Light masking of circadian rhythms of heat production, heat loss, and body temperature in squirrel monkeys

EDWARD L. ROBINSON AND CHARLES A. FULLER
Section of Neurobiology, Physiology and Behavior,
University of California, Davis, California 95616-8519

Robinson, Edward L., and Charles A. Fuller. Light masking of circadian rhythms of heat production, heat loss, and body temperature in squirrel monkeys. Am. J. Physiol. 276 (Regulatory Integrative Comp. Physiol. 45): R298–R307, 1999.—Whole body heat production (HP) and heat loss (HL) were examined to determine their relative contributions to light masking of the circadian rhythm in body temperature (Tb). Squirrel monkey metabolism (n = 6) was monitored by both indirect and direct calorimetry, with telemetered measurement of body temperature and activity. Feeding was also measured. Responses to an entraining light-dark (LD) cycle (LD 12:12) and a masking LD cycle (LD 2:2) were compared. HP and HL contributed to both the daily rhythm and the masking changes in Tb. All variables showed phase-dependent masking responses. Masking transients at L or D transitions were generally greater during subjective day; however, L masking resulted in sustained elevation of Tb, HP, and HL during subjective night. Parallel, apparently compensatory, changes of HL and HP suggest action by both the circadian timing system and light masking on Tb set point. Furthermore, transient HL increases during subjective night suggest that gain change may supplement set point regulation of Tb.

METHODS

Protocol. Six adult male squirrel monkeys (Saimiri sciureus) with an average body mass of 1.08 kg (0.95–1.26 kg) were studied. Animals were in a 24-h LD cycle of 12 h of L and 12 h of D (LD 12:12) in colony housing and for a week of acclimation to the experimental facilities before the experiment. Each animal was acclimated to a pelleted diet (Bioserv no. F0035 190-mg banana pellets) for at least 7 days before the experiment. Ambient temperature during acclimation was 27°C (± ca. 3°C), and ambient L intensity from an overhead cool white fluorescent fixture averaged 200 lx in the cage. D exposure was ~0 lx. At the beginning of the experiment, an animal was weighed and placed into a calorimeter system (27). The monkeys were unrestrained throughout the experiment. Ambient temperature in the calorimeter was 27 ± 0.5°C, within the thermoneutral zone of the squirrel monkey (30). L was supplied to the calorimeter by a fiberoptic lamp with a quartz halogen source (Dolan-Jenner Fiber Lite 180). Average L intensity was 200 lx. D exposure in the calorimeter was 0 lx.

During a 24-h acclimation period before the start of data collection and for the following 4 days, the animal was maintained in LD 12:12. Data from this 4-day period served as control data. The animal was then exposed to a nonentraining cycle of 2 h of L alternating with 2 h of D (LD 2:2) for 48 h, beginning at the expected time of L onset for the control LD 12:12 exposure (0700 Pacific Standard Time).

Measurements. Tb and activity were measured as previously described (27) using a biotelemetry transmitter (model
In this study, CO₂ production was not measured and an average respiratory quotient of 0.79 was assumed based on previous measurements (5 monkeys over 6 days). Continuous measurements of relative humidity with an electronic hygrometer (model 270, Setra Systems) were used to determine evaporative HL (EHL) and to correct gas measurements to fraction in dry air. Dry HL (DHL) was measured by direct calorimetry using a gradient-layer calorimeter (SEC-A-2401, Thermonetics).

Food and water were available ad libitum. Animals were supplied with a tap switch that operated an automated feeder (GS100, Gerbrands). Feeding was monitored continuously.

A PC-based hardware-software data acquisition system (Data Quest III, Data Sciences) was used for data acquisition and storage and for controlling lights and gas sampling, as previously described (27). All data were collected at 10-min intervals for later analysis.

