Photochemistry and Photobiology, 20**, **: *–*

Light Treatment Improves Sleep Quality and Negative Affectiveness in High Arctic Residents During Winter

Michel A. Paul[1], Ryan J. Love*[1], Andrea Hawton[1], Kaighley Brett[2,3], Donald R. McCreary[1] and Josephine Arendt[4]

[1]Defence Research and Development Canada, Toronto, ON, Canada
[2]Canadian Forces Health Services Centre (Atlantic), Halifax, NS
[3]Canadian Forces Station, Alert, NU, Canada
[4]University of Surrey, Guildford, Surrey, UK

Received 6 August 2014, accepted 3 January 2015, DOI: 10.1111/php.12418

ABSTRACT

The seasonal extremes of photoperiod in the high Arctic place particular strain on the human circadian system, which leads to trouble sleeping and increased feelings of negative affect in the winter months. To qualify for our study, potential participants had to have been at Canadian Forces Station (CFS) Alert (82° 30′ 00″ N) for at least 2 weeks. Subjects filled out questionnaires regarding sleep difficulty, psychological well-being and mood and wore Actigraphs to obtain objective sleep data. Saliva was collected at regular intervals on two occasions, 2 weeks apart, to measure melatonin and assess melatonin onset. Individuals with a melatonin rhythm that was in disaccord with their sleep schedule were given individualized daily light treatment interventions based on their pretreatment salivary melatonin profile. The light treatment prescribed to seven of the twelve subjects was effective in improving sleep quality both subjectively, based on questionnaire results, and objectively, based on the actigraphic data. The treatment also caused a significant reduction in negative affect among the participants. Since the treatment is noninvasive and has minimal associated side effects, our results support the use of the light visors at CFS Alert and other northern outposts during the winter for individuals who are experiencing sleep difficulty or low mood.

INTRODUCTION

Maintenance of good mood and a regular sleep pattern can be particularly challenging in the high arctic during winter. Since light is the dominant external time cue, or zeitgeber, for the human circadian system, the lack of a natural photoperiod places particular strain on an individual's circadian rhythm. This is often associated with poor mood, subsyndromal Seasonal Affective Disorder (SAD) and mild insomnia (1). Melatonin production is the best marker for circadian timing, and there is evidence that an increased quantity and duration of melatonin production in the winter of polar regions (2–4) may contribute to the somatic symptoms associated with SAD (5).

The purpose of the work reported here was to develop and implement light treatments that improve mood and sleep quality among staff at Canadian Forces Station (CFS) Alert (82° 30′ 00″ N – 62° 22′ 00″ W, and 817 km from the geographic North Pole) during the 24 h darkness of the winter. Previous studies at CFS Alert found that sleep difficulty is common on Station. During the winter, sleep quantity is generally sufficient (6), but mild insomnia and high negative affectiveness is frequently reported on Station, necessitating the need for treatment recommendations that are ideally noninvasive, relatively unimposing and with minimal or no associated side effects. Light treatment has previously been shown to improve scores on depression and negative affect scales (7–9), and the light treatment devices that were used for this study have been previously evaluated in our laboratory to be effective for suppressing endogenous melatonin and shifting the circadian system (10). We therefore believe that, if effective, such devices are an appropriate therapeutic modality for treatment of low mood or subsyndromal SAD and mild sleep quality problems present in polar environments during the winter months.

MATERIALS AND METHODS

Subject inclusion/Exclusion criteria/Age/Gender demographics. Data collection commenced on January 10, 2014 at Canadian Forces Station (CFS) Alert and ended on the morning of February 2, 2014. Therefore, the sun was at least 9 degrees below the horizon for the entire duration of the study. To qualify for the study, the subjects had to have been at CFS Alert for at least 2 weeks. The trial included 13 subjects (eight males and five females, age range of 22–42 years, with mean age and standard deviation of 31.2 ± 6.3 years), but one male subject had to be removed from the analysis of actigraphic sleep quality/quantity parameters due to preexisting insomnia associated with use of a methamphetamine used to treat a medical condition. Anyone taking beta-blockers, selective serotonin reuptake inhibitors, hypnotics, or supplementary melatonin was excluded from the study.

Data collection protocol. The subjects donned activity monitors (Motionlogger, version 14.000; Ambulatory Monitoring Inc., Amherst, NY) for the entire duration of the trial. The activity monitors were used to record various measures of sleep including time of sleep onset, total duration of sleep, sleep efficiency, arise time, total wake minutes after sleep onset, etc. To cover for the possibility of activity monitor failure, the subjects also maintained a sleep log for the entire duration of the trial. After a week of wearing the actigraph, all subjects underwent a 24-h pretreatment salivary melatonin assessment profile by providing 13 samples (one sample every 2 h for 24 h). For the salivary melatonin assessment, the subjects arrived at the Station gymnasium on Saturday January 18th, 2014 at 0830 h. They were assigned lounge chairs for the 24-h saliva

*Corresponding author email: ryan.love@drdc-rddc.gc.ca (Ryan J. Love)

