 Enhancement of REM sleep during extraocular light exposure in humans

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Enhancement of REM sleep during extraocular light exposure in humans. Am J Physiol Regulatory Integrative Comp Physiol 280: R1606–R1612, 2001.—This study examined the effects on sleep of light administered to an extraocular site. A 3-h photic stimulus was applied to the popliteal region during sleep in 14 human subjects. Each subject also underwent a control stimulus condition during a separate laboratory session. The proportion of rapid eye movement (REM) sleep during the 3-h light administration session increased by an average of 31% relative to the control condition. The frequency but not the duration of REM episodes was altered during light exposure, thereby shortening the REM/non-REM (NREM) cycle length. No other sleep stages were significantly affected during light administration nor was sleep architecture altered after the light-exposure interval. These results confirm that extraocular light is transduced into a signal that is received and processed by the human central nervous system. In addition, they expand to a novel sensory modality previous findings that REM sleep can be enhanced by sensory stimulation.

MAMMALIAN SLEEP IS CHARACTERIZED by an alternation between two distinct sleep states: rapid eye movement (REM) and non-REM (NREM) sleep. Whereas the function(s) of neither of these states has yet been established, a large body of evidence indicates that REM sleep is intimately tied to learning and memory. In addition to studies detailing the detrimental effects on memory of experimentally induced REM-sleep deprivation in animals and humans (41, 42, 47), evidence that REM sleep is associated with learning and memory processes in humans is provided by reports that substantial increases in REM sleep follow presleep learning (43) and that overnight improvement on certain performance tasks depends on the presence of REM sleep (21). Furthermore, age-related memory impairment is associated with significant decreases in REM sleep (9, 32, 46), and several of the drugs used to treat such age-related cognitive deficits have been shown to increase REM sleep in young healthy individuals (12, 16, 38).

These facts suggest that enhancing REM sleep amounts might have beneficial effects in humans. However, means of enhancing REM sleep in humans are rare. Most sedative or hypnotic drugs tend to have negligible or suppressant effects on REM sleep (34), although acetylcholinesterase inhibitors (16, 38, 40), cholinergic agonists (1), and dihydroxyepiandrosterone (DHEA) (12) have been reported to increase REM sleep in humans. Some reports have indicated that intense cognitive activity or visual stimulation before sleep onset increases REM sleep (6, 43). In addition, a handful of studies in mammals (2, 8, 28, 29, 45), including one report in humans (33), indicates that various sensory stimuli administered during sleep result in acute REM-sleep enhancement.

The current study evaluated the effects on polysomnographic sleep in humans of a novel sensory stimulus in the form of photic stimulation at an extraocular, peripheral site. This study follows our previous report that timed extraocular light exposure resulted in circadian phase shifts in humans similar to the phase shifts obtained with administration of ocular light (3). In humans, the phase-response curve to light dictates that the largest magnitude phase shifts are obtained at a circadian phase that coincides with the usual nocturnal sleep period. We reasoned, therefore, that the effectiveness of bright light-treatment regimens for circadian rhythm sleep disorders might be improved if exposure to extraocular light also induces phase shifts during sleep. As a result, we undertook the study of the effects of timed exposure to extraocular light during sleep on sleep and circadian variables.

One vital empirical consideration was whether extraocular exposure to photic stimulation during sleep affected sleep quality or architecture. On the basis of the literature detailing the effects of other sensory stimuli on sleep parameters, we hypothesized that administration of this novel sensory stimulus during sleep would enhance REM sleep.

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Methods

The paper describes the methods used in a study to investigate the effects of extraocular light on sleep. The protocol was approved by the Weill Medical College of Cornell University's Committee on Human Rights in Research. All subjects provided written informed consent and were compensated for participation.

Overview of protocol. Subjects were studied in two laboratory sessions, each lasting at least 10 days. Each session began with an adaptation night. During the next 48 h, subjects underwent either the active condition, in which the popliteal fossae were exposed to light during sleep, or the control condition, in which no exposure to light occurred. The study included the following steps:

1. The stimulus exposure interval times were identical for each subject (column contains subject’s initials) between the active and control lab sessions. Data from this sleep period during both the active and control lab sessions excluded from analysis because sleep efficiency either during stimulus-exposure interval or during entire sleep period was <80%.

