Constant light suppresses sleep and circadian rhythms in pigeons without consequent sleep rebound in darkness

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Berger, Ralph J., and Nathan H. Phillips. Constant light suppresses sleep and circadian rhythms in pigeons without consequent sleep rebound in darkness. *Am. J. Physiol.* 267 (*Regulatory Integrative Comp. Physiol.* 36): R945–R952, 1994.—Sleep patterns and circadian rhythms of body temperature, activity, body weight, and electroencephalographic (EEG) power spectra of pigeons were compared among three photic conditions: a 12:12-h light-dark cycle (LD), followed successively by constant bright (LL) and dim light (DD) periods. LL suppressed non-rapid-eye-movement and rapid eye movement sleep and circadian rhythms of the measured variables without producing increased drowsiness or other physiological or behavioral changes. Sleep patterns after LL-DD transitions also showed no evidence of prior sleep deprivation during LL. Sleep latency after LL-DD transitions was 93 min longer than after L-D transitions in LD. Total sleep and EEG slow wave activity during the first 24 h in DD did not differ from D in LD. Free-running circadian rhythms subsequently reappeared in DD after LL.

body temperature; melatonin; sleep homeostasis

MELATONIN is a major component of the avian circadian system, which has been modeled as a neuroendocrine loop between the pineal gland, eyes, and homologue of the mammalian suprachiasmatic nuclei (SCN), all of which have abundant melatonin receptors (7). Melatonin is synthesized from serotonin during darkness, primarily in the pineal gland and retinae. The rate-limiting enzyme in melatonin production is *N*-acetyltransferase (NAT). Constant bright light (LL) suppresses nocturnal synthesis of NAT and melatonin and circadian rhythms of feeding, activity (31), metabolic rate (20), body temperature (T_b) (2), heart rate (23), and electroencephalographic (EEG) slow wave activity (SWA) (32) in birds, and circadian rhythms of T_b and sleep in rats (11). LL also strongly suppresses or eliminates sleep in pigeons, which can then be rapidly restored by periodic or continuous intravenous melatonin infusions (19). The ability of melatonin to restore sleep in LL-exposed pigeons and to enhance nocturnal slow wave sleep in chickens (13) indicates its key role in the regulation of avian sleep.

In contrast to its effects on mammals (e.g., Refs. 4, 15, and 27), 24-h sleep deprivation in pigeons through exposure to constant light and sensory stimulation did not result in increased non-rapid-eye-movement (NREM) sleep or NREM SWA during the subsequent 12-h recovery period in darkness (25). An absence of compensatory recovery of sleep is contrary to homeostatic sleep theory (3). However, sleep rebounds during recovery might require periods of sleep deprivation longer than 24 h in birds, and/or suppression of sleep by light could be functionally different from instrumental methods of sleep deprivation that have been applied to mammals.

The objectives of the present study were to further elucidate the effects of LL and constant dim light (DD) on circadian rhythms of T_b, behavioral activity (ACT), body weight, EEG power spectral density (PSD), and sleep patterns in the pigeon. Three specific questions were addressed. *1*) Does prolonged LL-induced insomnia in pigeons result in progressive physiological and behavioral changes that resemble the detrimental effects of long-term sleep deprivation in rats (21)? *2*) Does prolonged suppression of sleep by LL result in subsequent compensatory increased amounts of sleep and/or EEG SWA when the pigeons are returned to darkness? *3*) Do normal circadian rhythms of the measured variables and sleep reappear when the pigeons are transferred from LL to DD, or does prolonged LL exposure produce permanent effects on the circadian and/or sleep systems?

METHODS

Animals. Seven adult male pigeons (*Columba livia*; weights 375–510 g) were studied. They were housed individually in stainless steel cages (46 × 76 × 60 cm) in a 12:12-h light-dark cycle (LD) with food and water ad libitum for ≥ 1 mo before the experiments began.

Surgery. The pigeons were chronically implanted under halothane anesthesia with cranial stainless steel screw electrodes to record the electroencephalogram (EEG) and electrooculogram (EOG), and with Teflon-coated stainless steel wire electrodes for the neck electromyogram (EMG). The electrode contacts were inserted into an Amphenol pin strip connector, which was permanently affixed to the skull with acrylic dental cement. Finally, a MiniMitter temperature telemetry capsule (model VM) was inserted into the intraperitoneal cavity. In a second operation, three of the pigeons were also implanted with a brachial venous catheter for infusion of melatonin and saline in another experiment (19). Two pigeons underwent several 4-day fasts as part of another experiment (to be reported elsewhere).

