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Effects of Light Treatment on Sleep Structure in Seasonal Affective Disorder

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Summary. Twelve outpatients with seasonal affective disorder (depression, winter type) were treated by 1 h of bright light exposure for five mornings. The intervention produced a significant reduction in depression scores, but no change was seen in the sleep electroencephalographic variables recorded after light treatment. Significant changes were seen, however, in ratings of subjective sleepiness. The acrophase of the circadian sleepiness rhythm was phase advanced, the mean level of the sleepiness rhythm was diminished, and the mean values of sleepiness scores were reduced at 8 and 10 a.m. This minimal influence of bright light on sleep structure is unlikely to explain the well-documented antidepressant effect.

Key words: Seasonal affective disorder – Sleep electroencephalography

Introduction

Seasonal affective disorder (SAD) is characterized by recurrent major depressive episodes, which are bound to a certain season (Rosenthal et al. 1984). The syndrome of winter depression includes fatigue and oversleeping as the core symptoms. Morning bright light exposure has been shown to be an effective treatment for winter depression (Terman et al. 1989). The antidepressant effect of light has been hypothesized to be a result of a correction of the phase angle between certain circadian rhythms and the sleep-wake cycle (Lewy et al. 1987). However, it has not been conclusively excluded that a sleep restriction might also play a role in the response to light treatment.

The aim of our study was to test the above mentioned sleep-related hypothesis in patients with winter depression. The effects of bright light exposure on sleep processes were investigated by repeated sleep electroencephalographic recordings.

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Methods

Outpatients were exposed to diffused bright cool-white fluorescent light in a white-walled room in Ullanlinna Sleep Disorders Clinic, Helsinki (60°N). The lights (30 lamps; 36 W, 4000 K, and DIN 5035 each) were mounted into the ceiling. The illuminance of light reached >3500 lux at floor level and the luminance of light from the walls was $>600\,cd/m^2$ all over the room. The light treatment was administered for 1 h for five consecutive mornings during February-March 1988, October 1988–March 1989, or November 1989–January 1990. The most suitable hour for treatment was allowed to be chosen by the patients within the range of 06.30–08.30 a.m. A nurse checked that patients had woken up and had kept their eyes open. Patients were allowed to move freely in the room.

Circadian data of the subjective sleepiness rhythm were collected during two 24-h stays in the clinic 1 week before and immediately after cessation of treatment. Sleepiness was recorded with a 100-mm Visual Analogue Scale (VAS) administered every other hour during 24 h (McCormack et al. 1988). The best fitting cosinor function was adjusted to individual data of the circadian rhythm using the least square method (Nelson et al. 1979).

Sleep electroencephalographic recordings were made during two 24-h stays in the clinic one week before and immediately after cessation of treatment. Twelve-channel recordings (including EEG, EMG, EOG, and ECG) were analysed manually using a standardized scoring system (Rechtschaffen and Kales 1968). Before the first recording, there was an adaptation night, when the electrodes were attached but no recording was made. Hamilton 21-item Rating Scale (HDRS) and several self-rating scales, including Beck Depression Inventory (BDI), were administered during the sleep and circadian data collecting stays and again week after cessation of treatment (Beck 1967, Hamilton 1967).

Standard statistical methods, including correlations and *t*-tests, were used for analysis of the data. The level of 0.05 was considered as indicating a significant difference. One patient's second electroencephalographic scoring was excluded from the analysis owing to missing data. Regression analysis models with multiple general linear hypothesis (MGLH) and cluster analysis methods were also used for interpretation of the data (Wilkinson 1990). Multiple stepwise regressions included the difference in HDRS score and the values scored 1 day and 1 week after cessation of the treatment as dependent variables. Age of the subjects, changes in the subjec-

tive sleepiness rhythm parameters measured, sleep latency, slow wave latency, and in REM latency were considered as independent variables in these analyses. The minimum tolerance for entry into a model was 0.01, and in a forward stepwise regression the alpha-to-enter and alpha-to-remove was 0.15. Cluster analysis included age, HDRS scores, BDI scores, subjective sleepiness rhythm parameters, sleep latencies, REM latencies and slow wave latencies as variables analyses. Cluster analysis was made between and within subjects.

Patients

Outpatients were referred to the study by local physicians familiar with our program. A psychiatrist in the research group interviewed the patients for the DSM-III-R diagnosis of major depressive episode of seasonal (winter) pattern (APA 1987). The inclusion criteria were: 1. an episode or a history of major depression, 2. a history of at least two consecutive years of winter depressive (minor or major) episodes remitting during summer, and 3. absence of any other DSM-III-R axis I psychiatric disorder. The exclusion criteria were: 1. a chronic somatic disease, 2. a primary sleep disorder, and 3. a present or past history of alcohol or drug abuse.

11 women and 1 man participated. The age of the subjects ranged from 21 to 61 years (mean = 37.1 years, standard deviation (SD) = 10.1, standard error of mean (SEM) = 2.9).

