Intermittent bright light and exercise to entrain human circadian rhythms to night work

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Baehr, Erin K., Louis F. Fogg, and Charmane I. Eastman. Intermittent bright light and exercise to entrain human circadian rhythms to night work. Am. J. Physiol. 277 (Regulatory Integrative Comp. Physiol. 46): R1598–R1604, 1999.—Bright light can phase shift human circadian rhythms, and recent studies have suggested that exercise can also produce phase shifts in humans. However, few studies have examined the phase-shifting effects of intermittent bright light, exercise, or the combination. This simulated night work field study included eight consecutive night shifts followed by daytime sleep/dark periods (delayed 9 h from baseline). There were 33 subjects in a 2 × 2 design that compared 1) intermittent bright light (6 pulses, 40-min long each, at 5,000 lx) versus dim light and 2) intermittent exercise (6 bouts, 15-min long each, at 50–60% of maximum heart rate) versus no exercise. Bright light and exercise occurred during the first 6 h of the first three night shifts. The circadian phase marker was the demasked rectal temperature minimum. Intermittent bright-light groups had significantly larger phase delays than dim-light groups, and 94% of subjects who received bright light had phase shifts large enough for the temperature minimum to reach daytime sleep. Exercise did not affect phase shifts; neither facilitating nor inhibiting phase shifts produced by bright light.

The light/dark (L/D) cycle is the strongest and most important zeitgeber (time cue) for the circadian clock, and it is well established that appropriately timed light and dark periods can phase shift human circadian rhythms (5, 6, 9). Simulated night-work studies have shown that appropriately timed medium intensity (≈–1,200 lx) or bright (≈–5,000 lx) light can help entrain circadian rhythms to a daytime sleep/dark (5/D) period (4, 7, 9, 16, 18). Light treatment is typically administered continuously over several hours. However, it is not always practical or possible for people to receive long durations of continuous medium intensity or bright-light exposure due to various environmental constraints (e.g., work schedules and duties). Preliminary studies have examined the effects of intermittent bright light (administered in pulses) on circadian rhythms and found that the effects were not as robust as with continuous light. For example, intermittent bright light (9,600 lx, 5 min every 25 min for 5 h) caused smaller phase shifts than continuous bright light during the same time period (15), and intermittent light (200 lx, 10 min every 20 min for 90 min) suppressed melatonin production less than continuous light exposure at both 200 and 111 lx for the same time period (2).

In addition to the L/D cycle, physical activity can serve as an effective zeitgeber to the circadian clock. Wheel running can induce phase shifts and entrain the circadian rhythms of rodents. Specifically, activity produces the largest phase-shifting effects in rodents during the subjective day or the inactive phase of their circadian cycle (20–22).

There is recent evidence that physical activity or exercise during the inactive phase can shift circadian rhythms in humans. A previous simulated night work study in our laboratory (8) indicated that moderate intensity exercise during the night shift produced larger temperature-rhythm phase shifts compared with a sedentary control condition (6.6 ± 2.5 vs. 4.2 ± 3.4 h during days 5–8 of night work relative to baseline; means ± SD). In this study, the 5/D period was delayed 9 h from baseline, and the exercise involved eight 15-min exercise bouts at 50–60% of maximum heart rate during the first three night shifts. The difference between the groups did not reach statistical significance until degree of morningness/eveningness was accounted for (used as a covariate), because evening types in the control group tended to have larger phase delays.

Studies by another group indicated that both low-intensity exercise of 3-h duration and high-intensity exercise of 1-h duration during the night caused phase delays by the following day (3, 25). In the first of these studies, a constant routine was either uninterrupted (control condition) or was interrupted with a 3-h bout of low-intensity exercise (between 40 and 60% of peak VO2) that occurred at various times during the night (25). Phase shifts were assessed by measuring the onset of the rise of plasma thyrotropin (TSH) and melatonin on the evening just prior to the stimulus versus the day immediately following the stimulus. The partial phase-response curve (PRC) of these data indicated that the largest phase delays were observed when the middle of the exercise bout was 3–5 h before the estimated temperature minimum (Tmin). The second of these studies compared a 3-h bout of low-intensity exercise (same as before) and a 1-h bout of high-intensity exercise (40 min at 75% of peak VO2 with 10 min of warm up and 10 min of cool down), with the exercise centered at 0100 to a control condition (3). Phase shift was assessed in the same manner as in the previous study. TSH delayed 18 ± 8 min (mean ± SE) in the control condition, 78 ± 10 min in the low-intensity exercise condition, and 95 ± 19 min in the high-intensity exercise condition. Melatonin delayed 23 ± 10 min in the control condition, 63 ± 8 min in the

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low-intensity exercise condition, and $55 \pm 15$ min in the high-intensity exercise condition. Thus both types of exercise bouts produced greater phase delays than the control condition.

