Brief report

Is dawn simulation effective in ameliorating the difficulty awakening in seasonal affective disorder associated with hypersomnia?

David H. Avery*, Mary E. Kouri, Kathleen Monaghan, Mary Ann Bolte, Carla Hellekson, Derek Eder

University of Washington School of Medicine, Department of Psychiatry and Behavioral Sciences, Harborview Medical Center, 325 Ninth Avenue, Seattle, WA 98104, USA

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Abstract

Background: Patients with winter depression (seasonal affective disorder, SAD) frequently complain of difficulty awakening in the morning. Dawn simulation has been found effective in treating SAD, but its effect on difficulty awakening has not been assessed. Methods: Fifty medication-free patients with SAD associated with hypersomnia were randomized to receive either 1 week of dawn simulation (250 lux) or a dim (0.2–2 lux) placebo signal. The patients assessed their level of drowsiness upon awakening during the baseline week and during the treatment week using the Stanford sleepiness scale (SSS). A psychiatrist rated difficulty awakening after the baseline week and after the treatment week. Results: Dawn simulation lowered both the difficulty awakening score ($P < 0.05$) and the SSS score ($P < 0.05$) compared to the placebo dawn signal. Limitations: Replication is necessary. No biological markers of circadian phase were measured. Conclusions: Compared to a placebo condition, dawn simulation appears effective in decreasing both prospectively assessed morning drowsiness and retrospectively assessed difficulty awakening. The symptom of difficulty awakening is consistent with the phase delay hypothesis of SAD. Assessment of difficulty awakening could prove useful in the evaluation of SAD. © 2002 Elsevier Science B.V. All rights reserved.

Keywords: Seasonal affective disorder; Dawn simulation; Drowsiness; Difficulty awakening

1. Introduction

Many patients with winter depression (Seasonal affective disorder, SAD) report excessive duration of sleep during the winter months (Rosenthal et al., 1984; Rosenthal, 1993). However, many of these patients only report the longer sleep duration on weekends and force themselves out of bed during the weekdays despite feeling excessively drowsy.

Dawn simulation is a low illuminance light that gradually increases in intensity before awakening...
Dawn simulation has been found effective in treating the symptoms of SAD compared to placebo light signals (Avery et al., 1993, 1994), but the effectiveness of dawn simulation in improving the symptom of difficulty awakening has not been studied.

The primary goals of this paper are: (1) to assess the validity of the retrospective difficulty awakening assessment by comparing it with morning drowsiness, prospectively recorded in a daily log by the subject; (2) to assess the interrater reliability of the difficulty awakening assessment; (3) to determine whether dawn simulation is effective in improving the difficulty awakening assessment score and prospective assessment of morning drowsiness in patients with SAD.

2. Methods

Data were collected during three separate studies; two previous published studies (Avery et al., 1993, 1994) and previously unpublished data, each conducted during the fall–winter months. Subjects were recruited through advertisements and publicity concerning winter depression. Subjects fulfilled criteria for major depressive episode according to DSM-III-R (American Psychiatric Association, 1987) as well as primary affective disorder according to the Feighner criteria (Feighner et al., 1972). Subjects also fulfilled Rosenthal criteria for seasonal affective disorder (Rosenthal et al., 1984). All subjects reported fall–winter depression that occurred during at least two consecutive winters, and all reported hypersomnia as part of their winter depression. Hypersomnia was determined by retrospective self-reports, and was defined as sleeping at least 1 hour longer during fall–winter compared to spring–summer. Subjects were free of any psychotropic medication for at least 4 weeks before the study, and none had psychosocial variables that accounted for their seasonal changes in mood and sleep. All gave written informed consent.

In each study, data were collected during a baseline week in which no light treatment was administered. Subjects were asked to sleep only between the hours of 9:00 p.m. and 6:00 a.m. and to keep a log of their sleep and drowsiness in the morning.

