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The Effect of Evening Bright Light in Delaying the Circadian Rhythms and Lengthening the Sleep of Early Morning Awakening Insomniacs

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Summary: Past studies have predicted that early morning awakening insomnia is associated with advanced or early circadian rhythms. Because bright light stimulation in the evening can delay the phase of circadian rhythms, we tested its effects on nine (4 females, 5 males) early morning awakening insomniacs. Their sleep was evaluated with wrist actigraphy and their temperature and melatonin circadian rhythms were measured in constant routine procedures. In the initial evaluation, the temperature rhythm phase positions of these insomniacs did appear to be earlier than normal. The subjects were then exposed to bright light stimulation (2,500 lux) from 2000 to 2400 hours on two consecutive evenings. Following the evening bright light treatment, temperature rhythm phase markers were delayed 2–4 hours and melatonin phase markers were delayed 1–2 hours. Sleep onset times were not changed but the mean final wake-up time was delayed from 0459 hours to 0611 hours, resulting in a mean increase of total sleep time of > 1 hour. This pilot study suggests that evening bright light stimulation may be an effective nondrug treatment for early morning awakening insomnia. **Key Words:** Insomnia—Circadian rhythms—Bright light therapy—Body temperature—Early morning insomnia.

Sleep studies have shown the incidence of early morning awakening insomnia (EMA) ranges from 3% to 22% (1,2). Those who experience early morning awakenings also report midafternoon fatigue and have early sleep onsets, sometimes as early as 2100 hours. Usually there is no difficulty in initiating sleep. In fact, the tendency to fall asleep early in the night can be a social handicap. If the person does attempt to delay bedtime by remaining active in the evening, total sleep time is decreased since awakening still occurs at about the same time. They wake up much earlier than desired (e.g. 0300 hours) and because they are unable to get back to sleep, their total sleep time may range from only 5 hours to 7 hours (3). Excessive sleepiness may occur the following day due to the inability to "sleep in" in the morning (4).

It has been proposed that insomnia associated with sleep-scheduling disorders may be due to problems in the timing of a circadian pacemaker. Indeed, the clinical features described above are basically the same as those described for advanced sleep phase syndrome presumed to be due to an early or advanced circadian pacemaker (5). Normally people fall asleep 4–5 hours before their body temperature minima and wake up when their temperature is rising (6). Strogatz (7) and Zulley et al. (8) identified a wake-up zone 4–7 hours after the body temperature minimum, when sleeping subjects would wake, regardless of how long they had been asleep. If the body temperature rhythm is phase advanced in relation to the preferred sleep period, the wake-up zone would occur earlier (0300–0500 hours) and, predictably, terminate the sleep period before sufficient sleep is obtained (9).

A number of studies have shown bright light visual stimulation to be an effective manipulator of circadian rhythm phase. Exposure to bright light in the morning advanced the circadian rhythms of control subjects and subjects suffering delayed sleep phase syndrome (10–13). Conversely, evening bright light exposure has been shown to phase delay circadian body temperature rhythms in normal subjects (14–19).

Lewy et al. first suggested that delayed as well as advanced sleep phase syndrome can be helped by ap-

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propriately timed bright light stimulation (20). Singer and Lewy reported a single case of advanced sleep phase syndrome (21). Following 2 weeks of nightly exposure to evening bright light, the patient showed a delay of sleep cycle and dim light melatonin onset. The purpose of the present study is to ascertain whether evening bright light therapy will phase delay the temperature and melatonin circadian rhythms of early morning awakening insomniacs and whether their final wake up time will be delayed and sleep length will be increased.

METHOD

Subjects

A total of 120 potential subjects responded by telephone to requests made on radio, television and in local newspapers for people who had early morning awakening insomnia to undergo assessment and bright light therapy. Following an initial telephone interview, 85 respondents were sent a sleep questionnaire and a 1-week sleep–wake diary to complete and return by post. A preliminary selection from the postal respondents was made on the basis of the questionnaire and diary, taking into consideration the sleep parameters of early evening sleepiness, early final wake up time (0300–0500 hours) and a total sleep time of <7 hours. Fifteen people who thus reported early morning awakening insomnia for a period of at least 1 year were selected at this stage to participate in the laboratory procedures. The subjects were five females and 10 males ranging in age from 34 to 70 years for males and 32 to 77 for females. Subjects were in self-reported good health and were not habitual users of alcohol, caffeine, nicotine or hypnotic medications.