In humans, the effects of bright light and exercise on the circadian system have been studied independently. However, it is not known what the effect would be if both of these stimuli were presented together. Evidence from studies in rodents suggests that the effects of light and activity on the circadian clock interact in a complex manner. Light had both the effect of attenuating the phase shifts from exercise as well as enhancing the phase shifts from exercise, depending on when the two stimuli were presented (17, 19, 23, 24).

The purpose of this study was to determine whether intermittent bright light, intermittent exercise, and a combination of the two can help entrain human circadian rhythms to a night-work, day-sleep schedule. Both the bright light and the exercise were timed to occur during the phase-delay portion of their respective PRCs to entrain subjects to a 9-h delay of the S/D period. We predicted that larger phase shifts would be observed for bright light compared with dim light and for exercise compared with no exercise and that the combination of bright light and exercise would be more effective than either alone.

**MATERIAL AND METHODS**

Subjects. Thirty-three healthy young subjects (17 females, 16 males), aged 23.8 $\pm$ 4.6 yr (mean $\pm$ SD), completed the study. They had no evident sleep, medical, or psychological disorders as assessed by telephone and in-person interviews and several questionnaires including the Minnesota Multiphasic Personality Inventory-2. Participants were not taking any prescription medications except for four women who took oral contraceptives. Subjects signed informed consent forms and were paid for their participation.

Design. This was a between-subjects $2 \times 2$ design with factors light (bright vs. dim) and exercise (yes vs. no) during the night shift (see headings and number of subjects in each group in Table 1). The subjects in the bright light + exercise group alternated between sitting quietly in bright light and exercising in dim light. The bright light + no exercise group followed the identical schedule of intermittent bright light, and the dim light + exercise group followed the identical schedule of intermittent exercise.

S/D and night-shift schedule. There were 7 days of baseline with night sleep followed by 8 days of simulated night shifts with day sleep (see Fig. 1). S/D times during the simulated night-shift portion of the study were shifted 9 h later than S/D during baseline. Participants slept at home in rooms that we made dark by covering windows with black plastic. Subjects were required to remain in bed in the dark during the designated 8-h S/D periods, even if they could not sleep. During baseline, S/D periods were close to or slightly later than habitual sleep times, as recorded on a sleep chart that each subject kept for at least 1 wk prior to beginning the study. The scheduled baseline bedtime (in Central Standard Time) ranged from 2200 to 0200, and the average was 0014 $\pm$ 67 min (mean $\pm$ SD).

Subjects spent their first three night shifts in the laboratory under the supervision of a research assistant and the remaining five night shifts at home. During the three laboratory night-shift sessions, up to three subjects sat at a round table playing games (when they were not exercising) to ensure they were awake and interacting and were allowed a 5-min break every hour to get up and stretch. During the night shifts spent at home, subjects were allowed to move about in their homes but were required to stay indoors in dim light ($<500$ lx) and to refrain from exercising.

Light exposure. Subjects in the bright-light groups were exposed to intermittent pulses of bright light ($\sim5000$ lx, 40 min) alternating with dim light ($<500$ lx, 20 min) for the first 6 h of the first three night shifts. Three light boxes were spaced around the perimeter of a large round table, shining in toward the center, such that each subject could sit directly opposite one light box $<1.3$ m away. For the bright-light groups, the overhead ceiling fixture was fully illuminated as were the light boxes on the table. The ceiling fixture contained eight 122-cm cool white fluorescent lamps, and the Apollo Light Systems light boxes ($77.5 \times 61 \times 11.8$ cm) contained four U-shaped cool white fluorescent lamps. During the dim-light laboratory conditions, only two of the overhead lamps were illuminated, and the light boxes were off, resulting in $<500$ lx. During the remaining five night shifts that subjects spent at home, all subjects were required to remain in lighting conditions $<500$ lx.