Data analysis. Average rhythms over a 24-h period were determined by eduction of the data for each variable. In this procedure, all data for a variable at a given time of day are averaged, giving an average 24-h waveform. Activity waveforms for each animal were normalized (z transform) across LD cycles to permit comparison among animals. Relative masking of each rhythm was determined by spectral analysis using the linear-nonlinear least-squares method (28). This method fits cosine waves of different periods to the data and estimates the amplitudes of statistically significant spectral components. Spectral amplitudes were summed over 4-h bands, or windows, for periods between 2 and 28 h (e.g., 2-6 h, 22-28 h). Summed spectral amplitudes for dependent variables (Tb, HP, HL, activity, and feeding) were compared using two-factor multivariate ANOVA (MANOVA) (SPSS) by LD cycle and period. Periods between 2 and 6 h included masking components resulting from LD 2:2 exposure, and periods between 22 and 28 h included the endogenous circadian components. Pillai's trace statistic was used to determine significance of the MANOVA overall, and univariate F tests were used to determine significant effects on each dependent variable. Deviation contrasts were used to assess changes to both masking and circadian spectral windows.

To compare LD cycles, we calculated 2-h means for each variable in LD 2:2 and LD 12:12. Periods in which one L cycle presented L and the other D were tested for the effect of L by repeated-measures ANOVA (SPSS) on the difference between L and D means. In this analysis, D periods in LD 2:2 during subjective day were compared with L in LD 12:12 by subtracting D means from L means. Conversely, L periods in LD 2:2 during subjective night were compared with D in LD 12:12. A single within-subjects factor, circadian time (CT), was tested without grouping factors. Because our data for the ANOVAs consisted of L-D differences, testing the constant effect was equivalent to testing the null hypothesis that the overall difference between L and D, and thus masking response, was zero. Mauchly's test was used to ensure that univariate ANOVA assumptions were met.

Difference (reverse Helmert) contrasts were used to establish the time of day at which differences in response appeared, by comparing each 2-h mean to all preceding means. Acute responses to L were evaluated by comparing average transient responses from the first 30 min of L or D exposure in LD 2:2. Individual L-D differences were determined in comparison with the corresponding times in LD 12:12 and tested using repeated-measures ANOVA without grouping variables (within-subjects factor, CT). Difference contrasts were used to compare 2-h means for changes in masking response with time of day.

Cumulative totals were calculated for HP, HL, activity, and feeding from average waveforms for each animal. Total energy expenditure (in kJ/kg) was estimated using measured HP and HL (in W/kg) as average rates over each 10-min sampling interval. Feeding counts (pellets delivered over 10-min intervals) were summed to give cumulative totals. Because activity countersensitivity differed among animals, counts over 10-min intervals were scaled to the 24-h total in LD 12:12, which for each animal was assigned a value of 1. Sums for 24 h in the two LD cycles were compared using paired t-tests.

RESULTS

Rhythms in LD 12:12. Figure 1 shows data from two consecutive days for a monkey in LD 12:12. Prominent daily rhythms were observed in all variables, and all show entrainment to the LD cycle. Elevated values were seen during L periods (subjective day), and decreased values were seen during D periods (subjective night).

Rhythms in LD 2:2. Figure 2 shows data for the same subject in 48 h of LD 2:2. A robust 24-h circadian rhythm is still observed in all variables. L and D masking of all rhythms is now also apparent, although the degree of masking appears variable, depending on the circadian phase. Masking changes in Tb, HP, DHL, activity, feeding, and apparently EHL, increase in parallel.

Average rhythms. Figure 3 shows average waveforms for six monkeys in LD 12:12 and LD 2:2. A small phase advance of HP with respect to HL is seen, which accounts for the diurnal rise in Tb, as well as the diurnal decrease.

In the LD 2:2 condition, both L and D masking were seen in all variables. Increases during L exposure and decreases during D exposure were consistently observed. However, all rhythms also continue to show circadian variation. Time of day dependency was also evident for masking effects. During the subjective day, recovery of control levels of Tb, HP, and HL followed D masking; however, L masking during the subjective night was not followed by return to control rest period levels. Activity and feeding do not show this pattern and are strongly inhibited during D.

Masking as a function of time of day. Figure 4 presents a summary of 2-h means in LD 2:2 and LD 12:12. Masking changes in the form of L-D differences were tested with repeated-measures ANOVA and showed a significant effect of L on all variables (Tb, HP, F1,5 = 73.8, P < 0.001; HP, F1,5 = 131.9, P < 0.001; HL, F1,5 = 184.5, P < 0.001; activity z scores, F1,5 = 216.0, P < 0.001; feeding, F1,5 = 38.6, P < 0.01).