2. Subjects were studied in two laboratory sessions, each lasting at least 10 days. Each session began with an adaptation night. During the next 48 h, subjects underwent either the active condition, in which the popliteal fossae were exposed to light during sleep, or the control condition, in which no exposure to light occurred (see Experimental conditions and light-delivery device for description of experimental conditions). Of 16 subjects who completed the protocol, 3 were exposed to the stimuli (active or control) for one 3-h period during each lab session; the other 13 were exposed to the stimuli twice (i.e., for two 3-h periods during two consecutive sleep periods) during each lab session. The clock times of the stimulus-exposure intervals for individual subjects are listed in Table 1.

3. Clock times for the active- and control-stimulus intervals were matched for each subject across lab sessions. For example, if a subject received light between 0700 and 1000 during the active condition, he also received light between 0700 and 1000 at the control condition. In addition, the clock time of the 3-h stimulus intervals was the same on both nights within a lab session for those subjects who received two stimulus “pulses.” However, as is apparent from Table 1, the stimulus-interval times (and sleep periods) were scheduled to occur at various times throughout the 24-h day for the entire subject sample.

Experimental conditions and light-delivery device. For both the active and control conditions, the fiberoptic pad of one light-delivery device (Biliblanket Plus Phototherapy System, Ohmeda) was placed on the popliteal region of each of the subject’s legs. The Biliblanket consists of a flexible metal housing containing a halogen lamp and a fan to disperse heat generated by the halogen lamp. Illumination from the halogen bulb exits the metal housing via 2,400 optic fibers encapsulated in a flexible opaque tube. The fibers terminate in a flexible woven pad, ~15 x 10 x .64 cm thick and covered with plastic. Because the pad is comprised of optic fibers, minimal heat is emitted from the pad. The fiberoptic pad emits illumination in the wavelengths from 450 to 540 nm (blue-green range), and with a lux meter placed directly on top of the pad, an intensity of ~13,000 lx is measured.

In the active condition, the fiberoptic pad was covered with a transparent sheath (for purposes of hygiene and comfort). In the control condition, the fiberoptic pad was covered with an opaque black fabric sheath. (A lux meter placed directly on top of the black fabric sheath covering an activated Biliblanket pad detects 0 lx). Just before lights out, subjects lay in bed with the bed linens covering their legs. One covered fiberoptic pad was placed on each popliteal fossa while subjects averted their eyes. The fiberoptic pads were then wrapped completely with a polyester athletic bandage. Subjects were permitted complete freedom of movement and could sleep in any desired position. Automatic timers were set to activate the Biliblanket devices at predetermined clock times and to turn them off 3 h later. The Biliblankets were activated in both the active and control conditions. Therefore, the slight noise from the fan and the light emitted through the vented metal housing (<15 lx at 1 m) were identical across experimental conditions.

Scoring of polysomnographic records. Sleep was polysomnographically (PSG) recorded throughout all sleep periods during which an active or control stimulus was administered. All PSG records were blind coded so that the subject and condition from which they were recorded could not be determined by the scorers. They were then scored in 30-s epochs according to standard criteria (37) by trained scorers. Sleep parameters calculated for the entire sleep period included the minutes and percentage of each sleep stage, latencies to sleep onset (the first occurrence of stages 2, 3, 4, or REM sleep), slow-wave (combined stages 3 and 4) and REM sleep, number of awakenings, minutes and percent wakefulness after sleep onset, and average duration of the

Table 1. Times of stimulus exposure for individual subjects

<table>
<thead>
<tr>
<th>Subject</th>
<th>Stimulus Exposure 1</th>
<th>Stimulus Exposure 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>SG</td>
<td>0200–0500</td>
<td>0200–0500</td>
</tr>
<tr>
<td>HB</td>
<td>0700–1000</td>
<td>0700–1000</td>
</tr>
<tr>
<td>MM</td>
<td>0400–0700</td>
<td>0400–0700</td>
</tr>
<tr>
<td>TR</td>
<td>0500–0800</td>
<td>0500–0800</td>
</tr>
<tr>
<td>GD</td>
<td>2200–0100</td>
<td>2200–0100</td>
</tr>
<tr>
<td>AG</td>
<td>1630–1930</td>
<td>1630–1930</td>
</tr>
<tr>
<td>DP</td>
<td>2330–0230</td>
<td>2330–0230</td>
</tr>
<tr>
<td>BA</td>
<td>0100–0400</td>
<td>0100–0400</td>
</tr>
<tr>
<td>GK</td>
<td>2400–0300</td>
<td>2400–0300</td>
</tr>
<tr>
<td>AK</td>
<td>1930–2230</td>
<td>1930–2230</td>
</tr>
<tr>
<td>SD</td>
<td>1100–1400</td>
<td>1100–1400</td>
</tr>
<tr>
<td>UR</td>
<td>0200–0500</td>
<td>0200–0500</td>
</tr>
<tr>
<td>LR</td>
<td>0200–0500</td>
<td>0200–0500</td>
</tr>
<tr>
<td>GM</td>
<td>0100–0400</td>
<td>0100–0400</td>
</tr>
<tr>
<td>RA</td>
<td>0500–0800</td>
<td>0500–0800</td>
</tr>
<tr>
<td>LO</td>
<td>0500–0800</td>
<td>0500–0800</td>
</tr>
</tbody>
</table>