Each patient entering the study had been drug-free for at least 6 months. Informed consent was obtained after the experiments had been fully explained. The study was approved by the ethics committee of the Ullanlinna Sleep Disorders Clinic.

Table 1. The sleep variables recorded in the study and their values in the two recordings[a, b]

	1st recording		2nd recording	
Total movement time	0.07	(0.16)	1.03	(2.55)
Wake after sleep onset	36.99	(25.20)	36.45	(43.53)
Total sleep time[c]	386.11	(37.92)	390.30	(64.74)
Total slow wave sleep[c]	7.35	(3.55)	6.84	(5.75)
Total REM time[c]	21.84	(5.62)	23.03	(5.76)
Number of awakenings	11.00	(8.99)	7.27	(5.06)
Number of awakenings over 2 min	3.67	(2.54)	44.82	(137.40)
Time in bed	440.25	(31.47)	437.91	(36.28)
Sleep period time	418.89	(32.34)	425.96	(38.89)
Sleep efficiency index	0.88	(0.07)	0.89	(0.13)
Total wake time	13.12	(7.95)	11.43	(13.70)
Total sleep stage 1	3.69	(1.83)	3.60	(2.35)
Total sleep stage 2	59.85	(6.41)	57.81	(12.00)
Total sleep stage 3	7.08	(3.08)	6.02	(4.99)
Total sleep stage 4	0.27	(0.94)	0.82	(2.49)
Sleep latency	17.29	(13.63)	10.30	(11.74)
Slow wave latency	37.02	(21.60)	64.46	(77.00)
REM latency	92.73	(40.13)	85.70	(58.64)

[a] All total times are computed as percentages of sleep period time
[b] Values: mean (SD)
[c] Each variable was analysed also by three fractions of night separately

Results

No significant change was seen in any of the 27 polysomnographic variables analysed (Table 1).

The acrophase of the subjective sleepiness rhythm was significantly phase advanced immediately after cessation of light treatment ($t = 2.425$, $P < 0.05$). The mean level (mesor) of the sleepiness rhythm was significantly decreased ($t = 2.600$, $P < 0.05$). The amplitude of the sleepiness rhythm did not change significantly ($t = 0.288$, $P =$ ns). The mean sleepiness ratings were significantly lower at 8 and 10 a.m. measured immediately after cessation of light treatment ($t = 2.566$, $P < 0.05$, and $t = 3.406$, $P < 0.01$, respectively). A mean of the 10 other ratings (12, 14, 16, 18, 20, 22, 24, 2, 4, and 6 o'clock) did not differ significantly.

The HDRS scores were significantly reduced immediately after cessation of light treatment: mean = 11.4 (SD = 4.5) for baseline, mean = 2.7 (SD = 2.5) for one day after ($t = 5.482$, $P < 0.001$). The BDI scores were also significantly reduced immediately after cessation of light treatment: mean = 15.3 (SD = 8.5) for baseline, mean = 3.8 (SD = 4.2) for one day after ($t = 6.247$, $P < 0.001$). The effect remained for one week after cessation of light treatment (mean = 4.6 (SD = 4.0), $t = 5.182$, $P < 0.01$, and mean = 8.2 (SD = 10.0), $t = 3.008$, $P < 0.05$ for HDRS and BDI scores, respectively). The reductions in HDRS and BDI scores were intercorrelated ($r = 0.768$, $P < 0.05$).

The reduction in HDRS scores correlated significantly with the change in sleep latency ($r = -0.866$, $P < 0.01$). The change in slow wave latency correlated with the change in REM latency ($r = 0.888$, $P < 0.01$)

Predictors of response to light treatment were evaluated by multiple stepwise regression analyses. The best predictors of the variation of HDRS scores were the change in sleep latency and the change in amplitude of the sleepiness rhythm in a MGLH subset model (adjusted squared multiple R = 0.778, standard error of estimate (SEE) = 2.670; $t = -4.765$, $P < 0.01$, and $t = -4.619$, $P < 0.01$, respectively; analysis of variance (ANOVA): F = 18.502, $P < 0.01$). The HDRS score one week after cessation of treatment was best predicted by the change in acrophase of the subjective sleepiness rhythm, the change in sleep latency, slow wave latency, and in REM latency in another MGLH formula (adjusted squared multiple R = 0.985, SEE = 0.521; $t = -14.067$, $P < 0.01$, $t = 6.390$, $P < 0.05$, $t = 7.943$, $P < 0.05$, and $t = -5.321$, $P < 0.05$, respectively; ANOVA: F = 94.556, $P = 0.05$). The HDRS score one day after cessation of treatment was not predicted by any of the variables entered into regression analysis.

Subjects were divided into two subgroups in a cluster analysis. The sleep latency, slow wave latency and REM latency in the second recording contributed significantly to the results ($F = 67.514$, $P = 0.001$, $F = 418.708$, $P < 0.001$, and F = 30.273, $P < 0.001$, respectively). An association was seen between a faster response to light treatment and longer latencies in the second recording in the minor subgroup (Table 2). In this subgroup light treatment resulted in lengthening of slow wave latency and REM latency (opposite to the other subgroup) and in shortening of sleep latency (similar to the other subgroup).