At the beginning of the second week, subjects were randomized to one of two treatment conditions: dawn simulation or a dim ‘placebo’ signal. The three studies had similar basic designs. The bedrooms were required to be dark. The subjects were asked to turn off any light source, such as a hall light or night light, and to cover their windows with black plastic if necessary. Typically, these hypersomnic subjects did not awaken during the night to use the bathroom. If they did, they were asked to use only a night light in the bathroom. They were asked to set an alarm for 6:00 a.m. If the subject awakened before 6:00 a.m., they were to avoid looking at the lights, close their eyes, and go back to sleep. Subjects were asked to avoid morning sunlight before 8:00 a.m. For each study, the dim placebo signal and the white treatment dawn used identical fixtures placed 4 feet from the subject’s pillow. Light intensity at this distance was confirmed with a photometer.

The placebo condition in each study was a dawn simulation signal that began at a very dim illumination and gradually increased in intensity, but peaked at very low illumination compared to the dawn condition. The placebo conditions were not identical in the three studies. In the first study (Avery et al., 1993), the placebo signal was a 30-min dawn from 5:30 to 6:00 a.m. peaking at 0.2 lux, an intensity similar to moonlight. The second study (Avery et al., 1994) used a 1.5-h dim red dawn, a placebo signal that began at 4:30 a.m. and peaked at 2 lux at 6:00 a.m. The placebo signal in the third study consisted of a 1.5-h dawn from 4:30 to 6:00 a.m., with a peak intensity of 0.5 lux.

The dawn simulation treatment in each study was a white light which gradually increased in intensity, peaking significantly brighter than the placebo dawn but not as bright as typical bright light therapy (> 2000 lux). In the first study (Avery et al., 1993), the dawn simulation was a 2-h dawn from 4:00 to 6:00 a.m. peaking at 250 lux (approximating room light). The light used was a 75-watt incandescent reflector flood light, as described above. In the second study (Avery et al., 1994), a 1.5-h dawn from 4:30 to 6:00 a.m. peaking at 250 lux was used. The
third study used a 2-h dawn from 4:30 to 6:00 a.m. peaking at 250 lux.

The difficulty awakening assessment we were testing consisted of a five point rating scale: 0 = wakes up without an alarm; 1 = needs an alarm to wake up on time; 2 = moderate drowsiness upon awakening; 3 = severe drowsiness when the alarm goes off, major effort needed to get out of bed; and 4 = often falls back to sleep after the alarm goes off. Each patient was interviewed and rated by a trained psychiatrist who was blinded to the patient’s treatment group at screening and at the end of each week during the study. A second psychiatrist (DHA) who was blind to the first psychiatrist’s ratings evaluated a subset.

Morning drowsiness was prospectively logged by the patients using the eight-point Stanford sleepiness scale (SSS) (Hoddes et al., 1973): 1 = feeling active and alert, wide awake; 2 = functioning at a high level, but not at peak, able to concentrate; 3 = relaxed, awake but not at full alertness, responsive; 4 = a little foggy, not at peak, let down; 5 = foggy, beginning to lose interest in staying awake, slowed down; 6 = sleepy, prefer to be lying down, fighting sleep, woozy; 7 = almost in reverie, sleep onset soon, lost struggle to stay awake; 8 = asleep, unable to wake up. The SSS scores upon awakening for the last 3 days of the baseline week were averaged into a single score. Treatment weeks were processed in an identical manner.

Subjects who experienced spontaneous early morning awakening (awakening before their desired wake-up time) during the baseline or treatment period were excluded from analyses. Subjects who did not complete the SSS data for the last 3 days of the baseline week were also excluded. The last 3 days of the baseline week were chosen to minimize the effects of sleep deprivation on morning drowsiness. During the treatment weeks, if a subject was missing a SSS score for only 1 of the last 3 days, the next most recent day was used.

