The Treatment of Early-Morning Awakening Insomnia With 2 Evenings of Bright Light

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Study Objective: To assess the effectiveness of brief bright-light therapy for the treatment of early-morning awakening insomnia.

Participants: Twenty-four healthy adults with early-morning awakening insomnia were assigned to either the bright-light condition (2,500-lux white light) or the control (dim red light) condition.

Measurements and Results: The circadian phase of rectal temperature and urinary melatonin rhythms were assessed with 26-hour constant routines before and after 2 evenings of light therapy. Sleep and daytime functioning were monitored using sleep diaries, activity monitors, and mood scales before light therapy and for 4 weeks during the follow-up period. While there were no significant circadian phase changes in the dim-light control group, the bright-light group had significant 2-hour phase delays of circadian temperature and melatonin rhythm. Compared to pretreatment measures, over the 4-week follow-up period, the bright-light group had a greater reduction of time awake after sleep onset, showed a trend toward waking later, and had a greater increase of total sleep time. Participants in the bright-light condition also tended to report greater reductions of negative daytime symptoms, including significantly fewer days of feeling depressed at the 4-week follow-up, as compared with the control group.

Conclusion: Two evenings of bright-light exposure phase delayed the circadian rhythms of early-morning awakening insomniacs. It also improved diary and actigraphy sleep measures and improved some indexes of daytime functioning for up to 1 month after light exposure. The study suggests that a brief course of evening bright-light therapy can be an effective treatment for early-morning awakening insomniacs who have relatively phase advanced circadian rhythms.

Key Words: Early morning awakening insomnia, circadian rhythm, body temperature, melatonin, bright light.

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INTRODUCTION

Recent American sleep polls indicate that up to approximately 25% of adults aged 18 years and older report waking too early and being unable to fall back to sleep, with the incidence increasing with age.1,2 In these polls, early-morning awakenings are also associated with decreased total sleep time, excessive daytime sleepiness, and fair to poor health.1 In an earlier study, we found that participants who experienced early-morning awakening insomnia had significantly advanced circadian temperature and melatonin rhythms compared to a control group of age-matched good sleepers.3 The average temperature minimum (Tmin) of participants with early-morning awakening insomnia occurred at 00:20 AM, more than 3 hours earlier than in the control group, and the mean urinary melatonin onset occurred at 8:30 PM, more than 2 hours earlier than in the control group. The average wake-up time of 4:49 AM was also significantly advanced compared to the control group, who awoke on average, at 7:24 AM. The total sleep time of 5.5 hours was significantly less than in the control group.

Evening bright-light therapy has been found to be effective in phase delaying the circadian parameters and the sleep-wake cycle of individuals with early-morning awakening insomnia. A number of single case studies of individuals with early-timed sleep and circadian rhythms have demonstrated the efficacy of evening bright-light pulses (2500-10,000 lux) being administered between 8:00 PM and 11:00 PM.4-6 Evening bright light has also been administered to older subjects (60 years and older) who had sleep-maintenance insomnia. A 2-hour light stimulus of 4000 lux was administered to participants in their own home environments, between the hours of 8:00 PM and 11:00 PM, for 12 consecutive days.7 Before light exposure, participants had a somewhat early average Tmin at 3:11 AM, a wake-up time of 7:06 AM, and less than 6 hours total sleep time. Following light exposure, the average Tmin of the bright-light group was delayed by more than 2 hours, wake-up time was delayed by 18 minutes, and total sleep time increased by 45 minutes. A control group exposed to dim red light had no significant changes in circadian or sleep parameters. In a more recent study, participants with sleep-maintenance insomnia had an average Tmin at 2:59 AM, a final wake-up time at 6:32 AM, and less than 6 hours total sleep time. They were exposed to evening bright light (4000 lux) in their home environment for 2-hour intervals between 8:00 PM and 11:00 PM.8 Bright-light therapy was administered over 11 to 13 consecutive days (acute phase) and then twice weekly for a 3-month period (maintenance phase). Following the acute phase, evening bright light produced a significant phase delay of the Tmin of 94 minutes during the acute phase and of 47 minutes relative to baseline in the maintenance phase. There was a nonsignificant delay of wake-up time of 32 minutes during the acute phase and the maintenance period; however, total sleep time remained unchanged after light exposure. Therefore, eve-
ing bright-light therapy has had mixed success in treating sleep maintenance insomnia.