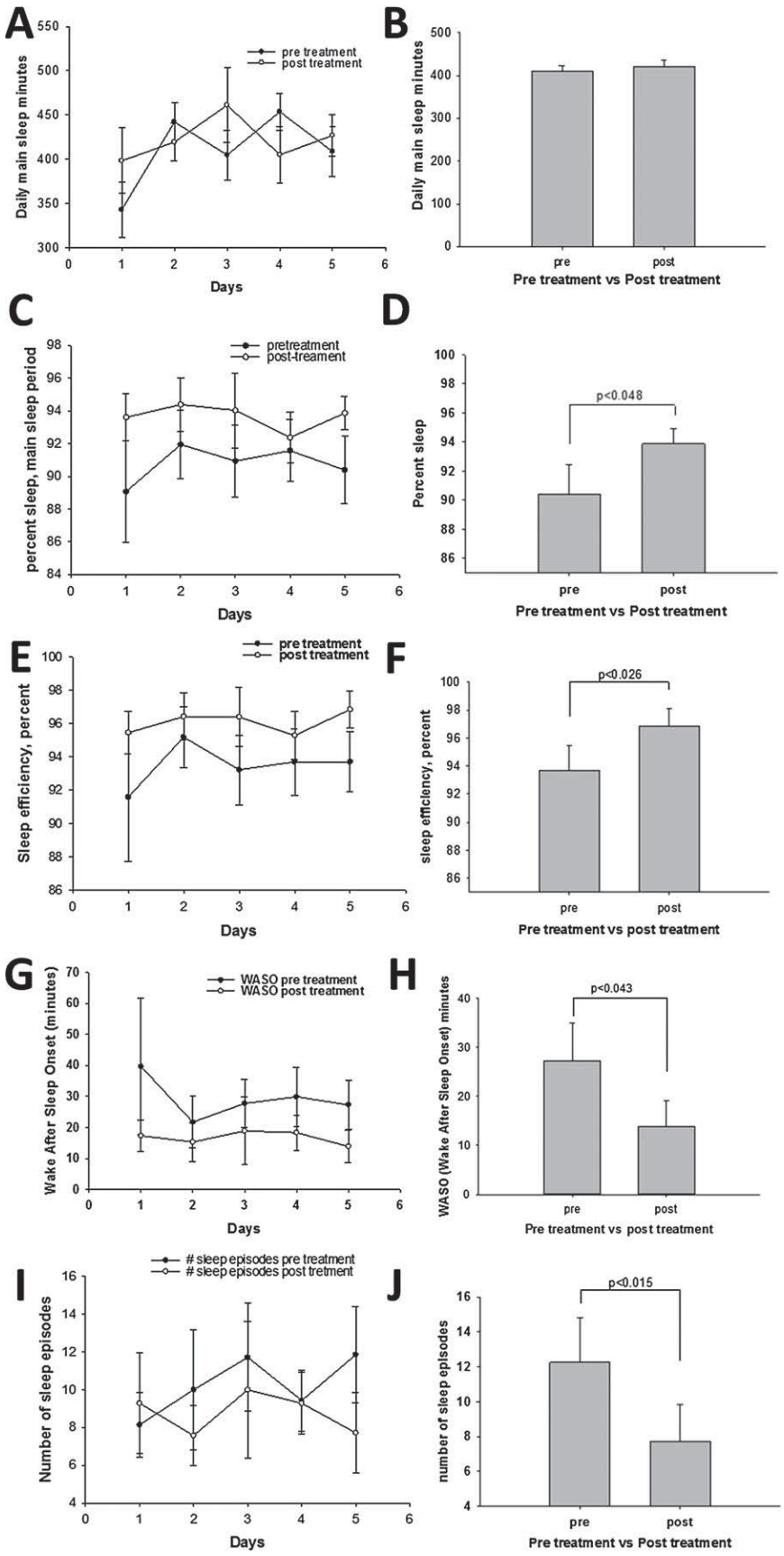
A
Daily main sleep minutes
pre treatment
post treatment
Days
B
Daily main sleep minutes
pre
post
Pre treatment vs Post treatment
C
percent sleep, main sleep period
pretreatment
post-treament
Days
D
Percent sleep
p<0.048
pre
post
Pre treatment vs Post treatment
E
Sleep efficiency, percent
pre treatment
post treatment
Days
F
sleep efficiency, percent
p<0.026
pre
post
Pre treatment vs post treatment
G
Wake After Sleep Onset (minutes)
WASO pre treatment
WASO post treatment
Days
H
WASO (Wake After Sleep Onset) minutes
p<0.043
pre
post
Pre treatment vs post treatment
I
Number of sleep episodes
sleep episodes pre treatment
sleep episodes post tretment
Days
J
number of sleep episodes
p<0.015
pre
post
Pre treatment vs Post treatment

Figure 1. Sleep quantity and quality measures derived from activity monitors (actigraphs) for the seven treated subjects. (A) Sleep minutes from the daily main sleep period. (B) Pre- *vs* Posttreatment sleep duration (plots are means of the 5-days immediately prior to pre/posttreatment). (C) The daily percentage of time spent sleeping of the total time spent in bed trying to sleep. (D) Pre- *vs* Posttreatment percentage of time spent sleeping of the total time spent in bed trying to sleep (plots are means of the 5-days immediately prior to pre/posttreatment). (E) The efficiency of main sleep periods following sleep onset, pre- *vs* posttreatment. (F) Mean Sleep Efficiency for the day prior to pre/posttreatment saliva data collection. (G) The number of minutes spent awake (WASO) after sleep onset, pre- *vs* posttreatment. (H) Mean WASO for the day prior to pre/posttreatment saliva data collection. (I) Number of individual sleep episodes during the main sleep period, pre- *vs* posttreatment. (J) Mean Number of Sleep Episodes for the day prior to pre/posttreatment saliva data collection.