Subjects wore special dark glasses with top and side shields (Supervisor, $\sim7\%$ transmittance) when they went outside during daylight throughout the study. Because real night-shift workers typically have to travel home from work after their night shifts and may be exposed to daylight at this time, subjects were required to go outside for at least 5 min during their “travel-home time” (the hour between the end of the night shift and the beginning of S/D). Compliance with the light-exposure rules throughout the study was monitored using measurements of light intensity collected once per minute by a photosensor connected to the portable monitor, which was also used to measure body temperature. The photosensor was worn on the chest during scheduled awake hours throughout the study. Figure 1 shows a representative light-exposure pattern from a subject in one of the bright-light groups.

Exercise bouts. Subjects in the exercise groups pedaled on a stationary cycle ergometer (Monarch model 818E) for 15 min
of each hour during the first 6 h of the first three night shifts (i.e., 6 times/night shift). Subjects took turns using the stationary cycle, which was situated near the table that contained the light boxes. For the bright light + exercise group, the light boxes were always on and the subjects wore the special dark glasses during the 15-min exercise so that they received <500 lx. Then, they were required to take a 5-min break (outside of the room, in dim light <500 lx) after each exercise bout. Thus they received the same pattern of light exposure as the subjects in the bright light + exercise group in which the light boxes were turned on for 40 min and off for 20 min. Figure 2 shows raw temperature and the bright-light exposure schedule for a subject in the bright light + exercise group. The increase in body temperature during the six exercise bouts alternating with the bright-light exposures stands out clearly.

Exercise intensity was tailored to each individual’s capacity by measuring maximum heart rate with a maximal cycle ergometer test to voluntary exhaustion during the baseline week. These tests were conducted at the University of Chicago Cardiac Stress Laboratory using the same model ergometer that was used during the study. For the 15-min exercise bouts during the night shifts, subjects cycled at 50–60% of maximum heart rate. They spent an average of 13.06 ± 0.84 min (mean ± SD) in this target heart rate zone.

Sleep. After waking from each scheduled S/D period, subjects estimated the times of sleep onset, awakenings during sleep >5 min, and final awakening on a daily sleep log. Subjects were asked to refrain from napping but were not penalized for unintentional naps. Subjects completed a sleep log following a nap. Sleep logs were verified for accuracy by comparison to activity data, which were collected in 1-min bins by an activity monitor (Ambulatory Monitoring) worn on the nondominant wrist. Subjects were questioned about any obvious periods of low activity that were not reported on sleep logs as naps and occasionally adjusted their estimates. Sleep durations within the 8-h S/D periods were calculated from daily sleep logs, with awakenings >5 min subtracted.

Additional procedures. To encourage compliance with the sleep schedule, subjects were required to call the laboratory voicemail system at bed time, wake time, one-half hour after waking, and every 2 h during at-home night shifts. Subjects visited the laboratory every 2–3 days to have their temperature, photosensor, and wrist activity data downloaded and checked. Compliance with scheduled in-bed times were verified with actigraphy data. Photosensor data were used to check that subjects were going outside during the travel-home time. Subjects were allowed to choose whether to consume caffeine during the study. However, caffeine consumption was required to remain consistent, was limited to the first 4 h after scheduled wake, and was recorded on a daily event log. Subjects were instructed to abstain from alcohol and were informed that they would be visited and given a random breathalyzer test (Alco-Sensor III; Intoximeters) and, if they did not pass, they would be dropped from the study.

The experiment was conducted July 1996 through December 1997. All groups were run in all seasons and subjects who participated during summer months had air-conditioned bedrooms to provide a comfortable temperature while sleeping. Before or during baseline, subjects completed the Horne-Ostberg Morningness-Eveningness Questionnaire (MEQ) (13) and the Circadian Type Inventory (CTI) (1). The CTI has two independent factors: flexible/rigid and languid/vigorous.

Temperature recordings and data analysis. Core body temperature was continuously monitored using a flexible, disposable rectal thermistor connected to a portable monitor (AMS-1000, Consumer Sensory Products) that stored measurements once per minute. The probes were inserted to maintain a constant depth of 10 cm.