In an earlier pilot study, we successfully treated a group of early-morning awakening insomniacs with just 2 evenings of bright-light stimulation. Subjects were exposed to a bright-light stimulus of 2500 lux from 8:00 PM to midnight on 1 evening and from 9:00 PM to 1:00 AM on the following evening. Immediately after light exposure, the average Tmin was significantly delayed from 2:31 AM to 4:22 AM, and the average dim-light melatonin onset (DLMO) was delayed by about 2 hours. In the 5 days following bright-light exposure, subjects’ average baseline wake-up time of 4:59 AM was significantly delayed by more than 1 hour, and total sleep time was increased by more than 1 hour to 386 minutes. We suggest that the discrepancy between the success of this study and the failure of the Suhner et al study to improve sleep arises from our selection of early-morning awakening insomniacs with relatively earlier Tmin times and wake-up times. However, our earlier pilot study lacked a control group and assessed acute sleep improvement only in the week following treatment.

The aim of the present study was to further explore the effectiveness of only 2 evenings of bright-light therapy for early-morning awakening insomnia. In this study, we used a control group exposed to dim red light and examined posttreatment effects over a 4-week period.

MATERIALS AND METHOD

Participants

Requests were made on radio and in local newspapers for people with early-morning awakening insomnia of at least 6 months duration to participate in a study involving assessment and treatment. Following an initial telephone interview, 230 respondents were sent a 1-week sleep-wake diary and sleep questionnaire. Selection criteria, based on returned questionnaires and diaries included (1) mean lights out time earlier than 11:00 PM, (2) mean sleep-onset latency less than 15 minutes, (3) mean nocturnal wake after sleep onset (WASO) of less than 20 minutes in the first 3 hours of the sleep period, (4) spontaneous final wake-up time within 6 hours of initial sleep onset and generally before 5:00 AM, (5) total sleep time of less than 6 hours, and (6) daytime symptoms of tiredness and irritability. All respondents were also to be in self-reported good health, have an average or below-average intake of alcohol or caffeine, not be using hypnotic medication, and have no clinical signs of obstructive sleep apnea, restless legs syndrome, or periodic limb movements in sleep. Essentially, the selection criteria were designed to yield participants with chronic primary insomnia with a principal difficulty of premature early final awakening (“terminal insomnia”). Of the 35 respondents who met the criteria, 6 declined to participate further in the study, and 4 were unable to participate for various personal reasons. Twenty-five early-morning awakening insomniacs (12 men, 13 women) with an average age of 51.2 years (age range 36-68 years) commenced the study.

Using general guidelines of cut-off scores for the Beck Depression Inventory, 16 participants had scores of less than 9 (minimal range), and 9 participants had scores between 10 and 16, indicating mild depression. There was no difference between groups on Beck Depression Inventory score. Consecutive participants were assigned alternately to the bright-light and dim-light conditions based on their order of presentation in the study. There were 13 participants in the bright-white-light group and 12 in the control (dim-red-light) group. Because 1 male participant could not continue after the initial polysomnogram due to family problems, 13 participants in the white-light group and 11 in the control group completed the study. Because of the allocation method, sex and age were not balanced between the 2 conditions. However, recent research suggests there is no sex or age difference in melatonin-suppression sensitivity to light exposure. Therefore, it seems unlikely that participant differences would confound light-treatment differences. Furthermore, we were mainly testing differential changes over treatment within participants rather than main effects of group differences in sleep.

All participants signed informed consent forms prior to baseline recording. Ethical approval was obtained from the Social and Behavioural Research Ethics Committee at Flinders University.

