In a randomized, parallel design, 19 patients with winter depression were treated with either a week of a white 1.5-hr dawn simulation peaking at 250 lux or a week of a red, 1.5-hr dawn signal peaking at 2 lux. The subjects were told that they would receive either a white or red dawn reaching in intensity that would be dimmer than standard bright light treatment. At the end of both the baseline week and the treatment week subjects were blindly assessed with the Hamilton Rating Scale for Depression (HDRS). Analysis of covariance was used to compare the two dawn treatments. The white, 1.5-hr, 250 lux dawn simulation resulted in significantly (p < 0.05) lower HDRS scores compared to the red, 1.5-hr, 2 lux dawn. This is the second controlled study which indicates that dawn simulation is an effective treatment for winter depression.

Key Words: Depression, seasonal affective disorder, light therapy, dawn simulation

Introduction

Bright light (greater than 2500 lux) has been shown to be effective relative to dim (less than 500 lux) light control conditions in the treatment of winter depression (Rosenthal et al 1984, 1985; Wirz-Justice et al 1986; Terman et al 1989a). Dawn simulation, light that approximates a dawn signal, is a more convenient technique and may be administered while the subject is asleep (Terman et al 1989b, 1989c, 1990). In an uncontrolled study of dawn simulation, Terman et al (1990) noted clinical improvement in six of eight subjects.

In a previous parallel-design treatment study of winter depression, we had found that a gradual 2-hr dawn peaking at 250 lux was superior to a 30-min dawn peaking at 0.2 lux, a placebo condition (Avery et al 1993). In that study, subjects were told that the dawn signal was “gradual” and the placebo condition, “rapid.” To increase the plausibility of the placebo condition, the present study uses a placebo condition, which has a maximal intensity ten times brighter than the previous placebo and has a duration that is equal to that of the dawn signal. Using a parallel design, the present study compares a white, 1.5-hr dawn peaking at 250 lux with a red, 1.5-hr signal peaking at 2 lux.

Methods

Subjects were recruited through advertisements and through publicity concerning our program. Subjects fulfilled criteria for major depressive episode according to DSM-III-R (American Psychiatric Association 1987) as well as primary affective disorder according to Feighner criteria (Feighner et al 1972). Subjects also fulfilled Rosenthal criteria for seasonal affective disorder (Rosenthal et al 1984). Subjects reported regularly occurring fall–winter
depressions (with at least two occurring during consecutive winters) remitting during the spring or summer. In addition, no psychosocial variables could account for the regular changes in mood.

All had hypersomnia as part of their winter depression. Hypersomnia was defined as sleeping at least an hour more during their winter depression compared to their euthymic summer sleep duration. Hypersomnia appears to be associated with a good response to morning bright light compared to a response to evening bright light (Avery et al 1991). Because dawn simulation is a morning light treatment, only hypersomnic subjects were chosen. Although this sample selection limits the relevance of the results to hypsomnic winter depressives, over 80% of winter depressives have hypersomnia (Rosenthal and Wehr 1987).

All subjects were free of any psychotropic medication for at least 4 weeks prior to the study. Subjects gave their written informed consent.

During the 2-week outpatient study, subjects were asked to sleep only between the hours of 9 PM and 6 AM and keep a log of their sleep. If sleep had not been standardized, the results would have been more difficult to interpret. If sleep were not controlled during the baseline week and subjects were allowed to sleep later, the effect of dawn simulation would be difficult to distinguish from the effect of partial sleep deprivation. If the subjects had maintained their usual time of awakening during the study and the dawn signal had been scheduled to start 1.5 hr prior to that time, the dawn signal administered to some subjects would have been only a winter dawn signal. The standardization of sleep resulted in the exclusion of hypsomnic subjects who at screening said that they would be unable to awaken at 6 AM.

The first week was a baseline week during which no light treatment was administered. At the beginning of the second week the subjects were randomly assigned one of two dawn simulation conditions, either a white dawn simulation gradually (approximately 3 log\textsubscript{10} lux/hr increasing over 1.5 hr (4:30 AM to 6:00 AM,) which peaked at 250 lux (similar to room light level) or a red dawn signal gradually (approximately 1.5 log\textsubscript{10} lux/hr) increasing over a 1.5-hr period (4:30 AM to 6:00 AM,) which peaked at an illuminance of 2 lux (greater than moonlight). The white dawn signal timing and duration approximated an equinox dawn signal at 45° latitude. During both weeks the subjects slept at home. The randomization was stratified according to gender and quarter of the menstrual cycle. Subjects were told that they would be randomly assigned to receive either a white, 1.5-hour dawn or a red, 1.5-hour dawn and that the final illuminance of both would be much dimmer than standard bright light treatment.