At the commencement of the initial constant routine, subjects completed the Beck Depression Inventory (BDI). Of the 15 subjects for whom there were complete data in the initial constant routine, nine were asymptomatic (BDI $<$ 10), five were mild to moderately depressed (10–18) and one subject was moderately to severely depressed (BDI = 28). The median BDI score was 7.

Of the 15 subjects who completed the initial phase assessment, six did not participate further for a variety of reasons, including lost temperature data and unwillingness to repeat the initial constant routine (2), inability to complete the final phase assessment procedure (2), and personal time commitment difficulties (2). Thus, nine subjects (4 females, 5 males) with a mean age of 53.4 years completed the full assessment and treatment program. These nine subjects did not differ significantly from the six noncompleting subjects in any of the initial assessment measures.

Apparatus and materials

Subjects completed a sleep questionnaire and sleep–wake diaries in order to ascertain sleep parameters. Wrist activity was recorded using a movement sensor and a Vitalog PMS-8 monitor. Actigraphs worn on the wrist of the nondominant hand recorded the total number of wrist movements in consecutive 3-minute periods during time in bed at home. The records were analyzed to estimate sleep onset, nocturnal awakenings and final wake-up time. Rectal temperature was measured at 3-minute intervals using a Yellow Springs Instrument Series 400 temperature probe, indwelling 10–15 cm into the rectum and connected to a Vitalog PMS-8 monitor.

Bright light was produced by two light boxes, each containing six incandescent tungsten filament 100-watt white globes behind a translucent diffusion screen. These boxes were placed on tables at eye level and approximately 60 cm from the subjects' faces. At this distance the light level, measured with a Gossen Lunasix light intensity meter, was 2,500 lux. A dim light condition was produced by a light box containing two incandescent tungsten filament 60-watt red globes behind a translucent diffusion screen. The box was placed as above. The light level intensity was measured at 150 lux at the subjects' faces.

Procedure

Subjects kept a sleep-wake diary and wore the wrist actigraph at home over a period of a week in order to obtain subjective and objective information on the sleep parameters of lights-out time (LOT), sleep onset latencies (SOL), nocturnal awakenings, final wake-up time (WUT), and total sleep time (TST).

The laboratory session included one night of sleep in the laboratory followed by a 26-hour constant routine starting at 0800 hours and finishing at 1000 hours the following morning. In the constant routine, as described by Czeisler et al. (22) and Morris et al. (23) subjects remained awake, in a near-supine position, in an atmosphere of controlled lighting (100 lux) and ambient temperature of 20°C. The subjects were given 2-hourly snacks of equal caloric value and 150 ml of water. Rectal temperatures and activity were recorded at 3-minute intervals. Melatonin activity was assessed with 2-hourly urine collections. Each collection volume was measured and a 5-ml aliquot taken for urinalysis. The concentration of the major melatonin metabolite, 6-sulphatoxymelatonin, was analyzed according to the radioimmunological method of Arendt et al. (24). Hourly metabolite output was cal-

culated by multiplying concentration by hourly urine volume.

At the completion of the laboratory session, subjects returned home and had a longer-than-normal sleep to recover from the sleep deprivation. Approximately a week later they returned for bright light treatment.

For the bright light treatment, subjects arrived at the Laboratory at 1930 hours. From 2000 to 2400 hours they sat in front of the light boxes at a distance of 60 cm in order to receive the required light intensity. Light intensity readings were taken prior to and during this time period. Subjects watched television or selected videos on a monitor placed at a level just above the light boxes. This ensured that the full intensity of light was perceived by subjects throughout the 4-hour period, as recommended by Dawson and Campbell (25). At 2400 hours, subjects were free to return home or to remain in the laboratory to sleep.

The following evening, subjects returned to the sleep laboratory at 1900 hours. After changing into their night attire, they again received the bright light from 2000 to 2400 hours, as in the previous evening. At 2400 hours, subjects attended toileting requirements and then retired to bed. After awakening the next morning, subjects commenced the 26-hour constant routine, which repeated the procedure of the first constant routine.