The stimulus exposure interval times were identical for each subject (column contains subject’s initials) between the active and control lab sessions. Data from this sleep period during both the active and control lab sessions excluded from analysis because sleep efficiency either during stimulus-exposure interval or during entire sleep period was <80%.
REM/NREM cycle [defined as the number of minutes from the first epoch of a REM period to the first epoch of the next REM period, with discrete REM periods (50), defined as any occurrence of REM sleep separated from another occurrence of REM sleep by more than 15 min]. These measures were also calculated for three discrete portions of each sleep period: sleep onset until activation of the Bililankets (pre-stimulus), the 3-h stimulus interval (during stimulus), and the interval from deactivation of the Bililankets until the terminal awakening (poststimulus). Although the duration of the pre- and poststimulus segments varied both within and between subjects, the during-stimulus segments were 3 h in duration for all subjects. Moreover, the during-stimulus segments occurred at identical clock times within a subject from sleep period to sleep period as well as between that subject’s active and control sessions.

Because the primary aim of the study was to assess the effects of light exposure during sleep, we applied the following criteria to each sleep period: if the proportion of time spent asleep during the stimulus interval in either the active or control condition was <80% or if the proportion of time spent asleep throughout the entire sleep period (from sleep onset to terminal awakening) was <80%, the data from that night and the subject’s matching record from the corresponding condition were excluded from further analyses.

Application of these criteria resulted in the exclusion of six pairs of records (i.e., 12 of the 58 records obtained, or 20%) from analyses. Of the pairs of records that did not meet the inclusion criteria, the record leading to exclusion was distributed evenly (3 each) between active and control sleep periods (see Table 1). Two pairs of records each were excluded from two individuals, thus completely excluding these subjects from subsequent analysis. The two remaining pairs of records excluded from analysis were from two individuals who were administered stimuli twice during each lab session; the records excluded were from the second stimulus administration for both subjects. Thus the data set analyzed was from 14 subjects (mean age 37 ± 13 yr, range 25–68 yr; 13 males, 1 female); nine of these subjects contributed two records in each condition (18 pairs of PSG records); 5 contributed one record in each condition (5 pairs of PSG records). Thus a total of 23 matched pairs of PSG records (46 records total) were included in these analyses.

Data analysis. Primary analyses focused on whether sleep was altered during administration of active and control stimuli (during stimulus). Of secondary interest was whether sleep was altered in the remainder of the sleep period immediately after the stimulus intervals (poststimulus). Thus sleep parameters were compared first across the 3-h active-light vs. control-stimulus intervals and then across the poststimulus segments. The frequency and duration of discrete episodes of REM sleep (50) as well as the REM/NREM cycle lengths were similarly compared between active and control conditions. In addition, the REM/NREM cycle length was compared within each condition from the during-stimulus to poststimulus segments. Unless otherwise stated, two-tailed paired $t$-tests were used to compare active vs. control conditions. Results are reported as means ± SD.