To reveal the endogenous component of the temperature rhythm, raw temperature data were “demasked” to compensate for the decrease in body temperature associated with laying down and sleeping and the increase associated with activity (10, 16, 26). A demasking factor (DF) was added to temperature values recorded around the scheduled sleep periods. To account for the gradual cooling off associated with rest and the gradual increase in temperature associated with activity, demasking followed a trapezoidal function; the DF increased from zero to the maximum (DFmax) during the first 60 min of scheduled in-bed time and decreased from the maximum to zero during the 60 min following scheduled wake time.

Because the amplitude of the circadian temperature rhythm varies among subjects, the magnitude of the DF was tailored to each individual. Each subject’s DFmax was derived from the average of the baseline temperature for each 24-h period (1400 to 1400 CST); 3) a mean baseline amplitude was calculated by averaging the daily amplitudes of the last five baseline days (for some, fewer days or earlier baseline days were used because of missing data or changes in menstrual phase); 4) the DFmax was 20% of this mean baseline amplitude.

For each female subject, the effect of menstrual phase on body temperature amplitude (14) was accounted for by calculating a DFmax for each menstrual phase. Menstrual phase (follicular or luteal) for women not on oral contraceptives (OC) was identified by counting forward and backward from the day of menses onset and by noting the day of the temperature rise from the follicular to luteal phase. Menstrual phase for women taking OC was identified by the 21 days during which exogenous hormones were taken and the 7 days without exogenous hormones. The menstrual phase of three females not on OC and three females on OC changed during baseline, and the DFmax for the follicular and luteal phases were calculated from the baseline data. Six other females not on OC and one female taking OC had a change in their menstrual phases during the night-work portion of the study. For these subjects, one DFmax was calculated from...
Table 1. Magnitude of phase shift

<table>
<thead>
<tr>
<th></th>
<th>Bright Light</th>
<th>Dim Light</th>
<th>Total</th>
</tr>
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<tbody>
<tr>
<td>Exercise</td>
<td>7.7 ± 2.7</td>
<td>5.7 ± 3.2</td>
<td>6.6 ± 3.0</td>
</tr>
<tr>
<td>n = 8</td>
<td>88%</td>
<td>56%</td>
<td>71%</td>
</tr>
<tr>
<td>(4M, 4F)</td>
<td></td>
<td>(6M, 4F)</td>
<td></td>
</tr>
<tr>
<td>No exercise</td>
<td>7.9 ± 1.0</td>
<td>4.8 ± 2.9</td>
<td>6.3 ± 2.6</td>
</tr>
<tr>
<td>n = 8</td>
<td>100%</td>
<td>50%</td>
<td>75%</td>
</tr>
<tr>
<td>(4M, 4F)</td>
<td></td>
<td>(2M, 6F)</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>7.8 ± 2.0</td>
<td>5.3 ± 3.0</td>
<td>94%</td>
</tr>
</tbody>
</table>

Values are means ± SD, average of last 4 night shifts relative to last 5 days of baseline. Circadian temperature-rhythm phase shifts (in hours), percentage of subjects whose temperature minimum (average of last 4 days of night work) fell within daytime sleep-dark, no. of subjects, and no. of males (M) and females (F) in each group.

baseline and the average temperature amplitudes calculated by Kattapong et al. (14) were used to algebraically determine the other $DF_{max}$. Mean (±SD) $DF_{max}$ were 0.27 ± 0.05 °C for males and 0.23 ± 0.04 °C for females.

The circadian $T_{min}$ was estimated for each subject for each day by fitting a 24-h cosine curve to each 24-h segment of demasked data. The 24-h segments began at 1400 for days 1-7 and at 2300 for days 8-15 so that the S/D period also occurred at the same time within each 24-h section. Two average $T_{min}$ values were calculated for each subject: a baseline $T_{min}$ for the last 5 days of baseline (days 3-7) and a night-work $T_{min}$ for the last 4 days of night work (days 12-15). Each subject's temperature-rhythm phase shift was calculated as the difference between the average night-work $T_{min}$ and the average baseline $T_{min}$.

ANOVA was performed, with factors light (bright vs. dim) and exercise (yes vs. no). Loglinear analysis (12) was also performed to compare the percentage of subjects in each group whose $T_{min}$ fell within daytime S/D (average for the last 4 days of night work). The z value is the z associated with the parameter in the loglinear analysis. All group data are expressed as means ± SD, unless otherwise stated.