collection period. The gymnasium lights were turned off but low-intensity supplementary lighting (to a maximum of 5 lux) was positioned around the gymnasium and in the washrooms. A large monitor was setup approximately 20 feet in front of the line of subjects (in their lounge chairs) to provide the subjects with movies during the 24-h period, with eye-level ambient light from the monitor measuring less than 5 lux for all subjects. The subjects were allowed to get up from their chairs to go to the washrooms or socialize for about 100 min of each 2 h block, but remained in areas with ambient light of less than 5 lux. The subjects returned to a semirecumbent posture in their lounge chairs for the 15 min prior to each sample. The subjects were required to remain awake from 0900 h until 2300 h, after which they could sleep but were awakened in the 15 min prior to each sample. The subjects were released Sunday morning after the 0900 h sample.

The saliva was collected in test tubes configured for that purpose (Salivette®; Sarstedt Inc., Montreal, QC). These saliva samples were analyzed for melatonin content at CFS Alert by DRDC, Toronto Research Center Staff using ELISA kits from Bühlmann Laboratories AG (Schönenbuch, Switzerland), which utilize the Kennaway G280 anti-melatonin antibody. These salivary melatonin levels were used to calculate the time of onset of the daily melatonin rhythms (DLMO or Dim Light Melatonin Onset) in each of the participating subjects. For subjects whose DLMO was either significantly advanced or delayed (relative to a normal DLMO for their particular work schedule) or had inappropriately-timed daytime spikes in their melatonin profile, an individualized light treatment schedule using a light visor (Feel Bright Light; Physician Engineered Products, Bangor, Maine) was prescribed. The pretreatment melatonin rhythms and the treatment prescriptions are illustrated and described in more detail in the supplementary information. The light treatments were implemented for 11 days, after which a posttreatment 24-h salivary melatonin profile was collected in the same manner as described above. The posttreatment salivary melatonin samples were also analyzed via ELISA at CFS Alert. The difference in timing of the pretreatment to posttreatment DLMO provided the magnitude and direction (advance or delay) and thus the efficacy of the phase shift for those participants who received a treatment.

Immediately prior to each of the pre- and posttreatment salivary melatonin profile assessments, all subjects completed several questionnaires to measure the psychological parameters of interest [Patient Health Questionnaire depression and anxiety symptoms scales (11), Pittsburgh Sleep Quality Index (12), Positive and Negative Affect Scale (13,14)]. The Horne-Ostberg questionnaire (15) was completed prior to the first 24-h salivary melatonin collection to establish chronotype (*i.e.* morningness/eveningness). All questionnaires used in this study have undergone rigorous validation and are commonly used in the psychological literature.

Light treatments. The light visor that we used for this study was previously evaluated against competitor products for its efficacy in suppressing endogenous melatonin production and inducing circadian phase shifts (10). The device has an array of three monochromatic LED lights (505 nm), mounted by Velcro on the brim of the visor about 10 cm in front of each eye. The illuminance can be set to either 8000 or 12 000 lux at eye level, but only the 8000-lx setting was used in this study. The LED lights on the visor are powered by rechargeable batteries and thus allow subjects to walk around or work without being tied down to a desk, as is the case for light treatment devices that are plugged into electric outlets.

The individualized light treatment schedules were based on each subject's melatonin rhythm. The pretreatment melatonin profile of most participants showed some unusually high levels for brief periods during the daytime. Therefore, light treatments were focused on the times of these points of raised melatonin. The pretreatment melatonin profile of each subject and their respective light treatment prescription are provided in the supplementary information. A declaration of treatment compliance was completed by each subject immediately prior to the posttreatment saliva collection.

Saliva collection and melatonin analysis. Saliva was collected from the participants every two hours using cotton Salivette® collection devices. During sample collection, the subjects chewed the cotton swab for 45 s, and then let the swab remain in their mouth to absorb saliva for another 45 s. The cotton swab was then replaced in the Salivettes® collection device and the samples were centrifuged for 5 min to extract the saliva. The cotton swab was then discarded and the samples were refrigerated at 4°C. Melatonin content was analyzed at CFS Alert by DRDC, Toronto Research Center Staff immediately following collection of the final sample. All samples from a given subject were analyzed in duplicate on the same ELISA plate. Dim light melatonin onset (DLMO) was calculated by linear interpolation of the samples that straddled the prescribed threshold of salivary melatonin, defined as two standard deviations above the baseline for the individual melatonin profile (with the exclusion of any spurious peaks in melatonin concentration).