RESULTS

Comparisons of sleep-stage variables across experimental conditions indicated that the number of minutes spent in REM sleep during the 3-h light interval increased significantly from a group average of 27.0 ± 13 min in the control condition to 35.5 ± 14 min in the active condition [$t(22) = 3.06, P < .01$]. This 8.5 min change in the group average represents a 31% increase in REM sleep during that period over control levels. An increase in REM sleep was observed in 12 of the 14 subjects studied [in 19 of the 23 matched pairs of sleep periods; binomial test $P(12) = 0.006$]. Nearly two-thirds of the subjects (9 of 14) exhibited increases in REM sleep greater than the group average. For individual subjects, the absolute change in minutes of REM sleep ranged from −19 to +35 min during light exposure (Fig. 1A), which corresponds to a mean rela-

![Fig. 1. A: percentage of rapid eye movement (REM) sleep during stimulus administration in control and active conditions. Values from matched control and active sessions are connected. Dotted lines connect the paired stimulus sessions during which the percentage of REM sleep was less in active than in the control session. The wide range of %REM values (e.g., 0–44% for control intervals) reflects the fact that although the clock times of light exposure were identical for the active and control sessions for a given subject, the 3-h stimulus sessions occurred at varying clock times/circadian phases across subjects, i.e., at times during which the propensity for REM sleep is both high and low (5). B: % change in minutes of REM sleep during the stimulus session for individual trials (% change from control to active session), arranged in order of magnitude, from −41% to +209%. The median % change in minutes of REM during light exposure was an increase of 30%; denoted by arrow. The mean change was +47%.](http://ajpregu.physiology.org/Downloadedfrom)
tive change in the proportion of REM sleep across conditions of +47% (range ~41% to +209%; Fig. 1B).

Only REM sleep was significantly altered by light administration. The significant increase in REM sleep was accompanied by small decreases in wakefulness and all NREM sleep stages (Table 2). None of the changes in other sleep stages or in wakefulness during the light interval was statistically significant. In addition, the changes in sleep were limited to the stimulus session; neither the amount of wakefulness, nor any stage of sleep, including REM sleep, differed between active and control conditions for the remainder of the sleep period (Table 2).

The duration of individual REM periods did not differ between active and control conditions (control: 22.4 ± 12.4 min vs. active: 22.8 ± 13.5 min, not significant). Rather, the observed augmentation of REM sleep was due to an increase in the number of discrete REM episodes that occurred during the 3-h stimulus sessions (Wilcoxon’s signed rank test of REM episode frequency in the control vs. active-light conditions: 1.17 ± 0.72 vs. 1.59 ± 0.59 episodes; Z = 2.11, P < 0.05). The number of REM episodes occurring in the rest of the sleep period did not differ between conditions. The increase in REM-period frequency necessarily resulted in a shortening of the REM/NREM cycle length during the fixed-duration stimulus session (Fig. 2). Indeed, an unpaired t-test comparing the duration of REM/NREM cycles that occurred completely within the stimulus session across conditions revealed that the mean cycle length was significantly shorter during the active (n = 14; 84.4 ± 7.6 min) than during the control condition (n = 11; 103.7 ± 6.5 min) [T(23) = 2.33, P < 0.05]. The mean REM/NREM cycle length did not differ between conditions either before or after the stimulus session (Fig. 2).

Although, as expected, the absolute amount of REM sleep obtained in a given sleep period was influenced by the time of day during which the sleep period and the light stimulus occurred (5), the observed enhancement of REM sleep was not time-of-day dependent. The 23 pairs of active and control stimulus sessions were categorized according to one of four 6-h bins (2400–0600, 0600–1200, 1200–1800, or 1800–2400) during which the majority of the 3-h stimulus occurred. There were 14 stimulus sessions in the first bin, and 5, 0, and 4 in the second, third, and fourth bins, respectively. The mean percent changes in REM sleep during the three time bins with data were 44 ± 68%, 43 ± 36%, and 62 ± 104%, respectively. A one-way ANOVA comparing the percent increase in REM sleep across time-of-day bins was not significant (F = 0.12, P = 0.89). A clearer demonstration of the fact that REM sleep increases occurred at times of day when the propensity for REM sleep is high and also at times when REM sleep propensity is low is illustrated by the following example. Three individuals who received a light pulse at 2200–0100, 0500–0800, and 1630–1930, respectively, exhibited changes in the percentage of REM sleep during the light interval of +30%, +39%, and +44%, respectively.