APPARATUS AND MATERIALS

Sleep-Wake Diary

The sleep-wake diary required the recording of lights out time, estimated sleep-onset latency, number and length of awakenings during the night, and final wake-up time. Mean WASO, final wake-up times, and total sleep times were calculated for each week.

Questionnaires

Subjects completed a daytime symptoms questionnaire developed at the University on which they indicated on a 5-point scale how many days during the preceding week they felt daytime drowsiness, irritability, and depression. They also indicated how much more sleep they felt they needed each night.

Actigraphy

Subjects wore a Gähwiler Z80, 32k actigraph (Singh-Medical, Stafa, Switzerland) on their nondominant hand at night. The signals were processed on line with frequent sampling (0.1 seconds) and aggregated in 30-second epochs. Actigraphy sleep-onset time was estimated as the start of the first period of at least 15 consecutive minutes of no wrist activity after lights-out time. Amount of WASO was estimated from an algorithm using the number of epochs with movement from initial sleep onset to final wake-up time. Total sleep time was estimated as the time from estimated sleep onset to the time of final wake up (as indicated by the subject’s event marking) minus WASO. While actigraphic sleep estimates are only moderately related to polysomnographic sleep in insomniacs, it has been suggested that actigraphy reliably estimates polysomnographic changes over time within individual insomnia subjects who are undergoing therapy to improve sleep.

Core Body Temperature

Throughout the 26-hour constant routines, rectal temperature was measured at 1-minute intervals using an indwelling Yellow Springs Instruments (Yellow Springs, Ohio) Series 400 temperature thermistor, inserted 10 to 15 cm into the rectum. The thermistor was connected to an Apple Macintosh II fx computer through an opto-isolation interface box. All information was recorded using the Labview 2 (National Instruments, Austin, Tex) tempera-
ture-recording program. Recordings were continuously displayed in order to monitor for any possible thermistor failures.

**Melatonin Circadian Rhythm Measures**

Under dim-light conditions, urinary melatonin onset was assessed from urine samples collected every 2 hours during the 26-hour constant routine and from 4:00 pm to midnight in the subjects’ homes at the end of the first week and the fourth week of the follow-up period. For all urine samples, the total volume of urine was measured to the nearest 1 ml, a 5-ml sample was then mixed with boracic acid, and the sample was frozen. The urine samples were analyzed at the University of Adelaide, Department of Obstetrics and Gynaecology, for the major melatonin metabolite concentration using the 6-sulphatoxy melatonin radioimmunoassay.15 From the raw data, a value that was 100% above the baseline values was established. The time of DLMO was then determined by interpolating between the first data value, which consistently exceeded this value, and the previous 2-hourly value.

**Light Boxes**

The bright-light box comprised 8 incandescent 75-watt light globes behind a translucent acrylic screen and produced 2500-lux intensity measured at a distance of 70 cm. The dim-light box comprised 4 incandescent 40-watt red light globes behind a translucent acrylic screen, which produced < 100 lux measured at a distance of 70 cm. Red light was used in the control condition because it combined subjective treatment credibility with lack of circadian-delaying capacity.16

**PROCEDURE**

**Baseline**

After a full explanation of the study and a tour of the sleep laboratory, participants completed the Beck Depression Inventory. During the following week, they completed a sleep-wake diary and wore a wrist activity monitor each night. At the completion of the baseline week, participants slept overnight in the laboratory before commencing a 26-hour constant wakefulness routine. At approximately 7:30 AM, a rectal thermistor was inserted, and the 26-hour constant routine commenced at 8:00 AM until 10:00 AM the next morning. During the constant routine, participants remained in the near supine position, awake and engaging in minimal activity such as reading, listening to music, or watching video films. The ambient temperature was kept constant at 22°C, and room illumination was kept at less than 50 lux, as measured at the participant’s head position. Snacks of approximate equal caloric

<table>
<thead>
<tr>
<th>Condition</th>
<th>Temperature minima Before</th>
<th>After</th>
<th>Main treatment effect</th>
<th>Interaction effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bright light</td>
<td>2:03am (1.81)</td>
<td>4:27am (2.20)* †</td>
<td>8.04, 1.22, .009</td>
<td>9.39, 1.22, .006</td>
</tr>
<tr>
<td>Dim light</td>
<td>1:25am (2.59)</td>
<td>1:13am (3.0)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Data are presented as mean (SD), with analysis of variance main and interaction effects.