RESULTS

Table 1 shows the magnitude of the phase shift and the percentage of subjects whose $T_{min}$ fell within daytime S/D (during the last 4 days of night work). The subjects in the bright-light groups had larger phase shifts than the subjects in the dim-light groups, and more bright-light subjects had $T_{mins}$ that reached daytime S/D. Exercise had no significant effect on the phase shifts. ANOVA on the magnitude of phase shifts revealed a main effect of light [$F(1,29) = 8.14, P < 0.01$]. There was no main effect of exercise [$F(1,29) = 0.16$] and no interaction [$F(1,29) = 0.35$]. The dim light + exercise group had a slightly larger shift than the dim light + no exercise group, but this difference did not reach statistical significance [$t(15) = -0.61, P = 0.55$]. Loglinear analysis for the percentage of subjects whose $T_{min}$ fell within daytime S/D paralleled the statistics for the magnitude of phase shift; there was a significant main effect of light ($z = 1.96, P = 0.05$), no effect of exercise ($z = -0.31, P = 0.74$), and no interaction ($z = 0.95, P = 0.34$).

Figure 3 shows the $T_{min}$ for each subject during baseline and during the last 4 days of night work. All but one of the subjects in the bright-light groups had $T_{mins}$ that reached daytime S/D. Only about one-half of the subjects in the dim-light groups had $T_{mins}$ that reached daytime S/D. Due to research assistant error, two subjects in the bright light + no exercise group received the wrong pattern of light exposure during the first night shift. They were exposed to bright light for 55 min of each hour rather than 40 min of each hour. However, it does not appear that the added light exposure produced larger phase shifts in these subjects (see Fig. 3).

There were no differences among the groups in MEQ scores (bright light + exercise: 51.7 ± 10.1; bright light + no exercise: 45.3 ± 10.3; dim light + exercise: 45.6 ± 11.7; dim light + no exercise: 47.9 ± 13.1). There was a tendency for morning-type subjects to shift more in the bright-light groups (bright light + exercise: $r = 0.52, P = 0.19$; bright light + no exercise: $r = 0.38, P = 0.35$; all bright-light subjects: $r = 0.40, P = 0.13$) but for evening types to shift more in the dim-light groups (dim light + exercise: $r = -0.65, P = 0.06$; dim light + no exercise: $r = -0.38, P = 0.35$; all dim-light subjects: $r = -0.53, P = 0.03$). There were no differences among the groups in CTI languid/vigorous scores (bright light +...
exercise: 33.1 ± 3.1; bright light + no exercise: 33.4 ± 9.1; dim light + exercise: 32.0 ± 6.4; dim light + no exercise: 34.6 ± 8.8) or in CTI flexible/rigid scores (bright light + exercise: 22.1 ± 3.6; bright light + no exercise: 26.1 ± 5.2; dim light + exercise: 27.0 ± 5.0; dim light + no exercise: 27.5 ± 4.9). Amount of phase shift did not correlate significantly with CTI languid/vigorous scores (r = 0.16, P = 0.38) or flexible/rigid scores (r = -0.17, P = 0.34).

Subjects in the bright-light groups slept more during the last four daytime S/D periods than those in the dim-light groups (Table 2). ANOVA showed a main effect of light [F(1,29) = 10.06, P < 0.01], no main effect of exercise [F(1,29) = 0.19], and no interaction [F(1, 29) = 0.07]. Subjects in the bright-light groups also napped less than subjects in the dim-light group (average of 3 ± 8 min vs. 23 ± 28 min/day in the last 4 days of the study). As expected, greater phase shifts (last 4 days relative to baseline) were associated with longer sleep durations within designated 8-h S/D periods (r = 0.55, P < 0.01, n = 33) and less napping (r = -0.53, P < 0.01, n = 33) during the last 4 days of night work.

**DISCUSSION**

Phase-shifting effects of intermittent bright light. Subjects who received intermittent bright light during the night shift had significantly larger phase shifts than subjects who remained in dim light, and 94% of subjects who received bright light had phase shifts large enough for the Tmin to reach daytime S/D (compared with only 52% of dim-light subjects). Our results suggest that intermittent bright light may be sufficient to facilitate reentrainment to a night-shift, day-sleep schedule in a real-world setting.