Calculation of daily sleep quantity and quality. The quantity of total daily sleep and indices of sleep quality for the 5 days prior to each data collection were calculated from the wrist activity monitors worn by the subjects. The actigraphy data were collected in "zero crossing mode" using one-minute epochs. Sleep periods were marked on the actigrams with the help of sleep logs, using the manufacturer's software (ActionW version 2.6; Ambulatory Monitoring Inc., Amherst, NY). Sleep was scored using the Cole-Kripke algorithm (16). The sleep quality parameters generated by the actigraphy data, and calculated by the manufacturer's software were: Total Sleep Time, Percent Sleep, Sleep Efficiency, Wake After Sleep Onset (WASO) and the number of Sleep Episodes (per total sleep period). Total Sleep Time is defined as the duration of time spent asleep, percent sleep is the percentage of time spent sleeping after going to bed, Sleep Efficiency is defined as the percentage of time spent sleeping after initial onset, WASO is defined as the total time spent awake after initial sleep onset and before final awakening and the number of Sleep Episodes is defined as the total number of continuous sleep periods between sleep onset and final awakening. The validity of these measures has been assessed by de Souza *et al.* (17). A detailed review of the validity and role of actigraphy in sleep and circadian rhythm research has been published by Ancoli-Israel *et al.* (18).

Statistics. Statistical differences were assessed using ANOVA (Statistica, StatSoft Inc., Tulsa, OK) when and where appropriate. Details on the type of ANOVA and the layout of the data used for analysis are provided in the description of the statistical results below. Paired t-tests were performed for several of the quantitative and qualitative sleep quality measures to evaluate differences between pre- and posttreatment.

The estimated power of the various two-tailed statistical tests for $P = 0.05$, an effect size of 1 standard deviation (appropriate for a field trial), $n = 7$ (treated subjects) and a test–retest reliability for repeated measures of $r = 0.50$, was 68% (19). As this is somewhat lower than the standard (80%), concerns about the type 2 (false negative) are discussed below.

RESULTS

Sleep quantity is not affected, but sleep quality improves following 11 days of light treatment

The quantity of sleep obtained daily during the main sleep period was evaluated using a within-subjects repeated measures ANOVA (pre- *vs* posttreatment × 5 days). There was no significant main effect of treatment [$F(1,6) = 0.53$, $P = 0.49$], nor was there a significant main effect of Days [$F(3,24) = 1.47$, $P = 0.24$], or interaction between Days and Treatment [$F(4,24) = 1.76$, $P = 0.17$] (see Fig. 1A,B). Except for the first pretreatment day, where subjects averaged less than 6 h (360 min) of sleep, subjects generally attained, on average, 7 h

(420 min) of daily sleep or better. A table containing each subject's mean bedtime, mean arise time, sleep duration, sleep efficiency, melatonin phase (DLMO) and subjective questionnaire results are provided in Table 1 below.

Actigraphy data were used to compare sleep between pre- and posttreatment. Again, a within-subjects repeated measures ANOVA (pre- *vs* posttreatment × 5 days) was used to evaluate differences between pre- and posttreatment. A significant main effect of treatment was found for Percent sleep [$F(1,6) = 6.08$, $P = 0.048$] (see Fig. 1C,D). There was no significant treatment effect found for the other sleep parameters using a repeated measures ANOVA to analyze 5 days prior to each data collection; however, there was a notable trend toward improvement for the other parameters (sleep efficiency, see Fig. 1E; wake after sleep onset, see Fig. 1G; and number of sleep episodes, see Fig. 1I). Therefore, to evaluate the full effect of treatment, only the last night of sleep prior to each of the pre- and posttreatment salivary melatonin data collections was analyzed using paired t-tests. From this analysis, significant differences were found for sleep efficiency ($P = 0.026$; Fig. 1F), wake after sleep onset ($P = 0.043$; Fig. 1H), and the number of sleep episodes ($P = 0.015$; Fig. 1J).

Sleep quality improves subjectively following 11-days of light treatment

The seven treated research participants collectively reported a significantly improved quality of sleep on the Pittsburgh Sleep Quality Index (Fig. 2A), which indicates that the subjects subjectively believed the treatment improved their quality of sleep. The statistically significant improvement in reported "difficulty falling or staying asleep" over the last 2 weeks (Fig. 2B), a question from the Patient Health Questionnaire depression scale, also supports the finding that sleep quality subjectively improved due to the light treatment.

Light treatment reduces negative affectiveness

Positive Affectiveness did not change after the 11-day treatment; however, a statistically significant reduction in the mean negative affect scores was observed pre- to posttreatment for the seven treated subjects (Fig. 3).

Circadian system adaptation to the lack of photoperiod in the arctic winter

The decision as to who would receive circadian adjustment was strictly based on the melatonin profiles obtained from the subjects. Once treatment had commenced, we noted that the five subjects that did not require any circadian adjustments were the same individuals who had been on station the longest: 2 months, on average (Fig. 4). In contrast, the seven subjects that required circadian intervention with daily light treatments were on station for the shortest amount of time: 5 weeks, on average (Fig. 4). The pre- and posttreatment melatonin profiles for each subject (including nontreated subjects) are provided in the supplementary information.

DISCUSSION

Negative affect is a common problem in polar environments during the winter months and may be particularly problematic on stations in which residents are isolated from friends and family. The finding that negative affect is reduced after light treatment supports its use among residents in the arctic during winter months. Feelings associated with negative affect may stem from isolation from friends and family while on the arctic station combined with the confined nature of living on these types of stations, and may become further amplified by the circadian system's response to the lack of photoperiod during Arctic winter. To provide some context, PANAS negative affect scores range

Table 1. Pre/Post data for treated and nontreated subjects.