*Significant difference from baseline (<.05).
† Significant difference between groups (<.05).

**RESULTS**

**Circadian Parameters**

To examine circadian variation of rectal temperature, the 1-4 minute values were aggregated into 48 consecutive 30-minute averages. A best-fit 2-component (24-hour plus 12-hour periods) cosine function was calculated for the temperature data for each subject before and after light therapy using the general curve fitting routine in the software program Kaleidagraph V2.0 (Synergy Software, Reading, Penn). The average variance accounted for by the fitted cosine curves before and after light therapy was very high at 94.6% and 95.5%, respectively. The time of Tmin for each
participant from each constant routine was determined as the minimum of each fitted cosine curve.

Table 1 shows the mean and temperature minimum times before and after intervention for the 2 groups. A 2-way mixed-model analysis of variance (ANOVA) showed that there was a significant pretreatment and posttreatment time effect ($F_{1,22} = 8.04, P < .01$) and a significant interaction effect ($F_{1,22} = 9.39, P = .006$). There was no change for the dim-light group and a significant delay of over 2 hours for the bright-light group.

Figure 1 shows the DLMO means before and after treatment, as well as those at the ends of the follow-up week 1 and week 4 for the 2 groups. The 2-way mixed-model ANOVA of the DLMO before and after light exposure showed no main effects ($P > .5$) but did show a significant interaction effect ($F_{1,15} = 21.19, P = .0003$). The significant interaction appears to come mainly from the delay in the bright-light group ($t_{15} = 4.23, P = .0014$), with a possible contribution from some phase advance in the dim-light group ($t_{15} = 2.29, P = .051$). To explore if this differential phase-delay effect was maintained during the follow-up period, 2-way ANOVAs were applied between the pre-exposure condition and at the end of each follow-up weeks 1 and 4. A comparison of the pre-exposure condition and follow-up week 1 showed a significant interaction effect between groups and time ($F_{1,15} = 8.97, P = .009$), with a posthoc significant difference at follow-up week 1 between groups ($t_{15} = 2.18, P = .05$). The interaction between pretreatment exposure and follow-up week 4 continued to be significant ($F_{1,15} = 7.35, P = .02$), with a significant posthoc difference between groups at week 4 ($t_{15} = 2.61, P = .02$).

To further test the maintenance of the phase differences between the groups after light treatment, the mean DLMOs obtained immediately after light exposure and at follow-up weeks 1 and 4 were compared between groups. The 2-way mixed-model ANOVA showed a significant overall group difference ($P < .002$) and a significant overall ($P < .01$) phase advance but no interaction effect. Therefore, despite some phase advance of both groups, the difference in DLMO between groups was maintained across the follow-up period.

### Sleep Variables

The average weekly sleep parameters of sleep-onset time, WASO, final wake-up time, and total sleep time were calculated from the sleep-wake diaries and activity-monitor data. The sleep parameters were averaged separately for each subject over the 7-day period prior to light therapy and for the 7-day periods for the first and fourth follow-up weeks. Before treatment, there were no significant differences between groups for these diary or activity-monitor sleep variables. Changes in sleep parameters from before light therapy, at the 1-week follow-up, and at the 4-week follow-up period were analyzed using 2-way mixed-model ANOVA. Planned comparisons were used to test differences between the means of significant interaction and main effects.

There were no significant changes in sleep-onset times across weeks or any differences between the 2 groups. The overall average sleep-onset time was approximately 11:00 pm.