Difference contrasts showed that the L response of Tb increased at CT 12 relative to subjective day (F1,5 = 6.8, P < 0.05) and that it increased again at CT 16 (F1,5 = 12.9, P < 0.05). The L responses of HP and HL at CT 16 were significantly greater than at earlier times of day in difference contrasts (HP, F1,5 = 16.2, P = 0.01; HL, F1,5 = 14.7, P < 0.05). For activity, the masking response did not differ significantly by time of day.

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Fig. 1. Data from a representative monkey in light-dark (LD) 12:12, showing 2 days of data for body temperature (Tb), heat production (HP), dry heat loss (DHL), evaporative heat loss (EHL), activity, and feeding. LD cycle is indicated by bars at top of figure. Tb and HP increases occur in advance of L onsets. DHL, EHL, and activity change by 1.22 ± 0.33 °C on August 28, 2017.

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Fig. 2. Data from a representative monkey in LD 2:2, showing 48 h of data, plotted as in Fig. 1. LD cycle is indicated by bars at top of figure. Amplitude of masking component is relatively greater for HP, DHL, and activity than for $T_b$. 
however, feeding exhibited increased masking at CT 12 ($F_{1,5} = 11.4, P < 0.05$) compared with subjective day, followed by a decreased response at CT 20 ($F_{1,5} = 40.9, P < 0.001$).

Degree of masking. Spectral analysis by the linear-nonlinear least-squares method showed significant circadian and 4-h components in all variables in LD 2:2. Both circadian and ultradian components are apparent in LD 12:12. Figure 5 summarizes the significant spectral amplitudes for all animals. A two-way MANOVA (LD cycle × period) showed a significant difference of periodicities due to L masking (Pillais’ trace = 0.91, approximate $F_{25,300} = 2.66, P < 0.001$).

The interaction of LD cycle and period, due to masking, was significant for all variables in univariate F tests ($T_b, F_{5,60} = 10.6, P < 0.001$; HP, $F_{5,60} = 11.2, P < 0.001$; HL, $F_{5,60} = 9.0, P < 0.001$; activity, $F_{5,60} = 3.4, P < 0.01$; feeding, $F_{5,60} = 12.9, P < 0.001$). Deviation contrasts showed statistically significant differences in 2- to 6-h amplitudes, hence masking effects, for all variables ($T_b$, $F_{5,60}$...
For $T_b$, $HP$, and $HL$, but not for activity or feeding, the reduction in circa-
dian amplitude was also significant ($T_b$, $t_{60} = 3.21$, $P < 0.00001$; $HP$, $t_{60} = 4.86$, $P < 0.00001$; $HL$, $t_{60} = 8.64$, $P < 0.00001$).

The relative amplitude of the $T_b$ masking component was less than for the other variables. In LD 12:12, ultradians in the masking range (2–6 h) accounted for an amplitude of $0.14 \pm 0.03^\circ\text{C}$ (mean $\pm$ SE), or $6.6 \pm 1.4\%$ of total amplitude, and the circadian component for $1.06 \pm 0.05^\circ\text{C}$, or $52.4 \pm 1.1\%$ of total amplitude. In

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**Fig. 4.** Two-hour means ($\pm$ SE; $n = 6$) in LD 2:2 (circles) and LD 12:12 (squares). Open symbols indicate L periods, and filled symbols indicate D periods. *Significant difference contrasts, hence time of day differences in masking.

**Fig. 5.** Average amplitude ($\pm$ SE) of periodic components in LD 2:2 and LD 12:12 from linear-nonlinear spectral analyses ($n = 6$). st. dev., Standard deviation. *Statistically significant differences in deviation contrasts (2-way multivariate ANOVA, LD cycle x period), indicating spectral windows that changed significantly. $t$, Circadian period.
LD 2:2, the masking component, calculated as periods between 2 and 6 h, had an average amplitude of $0.41 \pm 0.04°C$, or $26.0 \pm 1.5\%$ of the total amplitudes of significant periodicities, and the circadian component, periods from 22 to 28 h, an average amplitude of $0.79 \pm 0.06°C$, or $50.8 \pm 2.3\%$ of total amplitude, indicating that the amplitude of the circadian component of $T_b$ in LD 2:2 remained greater than the masking component.

