. This method of delivering the light exposure has "less control" than, for example, the method used by Bjorvatn *et al*.[16] where the light treatment was administered in a controlled environment onshore. In addition, in the Bjorvatn *et al*.[16] study, light was administered to phase delay the circadian system, whilst in our study it was administered to phase advance the circadian system. In theory, to phase advance should have been quicker in accordance with a predicted 13.00 h aMT6s phi from a previous study investigating the timing of offshore aMT6s (Barnes *et al*.[11]). Unfortunately, the phi of the offshore shift workers studied in this population was very variable and in some cases much later than expected (e.g. subjects S11 and S9) leading to possible bright natural light exposure before the melatonin peak at a phase-delay time of the phase-response curve. If the subjects wore sunglasses as instructed this should have counteracted any delaying effects of natural light, unless the melatonin phase was so delayed that exposure to natural light occurred after removing glasses and before the melatonin peak. The limited evidence suggests that, in fact, most subjects in both study legs adapted by phase delay to home time. To force a phase advance in these circumstances, the avoidance of conflicting light (e.g. by wearing sunglasses) is likely to be more important than the light treatment itself.

The results from this study indicate that it is difficult to estimate the timing of an individual's aMT6s rhythm as it is individually variable, dependent on shift timing, and also season may have an influence. With hindsight, in very delayed subjects, the use of light treatment to reinforce phase delays may be more effective in hastening readaptation back to home life. The use of melatonin, correctly timed, should also be considered.

In our study, the improvements in sleep duration during and after light treatment could not be attributed to different rates of circadian adaptation as there were no observed differences in the rate of adaptation between the light treatment and no light treatment legs. This may be due to a number of factors. For example, compliance with the urine sample collection protocol was variable. Second, natural light exposure during the day could have suppressed melatonin production, and therefore calculation of the timing of the aMT6s acrophase would have been affected. In addition, as previously discussed, the scheduled timing of light treatment may not have been optimal to phase advance the circadian system in some cases.

The findings of this study thus suggest that the effects of light on sleep may be mediated by other factors in addition to circadian adaptation. Light can potentially affect sleep in several other ways, for example, increased circadian amplitude, increased sleep duration with a change in photoperiod, increased daytime activity and consequently better night sleep.[33–36] Recent publications[37,38] demonstrate that light acting via melanopsin can directly affect sleep. The Litebox used in this study delivers white light in the visible range (400–480 nm) with a peak at 460–480 nm (blue wavelength), and the maximal sensitivity of the melanopsin photopigment is known to be in the blue range.[39,40]

Riemersma-van der Lek *et al*.[33] investigated the effects of bright light and melatonin on cognitive and non-cognitive function in elderly residents. Their findings provide evidence for actions of light which are not necessarily related to circadian timing but may influence other circadian parameters such as amplitude and hence sleep characteristics.

Subject motivation may also be very important given that it is virtually impossible to blind such light experiments. The subjects recruited in this field study were motivated to try out the light treatment hoping that it would reduce their complaints of feeling "jet-lagged" upon returning home from night shift. This may have provoked them to provide more positive subjective sleep scores following the bright light treatment.

As with any field study, certain conditions such as natural light exposure or subject compliance with study instructions cannot be completely controlled. There is no control of the seasonal and daily changes in the outdoor levels of light; though participating subjects completed both study legs in the same season, either summer or winter. Subjects were asked when participating in the light treatment leg to wear the sunglasses provided at certain times; however, there was no way of checking that this had actually occurred. In addition, clothing covering the light monitor cannot be strictly controlled although subjects were instructed to wear the activity/light monitor on the outside of the clothing over their sleeves, and to wear it continuously apart from when bathing. Despite these limitations, light treatment significantly improved some sleep parameters.

It is possible either to phase advance the timing of the circadian system, if light exposure is timed on the declining phase of the melatonin rhythm, or to phase delay the circadian system, if light exposure is timed on the rising phase of the melatonin rhythm.[41–43] Thus if bright light exposure occurs at the "wrong" time, this may lengthen the time in which it takes to readapt back to day life, though if subjects are very phase delayed, which was the case for those working the 19.00–07.00 h shift schedule, it may be better to phase delay the melatonin rhythm rather than impose phase advances.

There was a clearly beneficial effect of light treatment on some sleep parameters, however, due to the study design using both light and sunglasses it cannot be determined if it was the light treatment, the avoidance of light, or a combination of the two which was responsible for the observed effect. This ambiguity could only be resolved if two additional study legs were carried out: subjects wearing sunglasses only and subjects only being exposed to light. A crossover design with four treatment conditions would have made recruitment difficult and would not have been possible within the same season due to the long length of each night shift.

The underlying cause of many of the adverse health effects that are associated with working night shifts is thought to be desynchronization of the circadian system with detrimental effects on sleep. Reduction in the length of sleep by between 1 and 4 h has been reported to lead to increased subjective sleepiness and performance deficits.[44,45] Therefore, the poorer sleep experienced in the no light treatment leg could have had a detrimental effect on the daytime function of this shift work population.

This study has demonstrated that timed light treatment administered to hasten adaptation to day life after working night shift significantly improved both actigraphic sleep duration and sleep efficiency. However, we could not show that this effect was due to faster circadian resynchronization.

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