Following the completion of the routine, subjects returned home and had a recovery sleep starting about 2 hours before their normal bedtime and ending an average of 2 hours after their normal wake-up time according to their sleep/wake diaries and wrist actigraph monitors. For a further 5 days following the recovery sleep, subjects kept a sleep–wake diary and wore the wrist actigraph at night. All subjects participated in the study during the winter months of the year, when daylight extended for <12 hours from 0630 to 1800 hours. Reported daytime napping was infrequent in these subjects and, in any case, was prohibited throughout the study.

The first two subjects in the study also participated in a control condition. A week following the initial assessment routine, the two subjects were exposed to dim red light (150 lux) on two consecutive evenings for 4 hours, from 2000 to 2400 hours, followed by a constant routine. Red light was used as a credible treatment placebo, but the intensity was below the threshold to suppress melatonin and affect temperature rhythms (26,27). The assessment of temperature and melatonin circadian rhythms and sleep parameters following the dim light condition was identical to that during the initial assessment and post bright light constant routines. Two weeks following the dim red light, these two subjects continued with the bright light treatment and follow-up identical to the other subjects.

Analysis

The statistical technique used for examining the circadian rhythm variations was based on the principle of Fourier cosine analysis, in which a 24-hour fundamental cosine component and a 12-hour harmonic component were included. The Fourier model was fitted to 24 hours of rectal temperature data from 0900 to 0900 hours according to the least squares harmonic regression technique outlined by Bliss (28). The circadian parameter estimates of mesor, acrophase of the 24-hour cosine fundamental, the temperature maximum of the 12-hour harmonic and temperature minimum (T_{min}) and maximum (T_{max}) of the complex (24-hour fundamental plus 12-hour harmonic) fitted curve were obtained from the Fourier Series analysis. The differences in the rhythm parameters between pre- and post-treatment conditions were analyzed with paired t tests.

Sleep parameters

The average correlation between subjects' diary estimate of their sleep onset time and cessation of movement indicated by the wrist actigraph was 0.91 and the average difference was only 5 minutes. The average correlation between subjects' estimate of their final wake-up time and resumption of movement indicated on the wrist actigraph was 0.98, with an average difference of only 3 minutes. For the purpose of sleep data analysis, the wrist actigraph estimates of sleep onset time, nocturnal awake time and final wake-up time were used. Total sleep time was estimated by adding the hours between sleep onset and final wake-up time and subtracting the nocturnal awake time. Each of these sleep parameters was averaged separately for each subject over the 5 days before the initial circadian assessment and 5 days after bright light treatment and second circadian rhythm assessment.

RESULTS

Circadian rhythm parameters

Figure 1 shows the group mean rectal temperature rhythm in the constant routines from 0900 to 0900 hours before and after the evening bright light treatment. Phase delays in both maximum and minimum values are apparent in the mean curves.

Rhythm parameters of each subject's temperature data were obtained using the Fourier curve-fitting technique. Table 1 shows the mean temperature rhythm parameters derived from the constant routine procedures pre- and post-treatment with bright lights. There were significant delays in all the temperature rhythm phase parameters.

Figure 1 shows an apparent decrease in amplitude of the temperature rhythm following the bright light treatment. The individual curve-fitted amplitude estimates indicate that the mean decrease was significant [$t(8) = 2.85$, $p < 0.02$]. This decrease seems to arise entirely from the decreased 24-hour fundamental amplitude ($p < 0.05$) because the 12-hour harmonic amplitude does not change. A further curve fit was applied to the temperature data of each subject, allowing the period of the "24-hour" fundamental to vary in order to derive a best fit period length of the circadian component. The mean best fit period length was 22.6 hours and 22.4 hours pre- and post-treatment with bright lights, respectively. These means were not significantly different nor did they vary significantly from 24 hours ($p > 0.05$). The time of dim light melatonin onset (DLMO) was estimated by taking the first 2-hour measure that was 100% above the average daytime level. A paired t test showed there was a significant delay in DLMO from before bright lights (2113 hours) to after bright lights [2327 hours; $t(8) = 4.26$, $p = 0.0027$].