Apparatus. The pigeons were placed in a 28 × 43 × 50 cm Plexiglas cage inside a shielded incubator (20.0 ± 0.5°C) within an acoustic chamber (45-dB attenuation). Food and water were provided ad libitum. Light intensity at the cage floor was ~200 lx (white incandescent bulbs or fluorescent tubes) during LL and the light (L, 0700–1900 h) portion of LD and was < 3 lx (red incandescent bulb) during DD and the dark (D) portion of LD. Ambient temperature was measured with a thermistor calibrated to an electronic thermometer (Physitemp/Sensortec), and light intensity was measured with a photoconductive cell calibrated to a photometer (Licor LI-185B). The signal from the abdominal MiniMitter, which was proportional to T_b, was detected by an AM receiver beneath the floor of the recording cage and then used to latch and reset a counter driven by a 10-kHz clock. Ambient conditions and T_b clock count were read by a microcomputer-based acquisition

system (Motorola 6809 or Intel 80286) equipped with analog and digital input/output cards, and 1- or 2-min averages were saved to disk.

In the case of three pigeons, the grid floor of the cage was suspended from an electronic balance (model EK-1200A, A & D) that was independent from the rest of the cage. The output of the balance, monitored via an RS-232 interface, was used to generate 1-min averages of body weight and ACT. An average of stable readings for each minute was stored as body weight, and the number of changes in consecutive 1-s samples that exceeded a selected threshold (2 g) each minute was stored as ACT.

Electrophysiological measures were recorded via an electric-hydraulic swivel (that allowed the birds freedom of movement) on an 8-channel polygraph (Beckman R-411 Dynograph, chart speed 6 mm/s) and on VHS magnetic tape (Mitsubishi VCR) after digitization by a pulse code modulator (PCM8, Medical Systems). PSD was also calculated continuously using a fast Fourier transform routine (n = 2). One EEG channel (low pass-filtered, 30 Hz), connected to the computer via the analog-to-digital board, was sampled at 64 Hz, and 1-min average power spectra (0–32 Hz in 0.25-Hz bins) were calculated from 15 4-s rectangular windows. Windows with EEG values that exceeded selected thresholds were rejected as artifacts by the computer.

Procedure. At least 1 wk after surgery, the pigeons were placed in the recording cage for a 1- to 2-day adaptation period in LD and then were connected to the recording cables for another 7 days of adaptation. Twenty-four-hour continuous polygraphic recordings were taken after ≥10 days of exposure to each of the three successive photic conditions: LD, LL, and DD. Recordings commenced when raster plots and periodograms displayed stable circadian T_b rhythms in LD or DD or absence of circadian rhythms in LL. A fourth 24-h recording commenced at the onset of the transition from LL to DD (1900 h).

Data analysis. The polygraphic records were coded and visually scored without knowledge of treatment condition for the following arousal states: waking, drowsy, NREM sleep, and rapid eye movement (REM) sleep, according to previous criteria (18). REM was scored in 5-s epochs and all other arousal states in 25-s epochs. The transitional phase of drowsiness, which is more prominent in birds than in mammals, is characterized by a polygraphic pattern intermediate between waking and NREM of high-voltage low-frequency EEG, rate of EOG activity $<1/s$ but $>1/5$ s, and an absence of phasic EMG activity. Behavioral correlates of the polygraphically defined drowsy episodes are body immobility and partial closure of the eyes, but with head and/or eye movements occurring at intervals of 1–5 s. Birds in which drowsiness was recognized include the pigeon (18, 25), goose (9), and emperor penguin (6).

Raster plots of T_b, body weight, ACT, and PSD were made for visual assessment of rhythms. Ten-day data sets, analyzed using a periodogram analysis (10), quantified the presence or absence and period of any rhythms between 18 and 30 h. Cosinor analysis (16) was used to determine the phase of T_b, body weight, ACT, and PSD in LD and DD conditions. Twenty-four-hour DD records were aligned using T_b acrophase, and subjective night onset in DD was designated as the mean phase angle between T_b acrophase and light offset in LD. Analysis of variance for repeated measures, followed by Newman-Keuls tests for multiple comparisons, where appropriate, were performed on values for each variable averaged over 12-h periods 0700–1900 h and 1900–0700 h for LD and LL (detectable or strong circadian rhythms were absent in LL), and consecutive 12-h periods beginning at the onset of subjective night for DD. The system to digitally collect and analyze EEG power spectra was fully functional for only two pigeons. Therefore, only descriptive statistics are reported for SWA (PSD 0.75–4.0 Hz), except that correlations between hourly percentage of arousal state and SWA were calculated separately for 12-h periods for each of these two birds. The corresponding Pearson r values from each bird were averaged after z transformation.

Because not all measured variables in each condition were obtained from all seven birds, the number of animals that yielded complete sets of data in each analysis is listed with the results.