In contrast, HP and HL were more strongly masked. For HP, the amplitude of the masking component in LD 2:2 was $1.49 \pm 0.20$ W/kg, or $48.6 \pm 5.4\%$ of total amplitude, and the amplitude of the circadian component was $0.96 \pm 0.21$ W/kg, or $19.5 \pm 6.2\%$ of total amplitude. The masking component for HL in LD 2:2 averaged $1.08 \pm 0.12$ W/kg in amplitude, and the circadian component $0.96 \pm 0.21$ W/kg, accounting for $42.1 \pm 3.5$ and $34.9 \pm 3.0\%$ of total amplitude, respectively.

Activity and feeding were the most strongly masked variables. Masking of activity in LD 2:2, comparing normalized data, accounted for $1.34 \pm 0.31$ standard deviations, or $57.7 \pm 5.1\%$ of total amplitude, and the circadian component accounted for $0.34 \pm 0.15$ standard deviations, or $12.9 \pm 6.2\%$ of total amplitude. The masking component for feeding in LD 2:2 had an amplitude of $5.15 \pm 0.75$ counts/10 min, and the circadian component had an amplitude of $1.44 \pm 0.20$ counts/10 min, or $71.6 \pm 6.6$ and $24.1 \pm 6.8\%$ of total amplitude, respectively.

Rates of change due to masking. Figure 6 summarizes the deltas, showing average transient changes, standardized to units per minute, occurring within the first 30 min of exposure to either L or D for both LD 12:12 and LD 2:2.

Differences in transients between LD cycles were evaluated using repeated-measures ANOVA to test L-D differences for animals at six CT. There was a statistically significant influence of L on $T_b$ change ($\Delta T_b$, $F_{1,5} = 12.0$, $P < 0.05$), HP change ($\Delta HP$, $F_{1,5} = 62.3$, $P = 0.001$), and HL change ($\Delta HL$, $F_{1,5} = 10.5$, $P < 0.05$) but not for activity change (from z scores) or feeding change.

In both LD 12:12 and LD 2:2, the largest $\Delta T_b$ in both LD cycles (LD 2:2, $0.21 \pm 0.03°C$; LD 12:12, $0.25 \pm 0.02°C$) occurred in L at the start of the active period (CT 0). Comparing LD cycles, we found that difference contrasts showed an increased masking response of $\Delta T_b$ at CT 12 ($F_{1,5} = 60.2$, $P = 0.001$) and a significant transient decrease at CT 20 ($F_{1,5} = 8.17$, $P < 0.05$).

Both $\Delta HP$ and $\Delta HL$ showed positive (L) and negative (D) masking in LD 2:2. However, transients in HL apparently differed from HP in both amplitudes and time courses (Fig. 6). HL transients are typically of lower amplitude but exceed $\Delta HP$ transients at both CT 16 and CT 18 during mid-subjective night. Difference contrasts showed an increase in the masking response of HP (L-D difference) at CT 6 relative to CT 2 ($F_{1,5} = 25.3$, $P < 0.01$) but no other statistically significant changes over time in $\Delta HP$ or $\Delta HL$ transients. Transients in activity and feeding were more variable but showed no significant time of day differences.

Average daily metabolic expenditure, activity, and feeding. Time courses for accumulated HP, HL, activity, and feeding, averaged over 24-h periods, are shown in Fig. 7. Changes in slope, indicating rates of accumulation, show both circadian and masking components in LD 2:2, although variables differ in relative degree of masking. Although different patterns of accumulation are seen in the two photoperiods, total energy expenditure is not significantly different between LD 2:2 and LD 12:12. Total daily energy expenditure estimated by
HP was 418 ± 23 kJ in LD 2:2 and 391 ± 19 kJ in LD 12:12 (t = -2.72, P = 0.042). Estimated by HL, total daily energy expenditure was 391 ± 19 kJ in LD 2:2 and 374 ± 14 kJ in LD 12:12 (t = -1.83, P = 0.127). In contrast, activity is more strongly masked by L, and activity in LD 12:12 particularly so, as shown by the slope changes at L-D transitions. Total activity is also not significantly different between the two photoperiods (LD 12:12, standardized, mean = 1, SE = 0; LD 2:2, mean = 1.04 × LD 12:12 total ± 0.09, t = -0.41, P = 0.70). Feeding behavior, in contrast, shows a clear preponderance of masking over the circadian component, especially in LD 2:2. Near-zero slopes during D periods indicate virtual absence of feeding. However, with large intersubject variability, total feeding did not differ significantly between LD 2:2 (396 ± 31 counts) and LD 12:12 (320 ± 23 counts, t = -1.896, P = 0.12).