To assess the efficacy of treatments, the averaged SSS scores upon awakening for the treatment week were subtracted from the scores from the baseline week, generating a score for the difference in drowsiness during the treatment compared to baseline.

Validity was assessed the Spearman rho test. Interrater reliability was assessed using Cohen’s kappa. Improvements in difficulty awakening and morning drowsiness scores were examined using the Wilcoxon sign-rank test. Between group comparisons were analyzed using the Mann–Whitney U-test. For hypotheses that predicted a null hypothesis, such as baseline comparisons, two-tailed $P$-values were used. Since dawn simulation was predicted to be superior to the placebo condition, one-tailed $P$-values are reported for the comparisons of improvement.

### 3. Results

The three dawn simulation–placebo studies were not significantly different in the baseline difficulty awakening scores or the change in the difficulty awakening scores. In all three studies, the post-treatment difficulty awakening scores were significantly lower than the baseline scores for each dawn simulation treatment group (one-tailed, $P < 0.05$). Because the patient selection, study designs, difficulty awakening scores and outcomes were similar across all three studies, the data from the three studies were consolidated into one dawn simulation group and one placebo group for the following analyses.

The final analyses included 50 subjects; 28 (5 men, 23 women) who received dawn simulation; 22 (2 men, 20 women) who received a placebo dawn. There were no significant differences in age (dawn, 36.3±9.89; dim placebo signal, 38.6±9.1), gender, difficulty awakening or morning drowsiness at baseline between the two study groups. Two subjects had missing difficulty-awakening data.

Difficulty awakening correlated significantly with morning drowsiness during the treatment week for both the placebo group ($r = 0.627; N = 20$; one-tailed $P < 0.01$) and the dawn simulation group ($r = 0.319; N = 28; P < 0.05$). No correlation was found during the baseline week for either group. Two psychiatrists (D.H.A. and M.A.B.) evaluated a subset of 41 SAD subjects in separate interviews. The interrater reliability of the post-treatment difficulty awakening scores showed a Cohen’s kappa of 0.64.

Dawn simulation resulted in a significantly greater
Table 1
Mean difficulty awakening and Stanford Sleepiness Scale scores for all treatment groups at baseline and after 1 week of light treatment, grouped by study

<table>
<thead>
<tr>
<th></th>
<th>N</th>
<th>Difficulty awakening</th>
<th>Stanford Sleepiness Scale</th>
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<th></th>
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<td></td>
<td></td>
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<td>Treatment</td>
<td>Difference</td>
<td>Baseline</td>
<td>Treatment</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Study 1</td>
<td>7</td>
<td>3.57</td>
<td>2.29</td>
<td>1.28 (^a)</td>
<td>5.71</td>
<td>5.42</td>
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<td>Study 2</td>
<td>7</td>
<td>3.67</td>
<td>2.83</td>
<td>0.84 (^a)</td>
<td>6.45</td>
<td>5.12</td>
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<tr>
<td>Study 3</td>
<td>8</td>
<td>3.50</td>
<td>2.57</td>
<td>0.93 (^a)</td>
<td>6.75</td>
<td>6.17</td>
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<tr>
<td>Mean</td>
<td></td>
<td>3.57±0.51</td>
<td>2.55±1.05</td>
<td>1.05±1.10 ^a</td>
<td>6.33±1.20</td>
<td>5.60±1.56</td>
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<td><strong>Dawn simulation</strong></td>
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<td></td>
</tr>
<tr>
<td>Study 1</td>
<td>7</td>
<td>3.29</td>
<td>1.29</td>
<td>2.00 ^a</td>
<td>6.05</td>
<td>5.14</td>
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<tr>
<td>Study 2</td>
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<td>0.89</td>
<td>2.22 ^a</td>
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<td></td>
<td>3.43±0.92</td>
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<td>1.75±1.48 ^a ^b</td>
<td>6.60±0.97</td>
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</tr>
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</table>