Subject No.	Age	Gender	Chronotype	Bed-time		Arise time		Sleep duration		Sleep efficiency		DLMO		PSS		+ve affect		-ve affect	
				Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
Treated Subjects																			
1	41	Male	DMT (73)	2207	2235	0640	0549	427	406	85.0	94.7	1303	1306	23	18	38	45	10	11
2	32	Male	MET (32)	2408	2422	0722	0805	208	376	77.7	89.6	2304	2316	29	20	28	38	28	22.5
3	22	Female	MMT (62)	2323	2258	0633	0449	413	376	98.3	96.8	1715	1913	16	10	45	44	11	10
8	25	Male	MMT (62)	2249	2313	0547	0603	366	375	88.7	94.2	2123	2101	19	15	37	32	14	13
9	28	Male	MET (34)	2450	2439	0707	0820	353	440	96.9	97.9	2509	2309	20	15	22	31	19	15
11	26	Male	NT (56)	2344	2342	0719	0751	388	433	90.6	91.3	2303	2318	21	18	36	36	18	15
12	34	Male	NT (53)	2310	2249	0701	0623	453	439	97.2	98.4	1910	1922	12	10	34	37	10	10
13	29	Female	MET (33)	2235	2240	0647	0659	472	484	97.7	99.2	1725	1703	16	16	34	32.5	13	12
Means	29.6		NT (50.6)	2321	2322	0649	0647	385	416	91.5	95.3	1956	1956	19.5	15.3	34.3	36.9	15.4	13.6
Nontreated Subjects																			
4	24	Female	NT (42)	2339	2333	0744	0804	417	422	97.3	96.5	2100	2103	14	14	22	26	38	36
5	32	Female	NT (56)	2152	2238	0708	0706	464	420	86.6	85.8	1914	2117	19	20	29	33	11	12
6	42	Female	MMT (61)	2351	2330	0711	0703	287	407	91.5	93.2	1953	2103	16	18	41	42	16	19
7	34	Male	NT (43)	2317	2356	0721	0741	457	437	96.0	96.7	2102	2043	22	16	35	34	12	10
10	37	Male	DMT (74)	2102	2114	0458	0622	412	434	88.0	81.2	1708	1903	23	16	17	25	11	9
Means	33.8		NT (55.2)	2244	2258	0652	0715	427	424	91.9	90.7	1939	2038	18.8	16.8	28.8	32	17.6	17.2

Note 1: Bedtime, Arise Time, Sleep Duration and Sleep Efficiency reflect the mean of the 5 days prior to pre- or posttreatment data collection. Note 2: Fig. 1E,G and I graph only the day prior to pre- or posttreatment salivary melatonin data collection. Note 3: Subject 2 was removed from the analysis of actigraphic sleep quality/quantity parameters due to preexisting insomnia. PSS, Pittsburgh Sleep Scale; DLMO, Dim Light Melatonin Onset; DMT, definitely morning type; MMT, moderately morning type; NT, neither type; MET, moderately evening type; DET, definitely evening type.

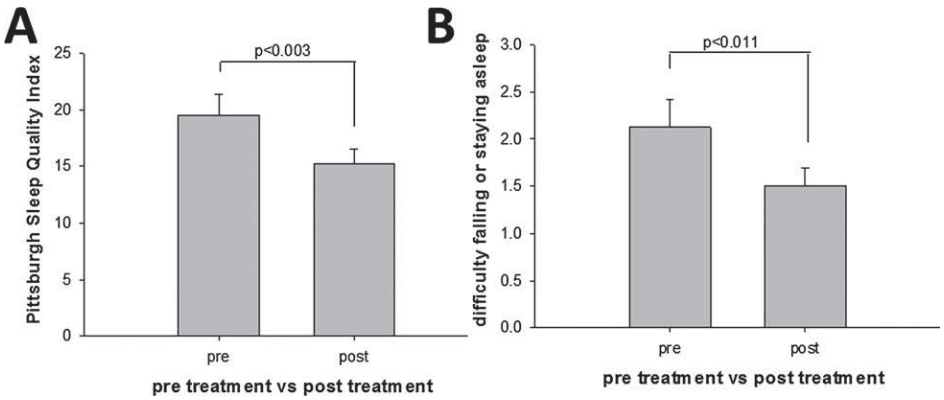

Figure 2. Pre- *vs* Posttreatment differences in subjective sleep quality scores from the (A) Pittsburgh sleep scale, and (B) Patient Health Questionnaire, difficulty falling or staying asleep question. Values are ± SEM.

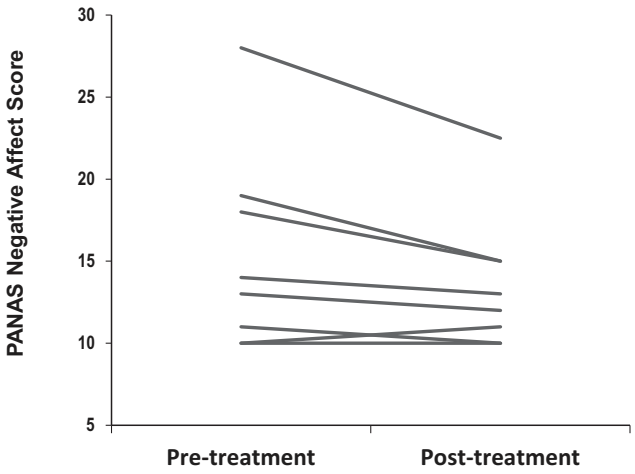

Figure 3. Pre- *vs* Posttreatment score on the negative affectivity scale.