Previous preliminary studies have found that the effects of intermittent light on the circadian system were not as robust as continuous light exposure (2, 15). Although we did not test a continuous light group in this study, we can compare these results with one of our previous simulated night-work studies, which had a similar design and the same bright-light intensity (18). In that study, subjects underwent a 9-h shift (either advance or delay) of the S/D period and received 3 h of continuous bright light that was timed to either “facilitate” or “conflict” with the 9-h shift of S/D, according to the light PRC. Our current bright light + no exercise condition, which had a 9-h delay of S/D, is similar to the facilitating light with a 9-h delay of S/D condition in the previous study. However, in the previous study, subjects received 3 h of continuous bright light for eight nights in a row in a moving pattern designed to hit the delay portion of the phase-response curve as the Tmin got later and later, and they did not wear special dark glasses outside. In the present study, subjects got intermittent bright light (for a total of 4 h/night) that occurred mostly before the Tmin, for only three nights and wore the special dark glasses while outside during waking hours. The phase delay of 8.8 ± 1.7 h in the previous study is similar to the 7.9 ± 1.0 h delay in the present study. Despite the obvious differences in design, this suggests that intermittent bright light may be as effective as facilitating entrainment to a 9-h delay of S/D as continuous bright light.

Phase-shifting effects of intermittent exercise. The pattern of exercise in this study did not help to entrain the circadian rhythms to a 9-h delay of S/D. The dim light + exercise group did have a slightly larger phase shift than the dim light + no exercise group (5.7 vs. 4.8 h), but this difference was not statistically significant. A power analysis indicated that we would need 364 subjects (182/group) to detect a significant difference between these two groups with a power of 0.80 and an alpha of 0.05. Thus the pattern of exercise we tested is not a practical solution for enhancing entrainment to a delay of S/D.

Our previous exercise study (8) had a similar design to the present study but only included the two dim-light groups. In that study, the exercise + dim light group shifted 6.6 ± 2.5 h, whereas the no exercise + dim light group shifted 4.2 ± 3.4 h. Thus the magnitude of the phase shifts in that study were similar to the present study except the exercise group shifted a little more and the no exercise group shifted slightly less. The difference between the exercise and no exercise groups in our previous study was at the borderline of statistical significance and an analysis of covariance (with MEQ score as the covariate) was necessary to reveal a significant difference between these two groups. In the present study, subjects cycled eight times per night shift, whereas, in the present study, exercise subjects had only six exercise bouts per night shift. The exercise intensity and the duration of each bout (15 min) were the same in both studies. Thus the subjects in our present study exercised 30 min less per night, a total of 90 min less than subjects in the previous study. Perhaps given the moderate intensity of exercise we used, a longer duration is needed for exercise to be effective. Evidence from animal studies supports this contention. There is a threshold in hamsters for the number of wheel revolutions that predict large phase shifts (20). In other words, if hamsters run in their wheels but do not complete a certain number of wheel revolutions, phase shifts will not be produced, even if this exercise is conducted during a portion of the PRC that is sensitive to activity.

It is of interest to compare our results with studies by another group that found exercise to be an effective zeitgeber to the human circadian clock (3, 25). Results from these studies indicated that nocturnal, low-intensity exercise significantly delayed the circadian rhythm of both TSH and melatonin and that nocturnal, high-intensity exercise significantly delayed the circadian rhythm of TSH compared with a control condition.

**Table 2. Average sleep duration within the 8-h daytime sleep-dark periods during last 4 days of the study**

<table>
<thead>
<tr>
<th></th>
<th>Bright Light</th>
<th>Dim Light</th>
<th>Total</th>
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<tbody>
<tr>
<td>Exercise</td>
<td>7.4 ± 0.6</td>
<td>6.6 ± 0.9</td>
<td>7.0 ± 0.9</td>
</tr>
<tr>
<td>No exercise</td>
<td>7.5 ± 0.5</td>
<td>6.8 ± 0.6</td>
<td>7.2 ± 0.7</td>
</tr>
<tr>
<td>Total</td>
<td>7.5 ± 0.5</td>
<td>6.7 ± 0.8</td>
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</table>