DISCUSSION

Masking has been suggested to augment circadian rhythmicity when it involves environmental variables with 24-h periodicity (23). There is also prior evidence for circadian variation in masking efficacy (3, 8, 12, 14). This study tested the hypotheses that 1) L masking of the Tb rhythm is due to regulated changes in both HP and HL and 2) masking responses of Tb are due to phase-dependent responses in HP and HL. The results support both hypotheses. Although not directly addressed in this study, the data are also consistent with action on Tb set point by both the circadian timing system and L masking.

Coordination of HP and HL changes in Tb masking response. Circadian variations in thermogenesis and HL occur along with the Tb rhythm (7, 20, 24). In LD 2:2, significant masking changes were seen in all variables, in addition to circadian rhythmicity. For all variables, L exposure produced positive masking and D exposure produced negative masking. Masking changes in Tb were typically accompanied by parallel masking responses in HP and HL, with different time courses (Fig. 6) and relatively greater magnitudes (Fig. 3). However, masking changes in HP and HL were also seen during late subjective night either without concomitant Tb changes or accompanied by an anomalous Tb response. In particular, increased HP and HL after L exposure at CT 20 result in decreased Tb. Masking responses of Tb thus appear to depend on the interaction of HP and HL changes.

Evidence for phase-dependent masking responses of HP and HL as well as Tb. Previous studies have shown time-of-day-dependent masking of rhythms in squirrel monkeys, including Tb (11, 14). This study shows that masking responses of HP and HL may also differ across the circadian cycle (Fig. 3). Two-hour means show relatively greater L-D differences during subjective night (Fig. 4) comparing LD 2:2 with LD 12:12 means. Persistent Tb elevation during subjective night in LD 2:2 is seen, however, and may result from redistributed activity, feeding, or as shown previously, redistributed sleep (11).

Transients appear to be greater during subjective day (Fig. 6), apparently due at least in part to a greater response in activity and possibly feeding. Increases in Tb are typically accompanied by a more rapid transient increase in HP relative to HL (Figs. 3 and 6), except during late subjective night. A transient decrease in Tb occurs after L exposure at CT 20, evidently resulting from a more rapid response of HL.
Evidence that L masking acts via T_b set point. Several studies have shown that coordinated changes in HP and HL produce the circadian T_b rhythm in humans through a phase lag in HL relative to HP (5, 22). In squirrel monkeys, this has been confirmed with measurements of whole body HL rhythms in LD 12:12 (27) and in data from the LD 12:12 control period of the present study. The lag in HL produces excess HP during the rising phase of the T_b rhythm and excess HL during the falling phase (5, 23) and is explainable as the result of set point changes and subsequent error signals (1). An alternate view favors independence of circadian drive from homeostatic temperature regulation, based on studies in rodents (24). However, rodents differ from our squirrel monkeys in several key respects, including nocturnality and lower body mass. We would thus anticipate somewhat different thermoregulatory responses due to these factors as well as due to greater surface-to-mass ratios and lower thermal inertia. A further distinction between this and prior studies is our ability to directly compare whole body HL with metabolic HP and T_b. Thus our interpretations may be based on differences in both species and methods.

The action of L masking on T_b set point in squirrel monkeys was suggested previously by the correlation of L intensity to T_b in continuous illumination (31). In the present study, masking changes in LD 2:2 are consistent with direct action of L on T_b set point. The positive masking response to L at most times of day consisted of transient increases in HP and T_b, although T_b was seen to decrease after L onset in late subjective night. After elevation of T_b increased HL appeared to compensate for elevated T_b and thus effectively regulate T_b around a new apparent set point. At most times of the circadian cycle, transient masking changes are followed by relatively constant T_b (Fig. 3). Continuously decreasing T_b during D between CT 10 and CT 12, however, might be interpreted as due to underlying circadian regulation.