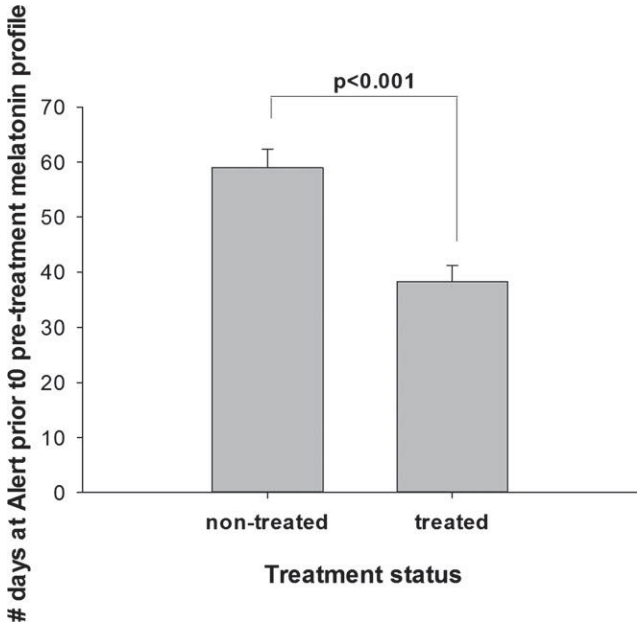

Figure 4. Difference in the duration of time on station between treated and nontreated groups.

from 10 (no negative affect) to 50. An individual who reports being moderately jittery, afraid, ashamed, nervous, irritable, guilty, scared, hostile, distressed and upset would score a 30. The mean pretreatment negative affect score for treated subjects was 15.4, and the posttreatment score was 13.6. Therefore, the negative affect observed was not particularly high. Nevertheless, reducing negative affect among residents should be a priority for health workers on polar stations, and the results presented herein support the use of light treatment as an appropriate countermeasure against cases of high negative affectiveness that may be linked to the circadian system.

Notably, the quantity of sleep obtained by the subjects did not increase in response to the light treatment; however, as we learned from our baseline studies that were performed at CFS Alert (6), the quantity of sleep obtained during the winter in the Arctic is generally sufficient. The light treatments had a positive impact on subjective (Pittsburgh Sleep Quality Index) and objective measures of sleep quality. Specifically, the treated subjects spent less time awake after sleep onset, as measured by the Wake after Sleep Onset (WASO) parameter (Fig. 3G), and had fewer awakenings, as measured by the number sleep episodes (Fig. 3I). These collectively lead to an improvement in sleep efficiency (Fig. 3E), and the percent of time spent asleep during the daily main sleep periods (Fig. 3B,C). The Pittsburgh Sleep Quality Index scores similarly improved between pre- and posttreatment (Fig. 1A), which supports the actigraphy findings. Therefore, although the subjects were already obtaining sufficient sleep, light treatment lead to qualitatively better sleep (*i.e*. more efficient sleep with fewer awakenings, etc.).

As described in the methods section, the estimated power for the various two-tailed statistical tests used in our analysis (for $P = 0.05$) was 68% (19). As this is somewhat lower than the standard (80%), there is a likelihood of type 2 error (false negative). The reader should note that despite the trend toward improvement for WASO, number of sleep episodes and sleep efficiency, our ANOVA analysis showed that there was no significant difference between the groups. Therefore, because the efficacy of light treatment is additive over multiple days, we analyzed only the last night of sleep prior to each of the pre- and posttreatment salivary melatonin data collections so as to evaluate the full effect of treatment. This resulted in the significant differences between pre- and posttreatment for the sleep quality scores mentioned in the paragraph above.

A possible limitation of this study is that all the sleep parameters reported were quantified by actigraphy, which does not provide a direct and precise assessment of sleep structure, duration and quality. We refer the reader to the reports by de Souza *et al.* (17), which describes the validity of these measures, and the review by Ancoli-Israel *et al.* (18), which outlines the validity and role of actigraphy in sleep and circadian rhythm research.

The baseline melatonin profiles obtained from many of the subjects were unusual, with high daytime "spikes" not normally seen in controlled laboratory conditions in our data or that of others. Therefore, treatment was often focused on reducing the daytime "spikes" of melatonin that were being produced. The five subjects that were not prescribed light treatment had a melatonin profile that was normal in onset and offset timing, and had no significant spikes of melatonin during the day. Treatment timing was based on the assumption that light treatment would both suppress (immediately) and phase shift melatonin (according to our phase response curve [PRC] (20), which is based on light treatment that is similar to that used in this study); however, the "spikes" of daytime melatonin made the identification of DLMO and the position of the PRC difficult in several cases. Although it is possible that some of these values might be due to salivary substances interfering with the ELISA, we refer to the tests of binding affinity for possible interfering agents performed by Buhlmann laboratories, and confirm that all melatonin-like molecules tested have only a negligible affinity for the Kennaway G280 antibody that is used on the ELISA plates. Furthermore, the timing of the spikes among the participants was not consistent, which suggests that these spikes were from endogenous production, and not due to residual melatonin or melatonin-like structures derived from diet during the trial. However, in two subjects (1 and 9) with high daytime spikes the light treatment clearly did not suppress "melatonin" and possible interference must be suspected.