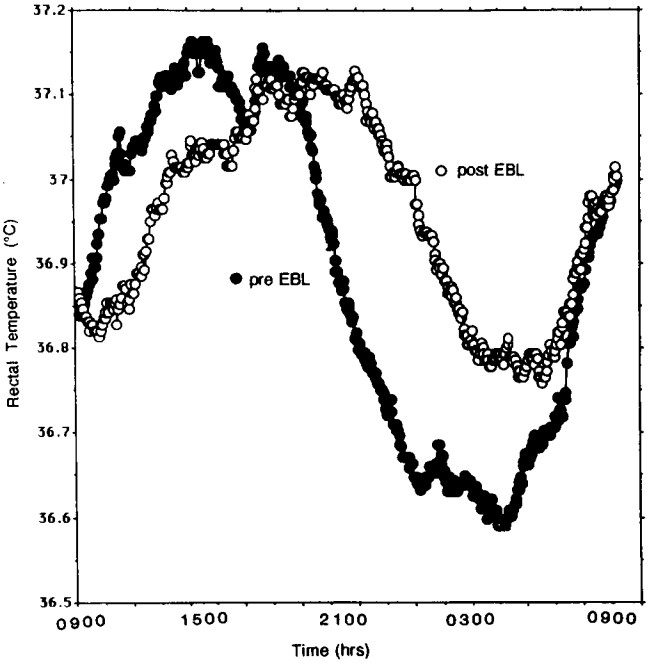

FIG. 1. Group mean rectal temperatures measured in the constant routines before (filled circles) and after (open circles) evening bright light (EBL) treatment.

Sleep parameters

Table 2 shows the group means before and after bright light treatment for the estimated sleep parameters of sleep onset time, final wake up time and total sleep time. There was no significant difference in sleep onset time. However, the subjects' final wake-up time was significantly delayed by a mean of 1 hour 12 minutes and their total sleep time was significantly lengthened by 1 hour 13 minutes.

TABLE 1. *Mean 24-hour clock times of the temperature rhythm phase parameters, mean amplitudes in degrees centigrade and fitted period length of the "24-hour" fundamental pre- and post-treatment with bright lights and* t *test of the difference between the group means*

Variable	Pre-treatment	Post-treatment	t	p[a]
Complex fitted T_{min}	0231	0422	2.47	0.05
Complex fitted T_{max}	1535	1942	6.78	0.001
Acrophase, 24-hour fundamental	1447	1824	4.45	0.01
Acrophase, 12-hour harmonic	0132	0649	6.25	0.001
Amplitudes (°C)				
Complex (24-hour + 12-hour)	0.31	0.23	2.85	0.05
24-hour fundamental	0.30	0.22	2.54	0.05
12-hour harmonic	0.06	0.08	0.09	ns
Fitted period length "24-hour" fundamental	22.6 hours	22.4 hours	0.09	ns

[a] Two-tailed p values.

TABLE 2. *Means of sleep onset, final wake-up time and total sleep time (hours and minutes) pre- and post-treatment with evening bright lights and separate bright light post-treatment days 1–5. Asterisks indicate a significant difference from the pre-treatment mean*

	Pre-treatment mean	Post-treatment mean	Post-treatment days 1	2	3	4	5
Sleep onset	2253	2300	2240	2315	2304	2304	2255
Wake-up time	0459	0611**	0630**	0558*	0556**	0620**	0608*
Total sleep time	5 hours 13 minutes	6 hours 26 minutes	7 hours 24 minutes	5 hours 43 minutes	5 hours 59 minutes	6 hours 36 minutes	6 hours 36 minutes

* $p < 0.05$.
** $p < 0.01$.

Table 2 also shows these means of the estimated sleep parameters separately for days 1–5 after treatment with bright lights. The significant differences of means before and after between bright light treatment are indicated with asterisks (paired *t* tests).

Dim red light control condition

Before bright light treatment, subjects 1 and 2 had exposure to the dim red lights on two consecutive evenings. After dim red light exposure, these two subjects had a small advance of their mean T_{min} and T_{max} of 15 minutes and 30 minutes, respectively. There was no change in their DLMO after the dim red lights. Following their recovery sleep after the constant routine, the sleep parameters of sleep onset time, final wake-up time and total sleep time remained within 30 minutes of their times before treatment in dim light. Because of these negative results and because the placebo control procedure added considerably to the cost of the study and efforts of the subjects, further subjects were not tested with the placebo control procedure.