Activity level has been shown previously not to account for circadian T_b changes (25). Masking changes in HP, HL, activity, and feeding in this study are also not accompanied by proportional changes in T_b at most times of day. Large masking changes in T_b may instead coincide with normal times of circadian adjustment, the beginning of subjective day and night, as shown in a prior study (11). The evidence for time-of-day-dependent HP and HL responses did not, however, support independent action of HP or HL, including thermogenesis of activity or feeding, in determining T_b. For example, large positive masking changes in HP and HL around CT 0 and CT 10 in LD 2:2 are accompanied by large T_b changes. However, T_b changes only slightly in response to D masking at CT 2, although large HP and HL decreases are seen. Conversely, large increases in HP and HL at CT 16 and CT 20 produce only small changes in T_b. Relatively constant T_b is seen after D onset at CT 18, when both HP and HL are decreasing.

Evidence for gain changes reinforcing set point regulation of circadian T_b rhythm. Time of day differences in magnitude of whole body HP and HL masking responses to similar stimuli might be viewed as instances of effector gain changes (18). This observation is consistent with prior evidence for time of day differences in specific effector systems. In particular, decreased circulating catecholamines and decreased capacity for vasoconstriction during rest have been suggested as a possible basis for day-night differences in HL (15, 29). There is also evidence that melatonin reduces T_b in humans (9, 10), and it has been suggested that melatonin may act by altering HL via vascular receptors (21, 32). Suppression of melatonin secretion by L has also been shown in squirrel monkeys as well as in humans (19). Sustained nocturnal elevation of T_b in LD 2:2 might be attributable in part to melatonin suppression from L exposure at CT 12 and CT 16. Other possible explanations might include diet-induced thermogenesis from feeding during L exposure or redistribution of sleep and activity (11).

Homeostatic or circadian regulation? Comparison of total daily HP, HL, and activity in LD 2:2 and LD 12:12 (Fig. 7) suggests that total energy expenditure was largely conserved, as suggested by Aschoff (4), although it may be redistributed due to masking. There is also evidence that the circadian timing system may influence total energy expenditure (26). Because we measured feeding behavior and not food consumption, it is not known if caloric intake was greater in LD 2:2. Similar total daily HP and HL in the two LD cycles and negligible changes in body mass (mean loss 0.4 g, SE 12 g; 0.4 ± 1.1% of body mass) argue against a change in maintenance energy requirements.

Masking and circadian changes do not appear to be simply additive. Interaction of circadian and masking influences on the T_b rhythm may derive from different central mechanisms (3); however, this study further demonstrates that they are not independent. Masking, for example, appears to obscure the circadian rise in T_b. From CT 22 to CT 24, when both LD cycles present D, the anticipatory circadian rise in T_b is absent in LD 2:2. In LD 2:2, however, T_b is elevated relative to LD 12:12, suggesting that the underlying set point change may be obscured. In both control (LD 12:12) and masking (LD 2:2) cycles, the same time courses of T_b rise are seen at CT 0, the beginning of the active period. Furthermore, control T_b values are attained during subsequent L intervals of the active period in LD 2:2, suggesting that the circadian component at this time. However, in LD 12:12, L masking appears to be permissive, rather than additive, with circadian drive.

Conversely, although the T_b decrease at CT 12 in LD 12:12 may result from combined action of circadian and masking drives, it is apparent that masking can overcome the circadian component at this time. However, in LD 2:2, the rapid increase in T_b at CT 12 due to L masking (ca. 0.25°C/min) is of the same order of magnitude as the ΔT_b at CT 0, which similarly suggests that L masking is permissive in reaching the active period T_b, which appears to be determined primarily by circadian phase.
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Address for reprint requests: C. A. Fuller, Section of Neurobiology, Physiology, and Behavior, Univ. of California, One Shields Ave., Davis, CA 95616-8519.

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