The light sources for both dawn simulations were housed in identical fixtures (Remcraft model #1051) with a Juno filter holder (#T570 4 3/4 inch size) attached. With the white dawn, a Roscolux gel filter Light Tough Frost (#102) (Rosco, Hollywood, CA) was placed in the filter holder. The white dawn light treatment utilized a 100-watt incandescent reflector floodlight (Philips, Somerset, NJ), which was 4 feet from the pillow. With the red dawn, a Roscolux Medium Red (#27) gel filter, 2 Tough Silk (#104) filters, and a Neutral Density (#3404) filter (Rosco, Hollywood, CA) were placed in the filter holder. The red dawn utilized a 30 watt incandescent reflector floodlight (Philips) 4 feet from the pillow. Even white incandescent lights have a spectral distribution that is predominately in the red spectrum. In neither condition was the lamp visible with the filters in place. The intensity of the light at 4 feet was measured using a Vactec Photometer, Model #3107, (Maryland Heights, MO). A string was attached to each fixture; the end of the string identified a point 4 feet from the fixture so that the subjects could position the fixture on the wall behind the bed in his or her own bedroom.

Subjects were asked to sleep until 6 AM at which time they were awakened by an alarm. If they awoke prior to 6 AM, they were asked to avoid looking at the lights, close their eyes and try to go back to sleep in an attempt to standardize sleep and light exposure (through closed eyelids). If subjects had been allowed to wake up and get out of bed before 6 AM, they would have received a different light stimulus than those who did not wake up and would have had different sleep duration. The incandescent light was plugged into a sunUp dawn simulator device (Pi Square, Inc., Redmond, WA), which created a gradually increasing voltage to the incandescent light starting at a time which could be specified. Subjects used these lights daily during the treatment week.

The subjects’ bedrooms were required to be dark. If a street light or security light did shine through the bedroom windows, subjects were given a sheet of black plastic to cover the windows. Subjects were asked to turn off any nightlights or hall lights that might shed light into the bedroom. These hypsomnic patients typically slept throughout the night and did not need to get up to go to the bathroom. If this occurred, they would attempt to do so only with a nightlight in their bathroom. During the study they were advised to avoid morning sunlight before 8 AM.

Expectations of the response to white 1.5-hr dawn simulation and red 1.5-hr dawn simulation were assessed at baseline prior to exposure to either dawn signal. They rated their expected response to white dawn simulation and to the red dawn simulation on a global scale (1 = worse, 2 = no change, 3 = slight improvement, 4 = much improvement, 5 = very much improvement). At the end of each light treatment, subjects rated their own responses to light treatment on the same global scale.

Subjects were rated blindly by experienced psychiatrists using Seasonal Affective Disorder Version of the Struc-
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The baseline expectations for the white dawn and red dawn were similar for our total sample (white dawn, 3.2 ± 0.7; red dawn, 3.2 ± 0.7, paired t = 0.69, df = 18, p = NS). Baseline expectations (± SD) for the treatment actually received were similar (white dawn, 3.4 ± .7; red dawn, 3.2 ± 0.8, t = 0.66, df = 17, p = NS).

Depression Ratings

The global improvement rating (± SD) after treatment was significantly better with the white, 250 lux dawn compared to the red, 2 lux dawn using ANCOVA with baseline expectations for the treatment received as the covariate (4.2 ± .7 versus 2.9 ± .9, F = 11.8, df = 1, 16, p < 0.005.) Among those who received the white dawn, there was no significant correlation between the baseline expectations of the white dawn and the global improvement (r = -0.47, df = 8, p = NS). Among those who received the red dawn, there was no significant correlation between baseline expectations for the red dawn and the actual global response (r = 0.21, df = 7, p = NS).

Table 1 summarizes the individual HDRS and SAD Subscale scores and the mean scores for both groups at baseline and after treatment. The white-dawn group and the red-dawn group did not have significantly different baseline scores for the HDRS (t = 0.12, df = 17, p = NS) or the SAD Subscale (t = 0.97, df = 17, p = NS). The HDRS scores following treatment with the white, 250 lux dawn were significantly lower than with the red, 2 lux dawn using ANCOVA with the baseline scores as the covariate; the SAD Subscale scores showed a nonsignificant trend (p = 0.10) for the white dawn to be superior (See Table 1). Both the HDRS and the SAD Subscale ratings significantly decreased relative to baseline in the white-dawn group; whereas the red-dawn group exhibited a nonsignificant trend for the HDRS to decrease and a significant decrease in the SAD Subscale.