Table 2 presents the mean sleep-diary and activity-monitor WASO for the bright-light and dim-light groups. The sleep-diary data indicated a significant decrease of WASO ($F_{1,22} = 13.6, P < .0002$) overall, with the bright-light group experiencing about 1 hour less WASO and the dim-light group experiencing about 25 minutes less than their baseline values. However, the interaction effect testing the greater decrease of WASO in the bright-light group did not reach significance ($P = .11$).

Although the activity-monitor data similarly indicated an overall significant decrease in WASO over time ($F_{1,22} = 6.8, P = .003$), there was a significant interaction effect between group and time ($F_{1,10} = 6.3, P = .005$). While there was no significant change in WASO for the dim-light group across the weeks, there was a significant decrease in WASO at the 1-week follow-up for the bright-light group ($t_{10} = 3.1, P = .006$) and a significant group difference at the 4-week follow-up period ($t_{10} = 3.24, P = .005$).

Table 3 presents the mean sleep-diary and activity-monitor data for the final wake-up time for the bright-light and dim-light groups. There was a significant delay of sleep diary final wake-up time across the follow-up weeks ($F_{2,24} = 13.69, P = .01$); however, the somewhat greater delay of the bright-light group of about 55 minutes did not produce a significant interaction effect ($P = .17$) between groups.

Comparison of the activity monitor data for the final wake-up time between baseline and the follow-up period showed a marginally significant main treatment effect ($P = .07$). More importantly, the time-by-group interaction effect was significant ($F_{2,19} = 3.53, P = .04$). There was a significant delay of wake-up time in the bright-light group from baseline to follow-up week 1 of about 19 minutes ($t_{10} = 2.42, P = .02$). However, the interaction effect would also have been driven by the significant advance of final wake time in the dim-light group from baseline to follow-up week 4 ($t_{10} = 2.64, P = .01$) of 22 minutes.

Total sleep time was calculated by subtracting the final wake-up time and WASO from sleep-onset time. Table 4 presents the mean sleep-diary and activity-monitor data for the total sleep time for the bright-light and dim-light groups. Subjective data for the total sleep time showed an overall significant increase across weeks ($F_{2,24} = 20.5, P < .0001$), with the bright-light group showing a greater increase than the dim-light group and follow-up, as indicated by a significant 2-way interaction effect ($F_{2,24} = 7.3, P = .002$). At the end of the 4-week follow-up period, the bright-light group had 90 more minutes of sleep than at baseline ($t_{15} = 8.11, P = .0001$) and 45 more minutes of sleep than the dim-light group at the 4-week follow-up period ($t_{22} = 3.5, P = .002$).

Actigraphy data regarding total sleep time indicated there was no overall significant main effect ($P > .05$); however, the interac-
tion effect was marginally significant ($F_{2,19} = 2.97, P = .06$). The 70-minute difference between groups at the 4-week follow-up period was significant ($t_{19} = 2.0, P = .03$).

**Daytime Symptoms**

To examine changes of daytime symptoms, subjects completed a series of questionnaires before light exposure and at the end of the follow-up weeks 1 and 4 (Table 5). Data obtained from these questionnaires were analyzed using 2-way mixed-model ANOVAs. To determine differences between the means for significant interactions, main effects, or both interactions and effects, planned comparisons were used.

Most daytime symptoms showed overall decreases: the number of days subjects had feelings of drowsiness ($F_{2,42} = 18.15, P < .0001$), irritability ($F_{2,38} = 4.67, P = .01$), and the need for more sleep ($F_{2,40} = 10.25, P = .0003$). However, despite the decreases being greater on average for the bright-light group, only the interaction effect for need for more sleep approached significance ($P = .13$), and the number of depressed days was significant ($F_{2,38} = 3.07, P = .05$), indicating a relatively greater decrease in the number of depressed days in the bright-light group.