Comparisons of sleep variables on the first vs. second night of light administration (for the 9 subjects whose sleep data from 2 consecutive sleep periods were included) revealed no significant differences between the consecutive sleep periods. The lack of first-to-second-sleep-period variation in the active condition suggests that there was not a “habitation effect” to the photic

Table 2. Sleep-stage percentages for segments of sleep periods occurring before, during, and after a 3-h interval of light exposure or a control stimulus to the popliteal fossae of human subjects during sleep

<table>
<thead>
<tr>
<th>Sleep Period Segment (Relative to Stimulus Interval)</th>
<th>EEG Stage</th>
<th>Wake</th>
<th>1</th>
<th>2</th>
<th>3/4</th>
<th>REM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prestimulus Control</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td></td>
<td>9(9)</td>
<td>5(3)</td>
<td>37(10)</td>
<td>31(12)</td>
<td>18(15)</td>
</tr>
<tr>
<td>Active</td>
<td></td>
<td>12(12)</td>
<td>6(6)</td>
<td>32(15)</td>
<td>33(19)</td>
<td>17(13)</td>
</tr>
<tr>
<td>During</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td></td>
<td>12(10)</td>
<td>6(3)</td>
<td>41(11)</td>
<td>27(15)</td>
<td>14(5)*</td>
</tr>
<tr>
<td>Active</td>
<td></td>
<td>8(5)</td>
<td>6(3)</td>
<td>38(14)</td>
<td>28(17)</td>
<td>20(6)</td>
</tr>
<tr>
<td>Poststimulus Control</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td></td>
<td>15(12)</td>
<td>9(9)</td>
<td>44(12)</td>
<td>11(7)</td>
<td>21(11)</td>
</tr>
<tr>
<td>Active</td>
<td></td>
<td>17(24)</td>
<td>6(4)</td>
<td>42(20)</td>
<td>11(9)</td>
<td>24(20)</td>
</tr>
<tr>
<td>Entire sleep period</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td></td>
<td>14(8)</td>
<td>6(2)</td>
<td>41(8)</td>
<td>25(13)</td>
<td>18(5)</td>
</tr>
<tr>
<td>Active</td>
<td></td>
<td>11(5)</td>
<td>6(2)</td>
<td>41(11)</td>
<td>24(12)</td>
<td>21(6)</td>
</tr>
</tbody>
</table>

Values are means (±SD). Wake- and sleep-stage percentages expressed as a proportion of minutes during the indicated segment (e.g., for entire sleep period, percentages were calculated as (minutes of stage / minutes from bedtime until final awakening) × 100). *Paired t-test, control vs. active percentage stage REM during stimulus sessions (P < 0.05). EEG, electroencephalogram; REM, rapid eye movement.
stimulus, because there was not a decrease in REM sleep amounts or any other change in sleep parameters from the first to the second stimulus periods.

Because extraocular light administration can affect the circadian timing system in vertebrates, including humans (3, 11, 17, 44), it is conceivable that the increase of REM sleep was due to an immediate shift in the timing of REM sleep, either within or after the light-exposure interval. To evaluate the possible immediate effects of extraocular light exposure on REM period timing, the latencies from stimulus-on to 1) the first occurrence of REM sleep during the stimulus presentation and 2) the first occurrence of REM sleep in the remainder of the sleep period after the stimulus presentation were calculated for each subject in both conditions. The average time to the first REM episode during stimulus administration did not differ between control and active conditions (control: 63 ± 50 min vs. active: 62 ± 36 min, not significant). Nor did the latency from stimulus-on to the first REM episode after the stimulus session differ across conditions (control: 228 ± 61 min vs. active: 221 ± 42 min). Together, these results suggest that an immediate shift in the timing of REM sleep was not responsible for the observed enhancement of REM sleep during light exposure. Moreover, an immediate phase shift (advance or delay) in REM-sleep expression would presumably result in differences in the amount of REM sleep not only during the 3-h light interval, but in the remainder of the sleep period after light exposure, as well. Such was not the case (Table 2).

**DISCUSSION**

The finding that extraocular light presentation during sleep results in an immediate enhancement of REM sleep provides further evidence that light presented to a site other than the eyes is transduced into a signal that can influence brain function in humans. Our previous study investigating the effects of extraocular light administration demonstrated that exposure of the popliteal fossae to a bright-light stimulus can affect the circadian timing system in humans (3). A 3-h pulse of bright light induced phase shifts in the circadian rhythms of body core temperature and melatonin similar to those obtained with ocular light exposure. In that study, subjects were exposed to light during wakefulness. In the current study, the light signal administered to a peripheral site during sleep was transmitted to the central nervous system and influenced the generation of REM sleep. Specifically, this sensory stimulus resulted in a group mean increase in REM sleep during the 3-h stimulus interval of 31% over control levels. This effect was not only robust, but also consistent. All but two of the subjects (86%) exhibited an increase in REM sleep. This finding is in contrast to several recent studies that have failed to demonstrate an effect of extraocular light administration on other central nervous system activities (10, 14, 18, 25, 27, 53).