Even with inclusion of the subject who was excluded because of severe flu symptoms, the main differences between the white dawn and red dawn hold. The HDRS is significantly lower after the white dawn than after the dim red signal (ANCOVA with the baseline HDRS score as the covariate, n = 20, F = 6.5, p < 0.05). The global improvement was significantly better with the white dawn compared to the dim red signal (ANCOVA with baseline expectations for the treatment received as the covariate, n = 20, F = 6.5, p < 0.05).

A 38-year-old woman randomized to the red dawn reduced her HDRS from 22 to 1. After the study she relapsed into depression, and bought a dawn simulator. She used the white, 250 lux dawn recommended by the investigators, but found that she had severe early morning awakening, awakening at 5 AM and being unable to return to sleep. By lowering the final illuminance to about 2 lux, she was able to...
respond as she had during the study and did well for the remainder of the winter.

**Side Effects**

All subjects receiving the white, 250 lux dawn experienced at least mild early morning awakening, usually a brief awakening during the first few days of treatment between 5 AM and 6 AM. Two women experienced moderate early morning awakening with the white dawn, awakening at 5:15 AM and 5:30 AM and being unable to return to sleep. Only four of the nine receiving the red dawn had slight early morning awakening. The proportion who experienced at least mild early morning awakening (10/10) was significantly greater than proportion of the red-dawn group (4/9) (two-tailed Fisher’s Exact test, p < 0.05). None receiving the white dawn and two in the red-dawn group experienced headache during the treatment week. None of the subjects became manic or hypomanic.

**Sleep Estimations**

The mean (± SD) retrospective estimation of the usual sleep duration in the winter was similar in the two groups (white dawn, 9.2 ± 1.4 hr; red-dawn group, 9.0 ± 0.6; t = 0.52, df = 17, p = NS). The retrospective evaluation of their usual sleep duration in the summer was similar in the two groups (white dawn, 6.9 ± 0.8; red dawn, 7.2 ± 0.4; t = 0.80, df = 17, p = NS).

Retrospective estimates of the time of sleep onset during the winter (white dawn 9:54 PM ± 60 rain.; red dawn, 9:48 PM ± 48; t = 0.24, df = 17, p = NS), the time of awakening during the weekdays (white dawn, 7:12 AM ± 60; red dawn; 6:54 AM ± 54; t = 54; t = 0.88, df = 17, p = NS) and the times of awakening during weekends (white-dawn, 7:36 AM ± 66; red dawn, 8:06 AM ± 66; t = 1.02, df = 17, p = NS) were similar for those receiving the white and red-dawns. Those who had to make a minor adjustment or no adjustment (half hour or less) to the imposed sleep schedule had a response similar to those who made a greater adjustment as measured by the HDRS after the treatment week (minor or no adjustment, 8.9 ± 4.8, n = 8; greater adjustment, 13.5 ± 8.7, n = 11; t = 1.37, df = 17, p = NS.) The SAD Subscale after treatment was also similar in the two groups (minor or no adjustment, 8.7 ± 4.4; greater adjustment, 9.3 ± 6.8; t = 0.19, df = 17, p = NS.)

The sleep logs revealed that compliance with the imposed sleep schedule was similar in the two groups. The mean
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Table 2. Times of Sleep Onset and Awakening and Sleep Duration

<table>
<thead>
<tr>
<th></th>
<th>White 1.5-hr, 250 lux dawn</th>
<th>Red 1.5-hr, 2 lux dawn</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Baseline</td>
<td>Treatment</td>
</tr>
<tr>
<td>Sleep onset</td>
<td>11:11 PM ± 58</td>
<td>10:32 PM ± 31</td>
</tr>
<tr>
<td>Wake-up</td>
<td>6:22 AM ± 34</td>
<td>6:17 AM ± 28</td>
</tr>
<tr>
<td>Sleep duration</td>
<td>6.9 ± .8</td>
<td>7.2 ± .8</td>
</tr>
</tbody>
</table>

(± SD in minutes) sleep onset times and wake-up times and sleep durations for the two groups are noted in Table 2. The sleep durations do not equal the difference between time of awakening and sleep onset because some subjects awakened in the middle of the night. A two-way repeated-measures ANOVA revealed no differences in sleep onset time during the baseline week and treatment week (F = 1.50, df = 1, 17, p = NS), no differences between groups (F = 0.51, df = 1, 17, p = NS) and a nonsignificant trend for interaction between group and order (F = 4.2, df = 1, 17, p = 0.06). A two-way, repeated measures ANOVA revealed no differences in the sleep offset time during the baseline and treatment weeks (F = 0.75, df = 1, 17, p = NS), no differences between groups (F = 0.79, df = 1, 17, p = NS) and no significant interaction between order and group (F = 0.39, df = 1, 17, p = NS). A two-way, repeated-measures ANOVA revealed no differences in sleep duration during the baseline and treatment weeks (F = 0.35, df = 1, 17, p = NS), no differences between the two-treatment groups (F = 0.00, df = 1, 17, p = NS), and a significant interaction between order and group (F = 12.0, df = 1, 17, p < 0.01).