**DISCUSSION**

Following just two 4-hour exposures to evening bright white light, individuals with early-morning awakening insomnia had average phase delays of 2 hours of the temperature and melatonin rhythms. The greater phase delay of the bright-light group, although diminished somewhat, continued to be significantly great-

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**Table 2**—Sleep Diary and Actigraphy Data for Wake After Sleep Onset Before Light Exposure and After Follow-Up Week 1 and Follow-Up Week 4 for the Bright-Light and Dim-Light Groups

<table>
<thead>
<tr>
<th>Condition</th>
<th>WASO, min</th>
<th>Main treatment effect</th>
<th>Interaction effect</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before</td>
<td>FU1</td>
<td>FU4</td>
</tr>
<tr>
<td>Sleep Diary</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bright light</td>
<td>80.19 (59.3)</td>
<td>45.10 (27.9)</td>
<td>20.94* (21.1)</td>
</tr>
<tr>
<td>Dim Light</td>
<td>71.42 (47.7)</td>
<td>60.20 (39.5)</td>
<td>47.06 (38.8)</td>
</tr>
<tr>
<td>Actigraphy</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bright light</td>
<td>94.45 (53.3)</td>
<td>69.11 (34.1)</td>
<td>55.77** (21.1)</td>
</tr>
<tr>
<td>Dim Light</td>
<td>78.08 (21.1)</td>
<td>71.27 (29.1)</td>
<td>79.70 (30.9)</td>
</tr>
</tbody>
</table>

Data are presented as mean (SD), with analysis of variance main and interaction effects.

*Significant difference from baseline (<. 05).
† Significant difference between groups (<. 05).
FU1 refers to follow-up week 1; FU4, follow-up week 4.

**Table 3**—Sleep Diary and Actigraphy Data for Final Wake-Up Time Before Light Exposure and After Follow-Up Week 1 and Follow-Up Week 4 for the Bright-Light and Dim-Light Groups

<table>
<thead>
<tr>
<th>Condition</th>
<th>Final Wake-Up Time</th>
<th>Main treatment effect</th>
<th>Interaction effect</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before</td>
<td>FU1</td>
<td>FU4</td>
</tr>
<tr>
<td>Sleep Diary</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bright Light</td>
<td>5:30 (1.41)</td>
<td>6:19 (1.15)</td>
<td>6:27* (0.87)</td>
</tr>
<tr>
<td>Dim Light</td>
<td>5:53 (0.74)</td>
<td>6:19 (0.44)</td>
<td>5:58 (0.67)</td>
</tr>
<tr>
<td>Actigraphy</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bright Light</td>
<td>6:16 (32.4)</td>
<td>6:35* (35.9)</td>
<td>6:36 (73.3)</td>
</tr>
<tr>
<td>Dim Light</td>
<td>6:14 (0.54)</td>
<td>6:32 (0.46)</td>
<td>5:52* (0.59)</td>
</tr>
</tbody>
</table>

Data are presented as mean (SD), with analysis of variance main and interaction effects.

*Significant difference from baseline (<. 05).
FU1 refers to follow-up week 1; FU4, follow-up week 4.
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er than that of the dim-light group throughout the 4-week follow-up period. This last conclusion based on DLMO data collected by participants in their homes needs to be treated with a degree of caution. Although they were instructed to remain in relatively dim indoor light on these collection nights, there was no monitoring of light levels. Because some indoor lighting (> 100 lux) has been shown to produce mild melatonin suppression, some melatonin data values may be underestimated in the case of inadvertent higher-intensity light exposure. However, this is most likely to be an unsystematic source of error in DLMO measures and work against the observed statistical conclusions.

The circadian-rhythm changes following bright-light therapy coincided with improvements in sleep measures. The evening bright-light group showed a significantly greater decrease of WASO, a tendency for a later final wake-up time, and greater increase in total sleep time than the nondelayed control group. In addition, negative daytime symptoms tended to reduce more following bright-light therapy, with that trend being significant for “number of days depressed.”