Enhancement of REM sleep by extraocular light stimulation may be explained by several possible mechanisms that are not necessarily mutually exclusive. Effects of photic stimulation on peripheral nerve transmission (49) and smooth muscle tissue (47) have been documented. In addition, recent reports have established that peripheral tissues in vertebrates, including humans, are involved in transducing photic information for purposes of circadian rhythm entrainment (31, 51, 52). Once the light signal is transduced by a putative peripheral photoreceptor, the (as yet unidentified) signal might be transported via the vascular system to the brain (35), a process potentially involving an increase in peripheral blood flow velocity via a nitric oxide–related mechanism (22).

Neurotransmitters possibly involved in affecting REM sleep include acetylcholine, serotonin, and/or glutamate. It is generally acknowledged that the initiation and maintenance of REM sleep depends on cholinergic networks (4, 15, 23, 39). Cholinergic pathways that are activated by sensory stimuli (28) could partially account for the observed effects on REM sleep. Alternately, serotonergic activity, which is, in part, responsible for maintaining NREM sleep and inhibiting the initiation of REM sleep (19, 20, 23) is transmitted from the dorsal raphe nuclei during sleep to brain structures involved in the modulation of REM sleep (19, 23, 36). The dorsal raphe, in particular, have been shown to modulate noncircadian responses to light (30); thus serotonin might play a role in changes in REM sleep resulting from light administration during sleep. Yet another possibility is provided by evidence that implicates the excitatory neurotransmitter glutamate in the control of phasic events during REM sleep [e.g., eye movements, muscle twitches (24, 26)]. Glutamate has been identified as the synaptic neurotransmitter in the retinohypothalamic tract, which carries the signal of ocular light from the eye to the suprachiasmatic nuclei in the hypothalamus (26). Glutamate, acting in alternate light-transducing pathways, might also be responsible for the observed REM-sleep changes.

Would a longer interval of light exposure lead to proportional increases in REM sleep? Results from previous studies have suggested that the effect on REM sleep induced by sensory stimulation appears to depend on several factors, including presleep brain-activation levels (2), stimulation modality (13), and whether or not the sleep period was preceded by an intensive learning session (13, 28). In addition, the timing of sensory stimulation relative to the onset of REM periods or even individual rapid eye movements (33) may affect the manner in which REM sleep is altered by sensory stimuli. In light of results from such studies, any hypotheses concerning effects of longer-duration light administration on REM sleep must be regarded with caution. However, a study using auditory stimulation for varying durations and across multiple sleep periods in rats (45) indicated that there was no habituation effect to this stimulus, suggesting that further increases in REM sleep may be possible with
more prolonged (or altered schedules) of photic stimulation.

In conclusion, it should be pointed out that in the current study, although every effort was made to ensure that all possible influences (e.g., noise, heat from the halogen bulb, stimulus timing) other than pupillary light exposure were identical between active and control conditions, it was not possible to completely blind subjects to conditions. That is, subjects may have been aware that the opaque fabric sheath was in place during one laboratory session but not during the other. Because subjects were unaware of the study’s objectives or even of which was the active and the control condition, we consider it unlikely that such knowledge was responsible for the observed, systematic increase in REM sleep.

**Perspectives**

Our finding may have both basic and clinical implications. A large body of literature indicates that REM sleep is intimately linked to learning and memory processes in humans. Administration of extraocular light in this study resulted in an average within-subject increase in REM sleep of 31% during the light-exposure interval, with an absolute increase of up to 35 min. This degree of enhancement is comparable with or larger than the increase in REM sleep after administration of acetylcholinesterase inhibitors (16, 38, 40), DHEA (12), or other modalities of sensory stimulation (2, 7, 28, 29, 33, 45). As such, our finding that REM sleep is enhanced during extraocular light exposure may conceivably be used as a tool for studying the regulation of REM sleep as well as a novel approach to examining the relationship between REM sleep and memory in humans. Because means of augmenting REM sleep are rare, pursuit of this initial finding seems warranted.

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