From our data, it appears that there is an adaptation by the circadian system that occurs in response to the (lack of) natural light in the Arctic during the winter, as the five subjects that did not require any circadian adjustments were the five individuals who had been on station the longest: 2 months, on average (Fig. 4). In contrast, the seven subjects that required circadian intervention with daily light treatments were on station for the shortest amount of time: 5 weeks, on average (Fig. 4). The most likely reason for this is an increased sensitivity to the entraining and melatonin-suppressing power of ambient dim light that develops while on Station (21–24).

A notable drawback of any treatment, including light treatment, is the side effects that may arise. In our previous study using the light visor (*i.e*. The Feel Bright Light, Physician Engineered Products, Bangor, Maine USA), several subjects reported side effects including psychomotor performance impairment, eye discomfort, difficulty viewing a computer screen and difficulty reading a book/magazine(10). The subjects in the study reported herein noted that eye discomfort was particularly pronounced when the light visor was used in rooms with low ambient light, but otherwise side effects were not objectively assessed or reported by the subjects. In general, side effects are an operational concern, and the decision to use a light treatment device should be based on the pros and cons of the particular device. An optimal light treatment device will have a monochromatic light source with a wavelength of 480–500 nm, and an eye-level light intensity that does not provoke eye discomfort but is effective in suppressing melatonin production. The light treatment device should also be easy to use, and not impose on the individual's work or take time away from scheduled tasks, with the exception that driving with any light treatment device is not recommended.

Several research groups have looked at nonvisual spectral sensitivity to light via melatonin suppression. For example, Wright and colleagues compared short-wavelength (blue/green) monochromatic light and found that the best suppression of melatonin occurred at 497 nm (25), but the best circadian phase shift (advance) was caused by 470 nm (26). Thapan (27) and Brainard (28) independently developed extremely similar action spectrums for melatonin suppression with peak suppression being at about 460–470 nm. Najjar and colleagues have recently reported that peak sensitivity was shifted to a longer wavelength in older participants (494 nm, average age = 61) compared to young participants (484 nm, average age = 25), but surprisingly melatonin suppression was not reduced despite decreased short-wavelength light transmittance through the lens (29). In comparison, the wavelength of the light visor used in our experiment was slightly longer (505 nm) than the ideal wavelength reported above, but has been previously evaluated to be effective for melatonin suppression and circadian phase shifting (10).

In summary, the light treatment that was prescribed to the subjects was effective in improving subjective and objective measures of sleep quality and reducing negative mood. The light treatment that was used for our study was in the form of a light visor that was worn by an individual for 1 h each day (2 h in the case of Subject 13). It should be noted that similar improvements in sleep quality have been observed on an Antarctic base by increasing ambient light levels throughout the working day (2,30). Although this may be possible for some research or defense stations, we believe that utilization of individual light treatment devices is appropriate for stations that have multiple buildings, with personnel working in various locations, as is seen at CFS Alert. Furthermore, the light treatment visors are likely simpler and less expensive to implement and operate. As a final note, since statistical power of the experiment described herein was limited due to the low number of treated subjects, we hope to perform similar studies in the near future to improve our understanding of the relative efficacy of our circadian countermeasures.

Acknowledgements—Support: Canada DND Research Project 04KD07 awarded to MAP.

SUPPORTING INFORMATION

Additional Supporting Information may be found in the online version of this article:

Data S1. Melatonin Profiles.

REFERENCES

1. Pallesen, S., I. H. Nordhus, G. H. Nielsen, O. E. Havik, G. Kvale, B. H. Johnsen and S. Skjotskift (2001) Prevalence of insomnia in the adult Norwegian population. *Sleep* **24**, 771–779.
2. Arendt, J. (2012) Biological rhythms during residence in polar regions. *Chronobiol. Int.* **29**, 379–394.
3. Kauppila, A., A. Kivela, A. Pakarinen and O. Vakkuri (1987) Inverse seasonal relationship between melatonin and ovarian activity in humans in a region with a strong seasonal contrast in luminosity. *J. Clin. Endocrinol. Metab.* **65**, 823–828.
4. Paakkonen, T., J. Leppaluoto, T. M. Makinen, H. Rintamaki, A. Ruokonen, J. Hassi and L. A. Palinkas (2008) Seasonal levels of mela-