DISCUSSION

Circadian phase of early morning insomniacs

Inability to initiate sleep before 0100–0200 hours has been predicted (9,29) and confirmed to be associated with a phase delay in the circadian temperature rhythm (23). Conversely, early morning awakening insomnia has been predicted to be associated with an advanced circadian temperature rhythm (9,29,30). A mean temperature minimum at 0231 hours was found in the nine early morning awakening insomniacs of the present study. This compares with 0315 hours for the good sleepers and 0718 hours for the sleep onset insomniacs in a previous study (23).

The mean age of subjects in the previous study was 32 years, whereas the mean age of the nine insomniacs in the present study was 53.4 years. Early morning awakening insomnia does appear to be age related (31). Metanalyses of population surveys of sleeping problems have found that virtually all surveys show greater prevalence of problems with sleep maintenance and early awakening in older age groups (32,33). One of the reasons for this age effect may arise from changes in the endogenous circadian timing mechanism. Weitzman et al. (34) and Monk and Moline (35) found that older subjects had a shorter period length of the endogenous temperature rhythm than young subjects. This is consistent with the findings for the older sample in our study, which found mean period estimates of 22.6 hours and 22.4 hours pre- and post-treatment with lights. Those older individuals whose period length is shorter than 24 hours would have a chronic tendency to phase advance in the 24-hour environment. Therefore we would expect more individuals with phase-advanced rhythms and early morning awakening insomnia in an older population. To separate the phase-advance effect from an effect of age per se, the temperature rhythms of early morning awakening insomniacs should be compared with those of age-matched good sleepers. We would also predict a shorter endogenous period length in the early morning insomniacs compared with the age-matched good sleepers.

Effect of evening bright light on circadian rhythms

The main purpose of this study was to evaluate the effects of evening bright light stimulation on the circadian rhythms and sleep of early morning awakening insomniacs. Two consecutive evening sessions of bright light stimulation resulted in delays of the temperature and melatonin circadian rhythms. All phase markers of the temperature rhythm were significantly delayed. In particular, the curve-fitted temperature minimum was delayed by an average 1 hour and 51 minutes. This result is consistent with delays found in normal sleepers (17). The 2-hour and 14-minute delay of DLMO following evening bright light is also consistent with studies of normal sleepers (36). However, the magnitude of these delays is somewhat less than that which would be predicted for a 4-hour bright light exposure terminating, on the average, 2.5 hours before the body temperature minimum. An earlier study using greater-intensity light (10,000–12,000 lux) for only one night terminating 2–3 hours before the temperature minimum found greater delays of temperature minimum (17). Less delay in the present study may be due to the lower light intensity. Thorough parametric studies of the phase-shifting effects of varying light intensities and timing would be of considerable therapeutic value.

Less phase delay in the present study than was expected may also be due to differences in subject population such as age, the insomnia or a different phase response curve. It has been suggested that a young, normal population has a greater delay response to evening light than advance response to morning light (5). It may be that an older population, particularly one selected for early morning awakening, has a less responsive delay portion than advance portion of their phase response curve (37). This is an empirical question of some clinical relevance to these insomniacs and would be worthwhile to investigate.

The bright light exposure from 2000 hours to 2400 hours required all subjects to be awake later than normal on two consecutive nights. Furthermore, the constant routine procedure several nights prior to the bright light required total sleep deprivation. Could this sleep

deprivation and delay of bedtime have caused the delay of circadian rhythms? This seems unlikely for three reasons: 1) the two subjects who underwent an initial constant routine and a placebo condition, in which bedtimes were delayed by the same amount, showed no change of circadian rhythm phase; 2) there is evidence that a delay of sleep period in dim light laboratory conditions produces no or little delay of rhythms, whereas bright light stimulation before normal bedtimes while holding sleep times constant does produce circadian rhythm delays (15,38,39) and 3) we have shown that for a sample of eight subjects measured on two constant routines separated by a week, there was an overall difference of <5 minutes in circadian rhythm phase markers (40). Therefore, it seems most likely that the circadian rhythm delays are a result of the evening bright light stimulation.