In our initial study in which early-morning insomniacs were

Table 4—Sleep Diary And Actigraphy Data for Total Sleep Time Before Light Exposure and After Follow-Up Week 1 and Follow-Up Week 4 for the Bright-Light and Dim-Light Groups

<table>
<thead>
<tr>
<th>Condition</th>
<th>Total Sleep Time, min</th>
<th>F values</th>
<th>Main treatment effect</th>
<th>Interaction effect</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Before</td>
<td>FU1</td>
<td>FU4</td>
<td>M</td>
</tr>
<tr>
<td>Sleep Diary</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bright light</td>
<td>320.1</td>
<td>358.6</td>
<td>411.8* †</td>
<td>20.5</td>
</tr>
<tr>
<td>(42.5)</td>
<td>(58.2)</td>
<td>(37.1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dim light</td>
<td>343.2</td>
<td>366.5</td>
<td>366.3</td>
<td></td>
</tr>
<tr>
<td>(34.9)</td>
<td>(45.6)</td>
<td>(25.1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Actigraphy</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bright light</td>
<td>357.2</td>
<td>389.6</td>
<td>397.0†</td>
<td>1.16</td>
</tr>
<tr>
<td>(113.5)</td>
<td>(66.8)</td>
<td>(101.8)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dim light</td>
<td>357.6</td>
<td>367.6</td>
<td>327.3</td>
<td></td>
</tr>
<tr>
<td>(47.4)</td>
<td>(39.2)</td>
<td>(44.1)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Data are presented as mean (SD), with analysis of variance main and interaction effects.

*Significant difference from baseline (<. 05).
† Significant difference between groups (<. 05).
FU1 refers to follow-up week 1; FU4, follow-up week 4.

Table 5—Number of Days Per Week of Feeling Drowsy, Irritable, or Depressed and Number Of Minutes More Sleep Needed Before Light Exposure and After Follow-Up Week 1 and Follow-Up Week 4 for the Bright-Light and Dim-Light Groups

| Variable      | Condition | Before | FU1  | FU4  | M | F  | df | P  | F  | df | P  |
|---------------|-----------|--------|------|------|   |    |    |    |    |    |    |
| Drowsy        | Bright    | 3.8 (2.1)| 2.0 (2.0)* | 1.7 (2.0)* | 11.6 | 2,21 | < .0001 | 1.4 | 2,21 | .3 |
| Dim           | 3.9 (1.7) | 3.2 (2.0) | 2.6 (2.0) |          |                    |                    |      |       |      |     |
| Irritable     | Bright    | 1.5 (1.8) | 0.7 (0.7) | 0.5 (0.7) | 4.7 | 2,19 | .01 | 0.2 | 2,19 | .8 |
| Dim           | 2.0 (1.6) | 1.5 (1.1) | 1.2 (1.4) |          |                    |                    |      |       |      |     |
| Depression    | Bright    | 1.5 (2.0) | 0.5 (0.7) | 0.2 (0.5) | 1.8 | 2,19 | 0.2  | 3.1 | 2,19 | .05 |
| Dim           | 0.6 (0.9) | 0.6 (0.9) | 0.7 (1.0) |          |                    |                    |      |       |      |     |
| Need More Sleep, min | Bright   | 122.8 (70.3)| 49.1 (47.0)* | 49.1 (47.0)* | 10.3 | 2,20 | .0003 | 2.1 | 2,20 | .13 |
| Dim           | 117.3 (52.7)| 98.2 (67.2)| 76.4 (66.2) |          |                    |                    |      |       |      |     |

Data are presented as mean (SD), with analysis of variance main and interaction effects.