\(^a\) Pre-post difference is significant (Wilcoxon, one-tailed \(P < 0.05\)).
\(^b\) Dawn simulation significantly greater than placebo signal (Mann–Whitney, one-tailed \(P < 0.05\)).

decrease in both the difficulty awakening assessment (\(N = 48\); one-tailed \(P < 0.05\)) and SSS (\(N = 50\), one-tailed \(P < 0.05\)) compared to the placebo signals. (See Table 1)

4. Discussion

Some methodological issues deserve comment. Because the sample of SAD subjects was selected for hypersomnia, all but two subjects began the study with a difficulty awakening rating score of 3 or 4. The lack of significant correlation between difficulty awakening and morning drowsiness during the baseline week is probably due to the narrow range of the scores at baseline. Post-treatment scores varied considerably within the treatment groups, allowing significant correlations to be shown.

Subjects who failed to record SSS scores during the baseline week were excluded. It is possible that patients who do not comply with the study protocol are in some way different from patients who do comply, but it is difficult to speculate on the nature of those differences, or what, if any, impact they would have on the results. Subjects who experienced early morning awakening during the treatment week were excluded. Their exclusion would have biased against finding a statistically significant post-treatment decrease in morning drowsiness in the dawn simulation group; dawn simulation may sometimes cause early morning awakening.

Lewy et al. (Lewy et al., 1987b) has hypothesized that SAD symptoms result from a phase-shift of their circadian rhythms; in two separate studies the dim light melatonin onset (DLMO) is delayed in SAD patients compared to controls (Lewy et al., 1987a; Sack et al., 1990, 2000). In a constant routine study, the DLMO (Dahl et al., 1993), temperature rhythm and cortisol rhythms (Avery et al., 1997a,b) in SAD patients were found delayed compared to nondepressed controls even though the sleep–wake cycle was standardized (a 6:00 am wake up timer for 6 days) prior to the constant routine for both groups. In particular, at baseline, the mean time of the temperature minima was 3:16 am in controls and 5:42 am in the SAD group; with morning bright light therapy (2500 lux × 2 h from 6:00 am to 8:00 am), the temperature minimum in the SAD group phase advanced to 3:36 p.m. Sleep propensity studies show that the low temperature trough is a time of increased sleep propensity (Shochat et al., 1997) and marked drowsiness (Monk et al., 1989). The SAD patient sometimes expresses the complaint of increased drowsiness upon awakening by saying ‘it feels like the middle of the night’ when their alarm clock sounds. The data showing phase delays of circadian
rhythms relative to sleep suggest that, physiologically, 6:00 am may be the middle of the night for SAD patients before treatment. The lack of morning light may allow the circadian rhythms to delay and create a lack of synchrony between these circadian rhythms and sleep that is analogous to jet lag. Unlike jet lag, during winter depression, the desynchrony may persist for weeks or months. The desynchrony between the sleep–wake cycle and other circadian rhythms is a plausible candidate for explaining the difficulty awakening and increased morning drowsiness of the SAD patient.

Light is the main synchronizer of circadian rhythms. Bright light in the morning phase advances (shifts earlier) the circadian rhythms in controls (Czeisler et al., 1986, 1989) and in SAD patients (Lewy et al., 1987a; Sack et al., 1990, 2000; Dahl et al., 1993; Avery et al., 1997a,b). Dawn simulation has been shown to phase-advance melatonin onset (Danilenko et al., 2000). Theoretically, dawn simulation would also shift the temperature minimum to an earlier time resulting in less drowsiness upon awakening. Together, the circadian data suggest construct validity for the difficulty awakening symptom, but the lack of objective circadian measures in this study limit our interpretation.

The difficulty awakening assessment seems to have reasonable interrater reliability and validity and may be useful for evaluating and monitoring improvement in patients with SAD. Dawn simulation appears effective in improving the difficulty awakening scores and the morning SSS scores.

Acknowledgements

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References