Table 2. The variables contributing significantly to the result and their values in the cluster analysis[a, b]

Variable	Clusters		Control[c]
	No. 1	No. 2	
Sleep encephalography			
1st recording			
Sleep latency	14.0 (11.2)	33.9 (7.8)	7.8– 12.9 (6.0–9.6)
Slow wave latency	34.7 (18.7)	48.6 (25.5)	23.1– 56.3 (7.2–40.2)
REM latency	93.1 (41.2)	90.8 (19.4)	78.7–100.2 (19.5–44.2)
2nd recording			
Sleep latency	5.3 (15.1)	32.6 (2.1)	
Slow wave latency	30.2 (11.0)	218.6 (8.9)	
REM latency	62.6 (23.3)	189.8 (38.7)	
Subjective sleepiness rhythm			
Baseline			
Mesor (mm)	63.0 (10.6)	62.1 (9.8)	
Amplitude	25.3 (7.6)	22.7 (7.5)	
Acrophase (h)	–3.8 (1.8)	–4.3 (1.0)	
1 day after			
Mesor (mm)	53.1 (13.9)	52.0 (3.2)	
Amplitude	23.8 (7.3)	24.5 (3.6)	
Acrophase (h)	–2.4 (0.7)	–2.7 (1.3)	
HDRS score			
Baseline	11.2 (4.4)	12.5 (3.5)	
1 day after	3.0 (2.5)	1.0 (1.0)	
1 week after	4.3 (3.7)	6.0 (4.0)	

[a] $n = 10$ for cluster no. 1; $n = 2$ for cluster no. 2
[b] Values: mean (SD)
[c] Normal values adjusted for gender and age: range of mean (of SD); from Williams et al. (1974)

Discussion

Our results suggest that the antidepressant effect of light treatment is not reflected by significant changes in polygraphic sleep variables. This finding does not exclude, though, the possibility that sleep processes could influence the outcome of depressive symptoms. However, our findings do not support a recent hypothesis of the depressogenic nature of NREM sleep (Beersma and van den Hoofdakker 1992), since no significant changes or correlations were seen in any of the NREM sleep variables.

Bright light exposure has been used in treatment protocols to normalize circadian rhythm disturbances (Czeisler et al. 1990), and it has shown to have various psychophysiological effects in healthy volunteers (Clodore et al. 1990). The circadian sleepiness/alertness rhythm seems to be controlled independently of the sleep-wake cycle (Czeisler et al. 1980; Monk 1987). Our results support a hypothesis in which bright light treatment is thought to have a primary effect on the oscillator of sleepiness/alertness instead of a direct effect on the sleep-wake cycle (Folkard et al. 1985). The mean level of subjective sleepiness rhythm during 24 hours and the mean of sleepiness ratings in the morning were decreased significantly by light treatment. The latter finding suggests that light treatment could have a specific effect on the hypothesized oscillator.

Bright light exposure has been shown to be effective on resetting the circadian system without affecting various sleep processes (Czeisler et al. 1986; Dijk et al. 1989; Drennan et al. 1989). Our results suggest that the antidepressant effect of light could be mediated by mechanisms involving primarily the circadian subjective sleepiness rhythm processes in patients with winter depression. This hypothesis could be related to findings suggesting impaired serotonergic cell function in winter depression (Thompson et al. 1990). In our study, no relapse was seen 1 week after cessation of light treatment, which might be due to the high-intensity illumination (> 3500 lux at floor level) used and the design allowing patients to move freely.

The possibility that light might also have effect on the hypothesized process S could not be completely excluded by our results. Our results from cluster analysis revealed that sleep latency and REM latency were slightly shortened by light treatment in the major subgroup, and this finding could reflect a possible phase advance of the sleep-wake cycle. Our finding agrees with previous findings (Sack et al. 1986). Light treatment had no effect on slow wave latency in these patients. The resistance of stage 4 sleep to a treatment intervention was also documented in our study.

In the minor subgroup, however, all three latencies, except REM latency, remained abnormally long despite

bright light intervention. This unexpected finding suggests that the sleep-wake cycle was paradoxically phase delayed in these patients. Consequently, they might have become sleep deprived to a certain extent during the treatment protocol. It is not possible to conclude whether NREM sleep processes were affected directly by light treatment or sleep deprivation. In addition, all our findings according to phase shift of the circadian rhythms studied should be considered with caution, because no constant routine was applied.

In conclusion, although bright light reduced depression scores significantly, it did not have a major effect on sleep structure in patients with winter depression. Light intervention, however, reduced subjective sleepiness in the morning hours and phase advanced the circadian rhythm of subjective sleepiness significantly. These findings suggest that the antidepressant effect of bright light could be mediated primarily by mechanisms involving regulation of subjective sleepiness/alertness, and only secondarily, be sleep-related processes.

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