The HDRS when the subjects were screened for the study did not differ significantly from the HDRS score after the imposed sleep schedule of the baseline week (HDRS at screening; 19.1 ± 3.7; HDRS at baseline, 20.6 ± 4.7; t = 1.27, df = 18, p = NS.) The SAD Subscale when the subjects were screened for the study did not differ significantly from the SAD Subscale score after the imposed sleep schedule of the baseline week (SAD Subscale at screening, 15.2 ± 3.9; t = 0.78, df = 18, p = NS.)

Discussion

Response to Dawn Simulation

The response to the white 1.5-hr, 250 lux dawn as measured by the HDRS was superior to the red 1.5-hr, 2 lux “placebo” signal in treating patients with winter depression. Early morning awakening was common with the 1.5-hr, 250 lux dawn but usually was not perceived as a disturbing symptom in these hypersomnic SAD patients who had difficulty awakening in the morning. Because early morning awakening adds points to the HDRS, the superiority of the 1.5-hr, 250 lux dawn occurred in spite of the frequent early morning awakening associated with its use.

The SAD Subscale showed a non-significant trend for superiority of the 1.5-hr, 250 lux dawn. The SAD Subscale may be relatively insensitive to change because one item, hypersonmia, could not be adequately assessed because subjects were not allowed to sleep past 6:00 AM.

This is the second controlled study showing dawn simulation effective in treating winter depression. Avery et al (1993) found that a 2-hr dawn simulation peaking at 250 lux reduced the HDRS from 17.1 to 5.5 and was significantly better than a 30-min, 0.2 lux dawn that reduced the HDRS from 18.6 to 11.1.

The improvement in the HDRS from 20.7 to 8.0 is similar to the improvement seen in earlier studies with dawn simulation (Terman et al 1990; Avery et al, 1992a, 1992b, 1993) and similar to that seen with morning bright light therapy. Terman et al (1989a) in his pooled (n = 172) analysis that morning bright light therapy reduces the mean HDRS score from a 17.8 to 8.1.

Is Dawn Simulation a Placebo Response?

Because of significant placebo responses often observed among depressed subjects (Eastman 1990), the possibility that the therapeutic effect of the white, 1.5-hr, 250 lux dawn can be accounted for by a placebo effect should be considered. A true placebo condition for dawn simulation, as for other light therapies (Eastman 1990), may be difficult to achieve as subjects are able to see the treatment condition on awakening. The baseline expectations for the white and red dawns were similar. Because we did not want to bias the subjects against the dim dawn by showing both dawns, we may not have been measuring true expectations. Because of the parallel design, both groups were blind to the final intensity of the dawn simulation received by the other group.

The red, dimmer signal appeared to be a convincing control condition; the HDRS score dropped from 20.4 to 15.6. Winter depressives have not shown particularly striking placebo responses in previous studies using dim light boxes (Terman et al 1989a) or placebo medication (O’Rourke et al 1989; McGrath et al 1990; Rosenthal et al 1988). Studies using a dim (60 lux) light visor (Joffe et al 1993) and an deactivated negative ion generator (Eastman et al 1992) have been associated with more robust improvement.

In one of our previous studies (Avery et al 1992b), we found that a 10-min dawn peaking at 275 lux lowered the
HDRS from 17.2 to 7.0 and was not significantly different from a 2.5-hr dawn peaking at 275 lux, which lowered the HDRS from 17.7 to 5.9. Although the results could have been the result of a placebo effect, the short dawn may have been bright enough to have some active effect and the longer duration dawn may have "overdosed" the subjects (Avery et al 1992b). In that study, heterogeneity of responses of duration dawn may have "overdosed" the subjects (Avery et al 1993), one of 12 subjects receiving a 2.5-hr, 275-lux dawn (Avery et al 1992b) and none out of seven subjects receiving a 2-hr, 1700-lux dawn (Avery et al 1992a). In the present study, none became hypomanic. The dawn simulation in this study was well-tolerated except for some brief awakenings from 5 AM to 6 AM in all the subjects receiving the white dawn; two subjects had moderate early morning awakening, awakening at 5:15 AM or 5:30 AM and being unable to return to sleep. Avery et al (1992a) found that a 2-hr dawn peaking at 1700 lux resulted in at least mild early morning awakening in all seven subjects and severe early morning awakening in three. Thus an "overdose" of dawn is possible. Whether this effect is secondary to the nonnaturalistic dawn signal, an extreme phase-advance of the circadian clock, or other factors is not known.