RESULTS

Twenty-four-hour rhythms in LD. Twenty-four-hour rhythms of PSD, T_b, ACT, and body weight were evident in LD (Fig. 1). Sleep was almost entirely confined to D and was associated with increased SWA, decreased T_b, and ACT (Fig. 1, Table 1). NREM was lower ($P < 0.005$) and waking was higher ($P < 0.05$) during L than during D (Table 1). Although REM was completely absent during L, its meager and variable amount during D was not significantly greater.

SWA was lowest during wakefulness, increased during drowsy, and reached maximal levels during NREM (Fig. 2). Waking was negatively correlated with SWA during both D and L ($r = -0.83$, $P < 0.01$; $r = -0.80$, $P < 0.01$, respectively). Drowsy was positively correlated with SWA during L ($r = 0.80$, $P < 0.01$), as was NREM ($r = 0.79$, $P < 0.01$) during D. Because REM was dependent on prior NREM, REM was also positively correlated with SWA during D ($r = 0.50$, $P < 0.05$).

T_b was 1.0°C higher ($P < 0.01$) and SWA was 35.9% lower during L than during D (Table 1). The acrophase of T_b was 209.4° (0° = 1900 h clock time) ~1 h after the middle of the L period. Cosinor analysis of circadian rhythms of body weight and ACT showed mean phase angles from T_b acrophase of 138.4° for body weight and −5.3° for ACT (Table 2). Sleep was maximal during D and coincided with SWA (Fig. 3*A*), which had a mean acrophase of 174.6° after T_b acrophase.

Suppression of circadian rhythms and sleep by LL. LL strongly suppressed 24-h rhythms of T_b, PSD, ACT, and body weight previously evident in LD, as was confirmed visually in raster plots and by 10-day periodograms beginning a few days after the LL transitions (Figs. 1 and 3*B*). T_b stabilized at a relatively constant level within a few days after the LD-LL transition (Fig. 4), and mean T_b and SWA during successive 12-h periods remained constant (Table 1).

NREM and REM were either strongly suppressed (n = 3) or totally absent (n = 2) in LL (Table 1; Fig. 5, *top*). Arousal state patterns throughout LL were similar to those during L (Fig. 3, *A* and *B*). Percent total recording times of all arousal states during successive 12-h periods in LL did not differ from times during L and were statistically indistinguishable from one another (Table 1; Fig. 5, *top*).

Waking was negatively correlated and NREM positively correlated with SWA during the first 12-h period of LL ($r = -0.42$, $P < 0.05$; $r = 0.48$, $P < 0.05$, respectively).