Values are means ± SD, in hours.
There are major differences between our study and their studies that could account for the fact that they found a significant effect of exercise and we did not. First of all, their studies did not involve a shift of the S/D period, whereas our subjects underwent large shifts of S/D, which make the overall phase shifts larger (including the phase shifts in the control group). The duration, intensity, type, and number of exercise bouts were also different, which may account for the differential results. In their studies, subjects exercised on a single night for 3 h at a low intensity on an arm and leg exerciser, or for 1 h at a high intensity on a stair climber. Our subjects cycled intermittently for a total of 90 min each night for three nights. The timing of the exercise was also different. In their studies, the exercise was centered at 0100, whereas, in our study, the exercise was timed to begin around the time that subjects normally went to sleep and continued intermittently for 6 h. There are obviously numerous differences in design between our study and the studies conducted by this other group, and future research should address which of these differences could account for the differential results.

Phase-shifting effects of bright light and exercise in combination. There was no interaction between the phase-shifting effects of bright light and exercise. Both stimuli were presented during portions of their respective PRCs that would be expected to produce delays. Although animal studies have found that the effects of light and activity on the circadian clock interact either by light attenuating or enhancing the phase-shifting effects of exercise, depending on when the two stimuli were presented (17, 19, 23, 24), no such effects were observed in our human subjects. It is possible that the L/D cycle was a much stronger stimulus to the circadian clock than the exercise and overrode any possible interaction between the two stimuli.

Individual differences. In the dim-light groups, greater eveningness was associated with larger phase shifts (there was a significant negative correlation between temperature-rhythm phase shift and MEQ score, \( r = -0.53 \)). However, this relationship was not found in the bright-light groups (bright light facilitated phase shifts for all types). These results suggest that in the absence of a strong phase-shifting stimulus, individual differences in morning/evening tendencies become more meaningful. It is important to point out that those with evening tendencies (and thus later \( T_{\text{min}} \)) had larger overall phase shifts and did not simply have to phase shift less for their \( T_{\text{min}} \) to reach daytime S/D.

Our previous study (8) found that in the no exercise + dim light group, evening types had larger phase shifts (MEQ score and the magnitude of the phase shift correlated, \(-0.69, P < .05\)), but for the exercise + dim light group, morningness/eveningness did not influence the magnitude of phase shift (\( r = -0.02 \)). This may at first appear to be contradictory to what we report in our present study because we found a tendency for evening types to have larger phase shifts in both of the dim-light groups (despite the exercise). However, if we assume that exercise in the previous study was a strong phase-shifting stimulus, but was not in this study, the results are consistent. In other words, when S/D is delayed and there is no other strong zeitgeber, those with evening tendencies shift more. In our present study, we found a tendency for morning types to shift more in the bright-light conditions. This may be due to the timing of the bright light relative to the \( T_{\text{min}} \), perhaps the light was presented during a stronger phase-delay portion of the PRC for morning-type subjects than for evening-type subjects.

Although the CTI was originally developed to identify which individuals would readily adjust to shift work (1, 11), it did not correlate significantly with amount of phase shift. This suggests that the CTI may not be useful for predicting individual differences in ability to reentrain to a shift of S/D.

Sleep. During the last 4 days of the study, subjects in the bright-light groups slept more (within the scheduled S/D periods) and napped less than those in the dim-light groups. This most likely reflects the fact that these subjects had larger temperature-rhythm phase shifts, and it is easier to sleep when sleep is attempted at a more appropriate phase of the circadian cycle. The fact that exercise did not increase sleep duration is consistent with exercise having no effect on the magnitude of the temperature-rhythm shifts.

Conclusions. This study indicated that intermittent bright light can facilitate circadian rhythm adaptation to the night shift. Future research should investigate the phase-shifting effects of even shorter durations of intermittent light, as this could have implications for real-world applications. The pattern of exercise tested did not help to entrain circadian rhythms to the delay of the S/D period. However, this does not necessarily mean that exercise is an ineffective zeitgeber to the human circadian clock. It is possible that the intensity and/or duration of the exercise was insufficient to have an effect on the circadian rhythms of these subjects. There was no interaction of the combination of the bright light and exercise; exercise neither facilitated nor inhibited the phase shifts produced by bright light. Future studies should test bright light in combination with a stronger exercise stimulus to further understand the interaction of these stimuli in humans.

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