Possible Mechanism of Action

Bright light therapy is thought to work through the retinoreceptors (Wehr et al 1987). Even though the eyes are closed during dawn simulation treatment, the eyelids are translucent to light (Moseley 1988). The sensitivity of the retinoreceptors at the early morning hours may be increased (O'Keefe and Baker 1987; Bassi and Powers 1986). Increased retinal sensitivity might explain why a 250-lux signal partially blocked by the eyelids may be effective even though light boxes of similar illuminances administered later in the morning have been found relatively ineffective (Rosenthal et al 1989).

The therapeutic effect of bright light therapy has been hypothesized to be mediated by its circadian phase-shifting effect (Lewy et al 1984, 1987; Sack et al 1990; Avery et al 1990; Dahl et al 1993); dawn simulation may work in a similar way (Terman and Schlager 1990). Even low levels of light have been shown to shift circadian rhythms (Boulos et al 1992; Czeisler et al 1981, 1989). Terman et al (1990) found that in two patients, the nocturnal melatonin rhythm was phase-advanced (shifted earlier) by dawn simulation up to 250 lux relative to dim (< 1 lux) baseline conditions.

Does Dawn Simulation Work Through Sleep Deprivation?

The sleep interruption seen with dawn simulation raises the possibility that dawn simulation might work through partial sleep deprivation, an effective treatment for depression (Kuhs and Tolle 1991). The sleep logs showed a nonsignificant trend for the sleep duration to increase in those receiving the white, 250 lux dawn; however, the sleep duration did not change with the red, 2 lux dawn. With the sleep deprivation during the baseline week, the depression ratings did not change significantly. Similar adjustments to the imposed sleep schedule were made by the groups receiving the white and red dawns. The times of awakenings prior to the base-

Dawn Simulation Signal

Terman et al (1989b, 1989c, 1990) developed dawn simulation, an innovative technique that differs from standard bright light therapy in that dawn simulation occurs earlier, while the subject is asleep, at a dim light illuminance that gradually increases and peaks at illuminances that are much less than the standard bright light therapy. They used a computer and a sophisticated algorithm in conjunction with a photosensor feedback loop and light attenuation mechanism. The present study utilized a dawn simulator that created a gradual illumination ramp that is not as precise an approximation of a natural dawn as the Terman dawn simulator. Specifically, the light signal used in this study increases more rapidly than a natural dawn in the initial portion of the signal. This rapid increase in illuminance may be a reason for the early awakenings seen in this study. However, the dawn signal used may be close enough to a precise dawn signal to be therapeutic. In nature, variables, such as changing sleeping position, may influence the signal actually reaching the eyes. In addition there is no one dawn signal in nature: the duration of the dawn signal increases with latitude and increases during the summer at higher latitudes (Terman 1989c).

Determining the optimal dawn signal may be complicated by the heterogeneity of responses to light as suggested by one of our previous studies (Avery et al 1992b). One subject responded well to the 2-lux dawn during the study, relapsed off treatment, experienced severe early morning on a 250-lux dawn, and responded well again to the 2-lux dawn.

Side Effects

Dawn simulation is not without side effects. In three previous studies, two subjects have become hypomanic: 1 of 13 receiving a 2-hr, 250 lux dawn (Avery et al 1993), one of 12 subjects receiving a 2.5-hr, 275-lux dawn (Avery et al 1992b) and none out of seven subjects receiving a 2-hr, 1700-lux dawn (Avery et al 1992a). In the present study, none became hypomanic. The dawn simulation in this study was well-tolerated except for some brief awakenings from 5 AM to 6 AM in all the subjects receiving the white dawn; two subjects had moderate early morning awakening, awakening at 5:15 AM or 5:30 AM and being unable to return to sleep. Avery et al (1992a) found that a 2-hr dawn peaking at 1700 lux resulted in at least mild early morning awakening in all seven subjects and severe early morning awakening in three. Thus an "overdose" of dawn is possible. Whether this effect is secondary to the nonnaturalistic dawn signal, an extreme phase-advance of the circadian clock, or other factors is not known.

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