tonin, thyroid hormones, mood, and cognition near the Arctic Circle. *Aviat. Space Environ. Med.* **79**, 695–699.
5. Rosenthal, N., D. Sack, F. Jacobsen, S. James, B. Parry, J. Arendt, L. Tamarkin and T. Wehr (1985) Melatonin in seasonal affective disorder and phototherapy. *J. Neural Transm. Suppl.* **21**, 257–267.
6. Paul, M. A., R. J. Love, A. M. Hawton, D. Ebisuzaki, J. McHarg, W. G. Burfitt, T. P. Harris, K. S. Luomala, R. N. Watson, D. W. Williams and J. Arendt (2014) Melatonin production, sleep patterns and modeled performance effectiveness in subjects in the high Arctic. DRDC-RDDC-2014-R15, DRDC Toronto Research Centre.
7. Flory, R., J. Ametepe and B. Bowers (2010) A randomized, placebo-controlled trial of bright light and high-density negative air ions for treatment of Seasonal Affective Disorder. *Psychiatry Res.* **177**, 101–108.
8. Hsu, Z. Y., D. S. Moskowitz and S. N. Young (2014) The influence of light administration on interpersonal behavior and affect in people with mild to moderate seasonality. *Prog. Neuropsychopharmacol. Biol. Psychiatry* **48**, 92–101.
9. Sitnikov, L., K. J. Rohan, M. Evans, J. N. Mahon and Y. I. Nillni (2013) Cognitive predictors and moderators of winter depression treatment outcomes in cognitive-behavioral therapy vs. light therapy. *Behav. Res. Ther.* **51**, 872–881.
10. Paul, M. A., J. C. Miller, G. Gray, F. Buick, S. Blazeski and J. Arendt (2007) Circadian phase delay induced by phototherapeutic devices. *Aviat. Space Environ. Med.* **78**, 645–652.
11. Spitzer, R. L., K. Kroenke, J. B. W. Williams and P. Patient Hlth Questionnaire (1999) Validation and utility of a self-report version of PRIME-MD – The PHQ primary care study. *JAMA J. Am. Med. Assoc.* **282**, 1737–1744.
12. Buysse, D. J., C. F. 3rd Reynolds, T. H. Monk, S. R. Berman and D. J. Kupfer (1989) The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. *Psychiatry Res.* **28**, 193–213.
13. Watson, D., L. A. Clark and A. Tellegen (1988) Development and validation of brief measures of positive and negative affect: the PANAS scales. *J. Pers. Soc. Psychol.* **54**, 1063–1070.
14. Watson, D. and J. W. Pennebaker (1989) Health complaints, stress, and distress: exploring the central role of negative affectivity. *Psychol. Rev.* **96**, 234–254.
15. Horne, J. A. and O. Ostberg (1976) A self-assessment questionnaire to determine morningness-eveningness in human circadian rhythms. *Int. J. Chronobiol.* **4**, 97–110.
16. Cole, R. J., D. F. Kripke, W. Gruen, D. J. Mullaney and J. Gillin (1992) Automatic sleep/wake identification from wrist activity. *Sleep* **15**, 461–469.
17. de Souza, L., A. A. Benedito-Silva, M. L. N. Pires, D. Poyares, S. Tufik and H. M. Calil (2003) Further validation of actigraphy for sleep studies. *Sleep* **26**, 81–85.
18. Ancoli-Israel, S., R. Cole, C. Alessi, M. Chambers, W. Moorcroft and C. P. Pollak (2003) The role of actigraphy in the study of sleep and circadian rhythms. *Sleep* **26**, 342–392.
19. Cohen, J. (1988) *Statistical Power Analysis for the Behavioral Sciences*. Lawrence Erlbaum Associates, Hillsdsale, NJ.
20. Paul, M. A., J. C. Miller, R. J. Love, H. R. Lieberman, S. Blazeski and J. Arendt (2009) Timing light treatment for eastward and westward travel preparation. *Chronobiol. Int.* **26**, 867–890.
21. Owen, J. and J. Arendt (1992) Melatonin suppression in human subjects by bright and dim light in antarctica: time and season-dependent effects. *Neurosci. Lett.* **137**, 181–184.
22. Hebert, M. S. K., M. C. Lee and C. I. Eastman (2002) The effects of prior light history on the suppression of melatonin by light in humans. *J. Pineal Res.* **33**, 198–203.
23. Chang, A.-M., F. A. J. L. Scheer and C. A. Czeisler (2011) The human circadian system adapts to prior photic history. *J. Physiol.* **589**, 1095–1102.
24. Chang, A.-M., F. A. J. L. Scheer, C. A. Czeisler and D. Aeschbach (2013) Direct effects of light on alertness, vigilance, and the waking electroencephalogram in humans depend on prior light history. *Sleep* **36**, 1239–1246.
25. Wright, H. R. and L. C. Lack (2001) Effect of light wavelength on suppression and phase delay of the melatonin rhythm. *Chronobiol. Int.* **18**, 801–808.
26. Wright, H. R., L. C. Lack and D. J. Kennaway (2004) Differential effects of light wavelength in phase advancing the melatonin rhythm. *J. Pineal Res.* **36**, 1–5.
27. Thapan, K., J. Arendt and D. J. Skene (2001) An action spectrum for melatonin suppression: evidence for a novel non-rod, non-cone photoreceptor system in humans. *J. Physiol.* **535**, 261–267.
28. Brainard, G. C., J. P. Hanifin, J. M. Greeson, B. Byrne, G. Glickman, E. Gerner and M. D. Rollag (2001) Action spectrum for melatonin regulation in humans: evidence for a novel circadian photoreceptor. *J. Neurosci.* **21**, 6405–6412.
29. Najjar, R. P., C. Chiquet, P. Teikari, P.-L. Cornut, B. Claustrat, P. Denis, H. M. Cooper and C. Gronfier (2014) Aging of non-visual spectral sensitivity to light in humans: compensatory mechanisms? *PLoS ONE* **9**, e85837.
30. Mottram, V., B. Middleton, P. Williams and J. Arendt (2011) The impact of bright artificial white and 'blue-enriched' light on sleep and circadian phase during the polar winter. *J. Sleep Res.* **20**, 154–161.