*Significant difference from baseline (<. 05).
FU1 refers to follow-up week 1; FU4, follow-up week 4.
exposed to 2 evenings of bright light,9 there was no control group and sleep variables were monitored for only 5 nights following light therapy. In this present study, we exposed a control group to the same protocol but used dim red light on the 2 evenings instead of bright white light. No significant circadian or sleep changes followed evening exposure to a dim red light. This lack of circadian-phase change for the exposure to dim red light is consistent with findings from recent studies showing no melatonin suppression or phase change with the use of longer-wavelength light.16-18 In both the earlier and present studies, evening exposure to bright light induced significant and comparable circadian-phase delays. In the present study, we continued monitoring sleep and daytime symptoms for 4 weeks after light therapy, with all variables continuing to show improvement.

When comparing changes in sleep variables between these 2 studies at 1 week following bright-light therapy, there were several similarities. Neither showed significant changes in sleep-onset times. In both studies, subjects tended to go to bed and fall asleep at about 11:00 PM, which was only 3 hours before their pretreatment Tmin time. After light therapy, this same bedtime was about 5 hours prior to their delayed Tmin. Furthermore, both showed delays in wake-up times and increases in total sleep times.

In previous studies that have treated sleep-maintenance insomnia with evening bright light,10-14 the light pulse was presented no later than the subjects’ habitual bedtimes, or about 4 hours before their average Tmin time. In contrast, we administered the 4-hour light pulse up to midnight on the first night and up to 1:00 AM on the second night, within 1 to 2 hours of their Tmin time. Because the typical phase-response curve shows generally greater phase delays for light presented closer to the time of Tmin (for a recent review article see citation 20), each light pulse in our study is likely to have produced greater phase delays with each night of bright-light therapy than the earlier studies with sleep-maintenance insomniacs. Therefore, the total circadian-phase delays in the present study were similar to those of previous studies but were obtained much more rapidly. A more-rapid phase delay may enhance the efficacy of bright-light therapy for this type of insomnia.

It also may be the case that phase-advanced circadian rhythms were a greater contributor to the reduced sleep of our early-morning awakening insomniacs than in the sleep-maintenance insomniacs of previous studies. Generally, in our previous and present studies, the pretreatment circadian-rhythm phase was timed earlier relative to sleep-onset time as compared to that of the sleep-maintenance insomniacs phase timing of other studies. The circadian morning “wake-up zone”22 before treatment would be relatively earlier and, thus, more intense at a given interval (eg, 6 hours) after sleep onset in the present study. Therefore, the comparable circadian-phase delays of the present study would have provided greater reduction of the circadian early-morning interference to sleep and be of greater clinical benefit. If this is the case, it would be suggested that evening bright-light therapy will be more effective for early-morning awakening insomniacs with circadian rhythms timed abnormally early with respect to their intended bed period.

Although the results do suggest the involvement of phase delay induced by bright light in the improvement of sleep, it seems likely that there were other factors contributing to the relative improvements in the bright-light group. After the initial phase delay, there was a steady and significant phase advance over the follow-up period back toward the original circadian-phase timing. Despite this, the bright-light group continued to show greater differential improvement of sleep than the dim-light group. We speculate that these chronic insomniacs were likely to have had some psychophysiological or conditioned insomnia in the chronic early-morning awakenings. This conditioned insomnia may have been, to some degree, extinguished in the bright-light–treated group as a result of experiences of some improved sleep and reduced wake time in the first week or 2 after treatment. This could have led to further improvements despite the later regression of circadian phase. In any case, the gradual regression of circadian phase toward the original phase timing would be expected to rekindle early-morning awakening and eventually lead to a return of the hypothesized conditioned early-morning awakening insomnia. Therefore, as in the Suhner et al10 study, the use of occasional treatment nights of bright-light therapy, but to a later evening time as in the present study, would be recommended. Further research is needed to determine if the treatment benefits shown in the present study extend beyond 4 weeks and, if not, whether occasional therapy can maintain the gains.

CONCLUSION

In this study, we have demonstrated that a brief course of evening bright-light therapy is effective in delaying the circadian phase and improving sleep parameters and some daytime symptoms of individuals who experience early-morning awakening insomnia. Furthermore, these improvements continue for up to 1 month following light therapy.

REFERENCES