Two evenings of bright light stimulation also resulted in a decreased amplitude of the temperature rhythm. This is consistent with past studies, which showed a temperature rhythm amplitude suppression following bright light stimulation during the subjective night (41). It was shown that for bright light across the middle of the subjective night, the amplitude was suppressed maximally to 42% of the initial amplitude (41). However, bright light presented earlier or later than midway through the subjective night had less suppression effect (42). In the present study, bright light was presented in the first half of the subjective night and resulted in a mean amplitude that was 74% of the initial amplitude. This appears consistent with the proposed amplitude response curve (42). In the present harmonic analysis, it would appear that amplitude suppression was confined to the circadian component because the 12-hour harmonic amplitude was unchanged. The 12-hour harmonic phase was delayed by evening bright light without a change in amplitude. However, the circadian component showed both a decrease in amplitude and a delay in phase.

Effect of bright light on sleep measures

From a clinical point of view, the most important outcome of bright light stimulation is the effect on sleep. There was an average delay of estimated final wake-up time following evening bright light of 1 hour and 12 minutes. Because estimated sleep onset times and nighttime awakening did not change, estimated total sleep time was increased by a mean of 1 hour and 13 minutes. This improvement was statistically and clinically significant. The amount of improvement compares favorably with that from pharmacologic treatment (43,44).

In the first two subjects, there was no evidence that dim red light resulted in rhythm delays or improvement of sleep. Yet following bright evening light, these same two subjects showed an average delay of T_{min}, DLMO and final wake-up time of 6.4 hours, 2.0 hours and 1.4 hours, respectively. Because this was considered a pilot study, we did not think that a complete control comparison between bright light and dim light was warranted. The present significant results would now suggest the usefulness of such a comparison.

It is interesting that there was no change of recorded bedtime or sleep onset time. It may be that before treatment these subjects stayed up later than normal with respect to the timing of their circadian systems. The closer someone approaches the T_{min} phase, the greater his subjective and objective sleepiness. Normal sleepers go to bed about 5 hours before their T_{min}, as did our subjects following treatment. However, before treatment the subjects' bedtimes were only about 3.5 hours before their T_{min}. This would be consistent with their subjective reports that by their chosen bedtime they were usually very sleepy and fell asleep quickly. They also reported that they could go to sleep earlier and often did so inadvertently while reading or watching television. However, because of social reasons and also the belief that earlier bedtimes would eventually result in even earlier wake-up times, they purposely delayed bedtime until 2230–2300 hours. We infer that these individuals have a chronic tendency to phase advance. This could result from endogenous circadian rhythms that are shorter than 24 hours.

Duration of effect

Over the 5 days following bright light treatment there was no indication in the sleep parameters of a return to pre-treatment values. Table 2 shows that final wake-up time was still significantly later and total sleep time was significantly longer than the pre-treatment means and there was no trend apparent in these values over the five post-treatment days. Post-treatment day 1 shows a later wake-up time and longer total sleep time than subsequent days. However, this could have arisen from some continued recovery sleep following the first night of recovery sleep. Recovery was from two nights of reduced sleep as well as one sleepless night in the constant routine. However, over days 2–5 there is no indication in the sleep parameters of a diminishing effect.

Nevertheless, some of the subjects over the period 1–3 months following treatment contacted us to request further use of the light boxes for evening light stimulation. These subjects were again experiencing early morning awakening, presumably as a result of a phase advance of their circadian rhythms. Follow-up monitoring of sleep as well as circadian rhythm phase clearly needs to continue for longer than a week after

treatment. We presently recommend 1–2 months of follow-up measurement.

Early morning awakening and depression

Early morning awakening (EMA) is considered to be a symptom of endogenous depression because it is commonly found in depressive populations (45,46). However, among subjects in this study who were selected for early morning awakening insomnia, there were few apparently depressed subjects. The median BDI score was in the normal range and only one subject out of 15 had a score in the moderate to severe range. Therefore, although early morning awakening may be commonly found in the population of depressives, in this sample, which selected for EMA, depression was not common.

CONCLUSIONS

This pilot study shows that evening bright light stimulation has promise as an effective nondrug therapy for early morning awakening insomnia. EMA insomniacs appear to have phase-advanced circadian rhythms that are then delayed by evening bright light to a more normal phase position. They also show a delay in final wake-up time that results in an increase of more than an hour of total sleep time. Several questions raised by this study, such as the efficacy of other bright light stimulus protocols, the comparison with a placebo control and the duration of the effect, still need to be investigated.

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