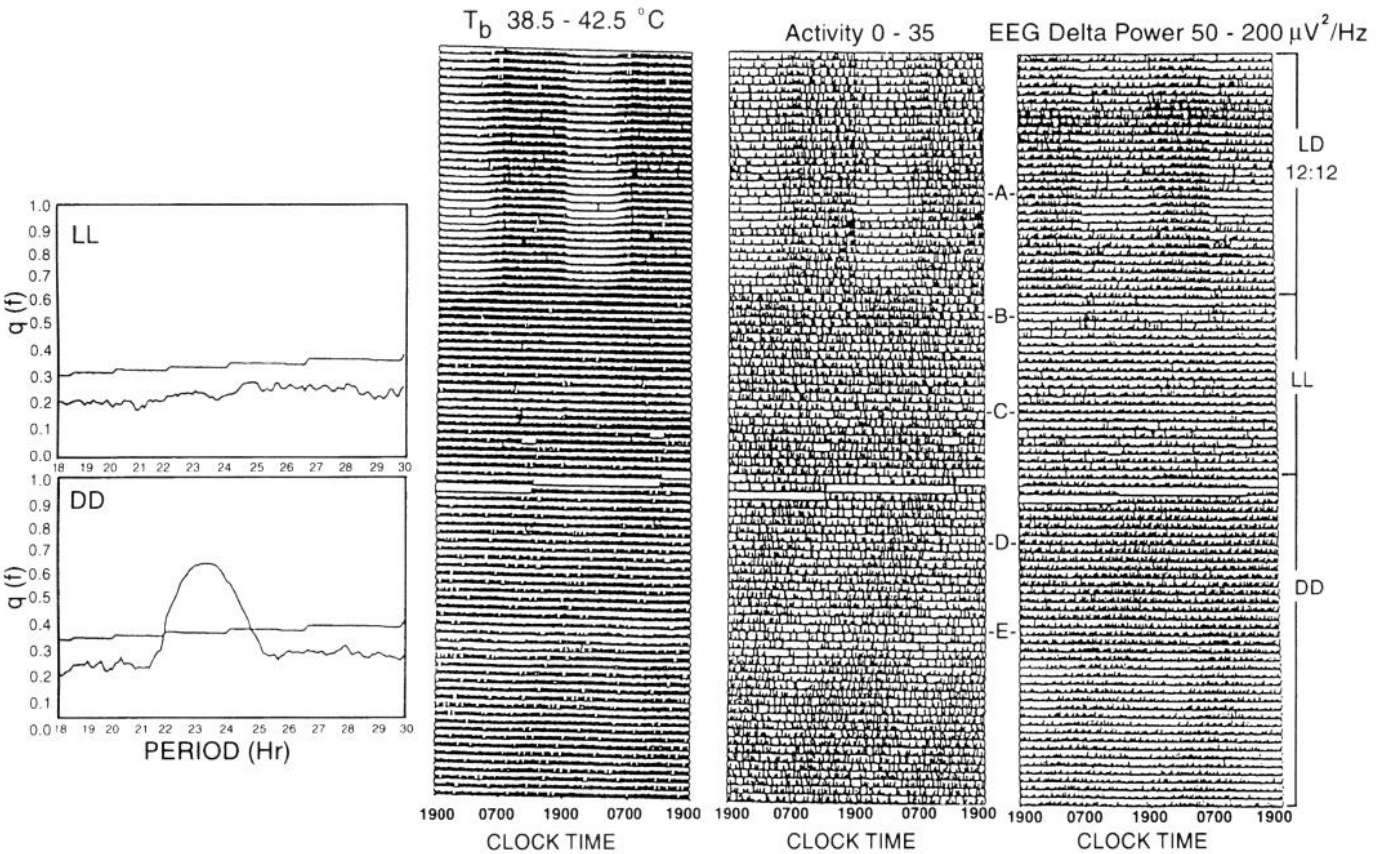

Fig. 1. Raster plots of body temperature (T_b, *left*), activity (*middle*), and EEG delta power (SWA, 0.75–4.0 Hz, *right*) of a pigeon sequentially exposed to a 12:12-h light-dark cycle (LD), constant bright light (LL), and constant dim light (DD) over a 92-day period. A 4-day fast in each photic condition began on days marked A, C, and E. Ten-day data sets for T_b periodograms (*far left*) began on days marked B (LL) and D (DD). Data missing on 3rd day after LL-DD transition due to computer disk failure.

Throughout LL, SWA did not exhibit any consistent changes indicative of increased sleepiness (Fig. 1), nor were there any qualitative changes in tonic or phasic EMG levels. Furthermore, the physical appearance (such as plumage and skin) and behavior of the pigeons remained normal throughout these prolonged periods of LL-induced insomnia.

Arousal states, PSD, and T_b after LL-DD transitions. Arousal state patterns during the first 24 h after transitions from LL to DD (TDD) also showed no evidence of prior sleep deprivation during LL. Latency to the initial onset of sleep (1 min of continuous NREM) after LL-DD transitions was almost six times longer than after L-D transitions in LD ($P < 0.05$; Table 3). Furthermore, the 24-h total sleep time (TST) in TDD did not differ from that in LD or DD, even though TST was higher during the second 12-h period of TDD compared with the 12-h L period in LD ($P < 0.05$) as well as with the second 12-h period of DD several weeks later ($P < 0.01$; Fig. 5, *top*). Similarly, if drowsy is

Table 1. *Body temperature and arousal state variables for each consecutive 12-h and combined 24-h polygraphically recorded period in 12:12-h light-dark cycle, constant bright light, and constant dim red light*

	1st 12 Hours			2nd 12 Hours			24 Hours		
	LD night	LL	DD subj. night	LD day	LL	DD subj. day	LD	LL	DD
T_b	40.13 ± 0.09	40.73 ± 0.19[a]	40.33 ± 0.28	41.16 ± 0.21[f]	40.74 ± 0.18[a]	40.89 ± 0.21[e]	40.64 ± 0.12	40.73 ± 0.18	40.61 ± 0.23
Waking	34.90 ± 6.92	71.59 ± 12.49[bc]	50.11 ± 7.49	71.69 ± 9.50[e]	74.71 ± 10.68	64.81 ± 12.63	53.30 ± 7.26	73.20 ± 11.52[ac]	57.46 ± 9.66
Drowsy	7.05 ± 1.87	25.35 ± 12.04	8.82 ± 2.81	27.89 ± 9.57	23.95 ± 10.24	21.02 ± 9.48	17.47 ± 4.84	24.61 ± 11.10	14.92 ± 5.97
NREM	56.25 ± 8.17	3.05 ± 1.85[bd]	39.95 ± 6.21[a]	0.42 ± 0.28[f]	1.33 ± 0.81	14.06 ± 6.36[e]	28.33 ± 4.19	2.18 ± 1.30[bd]	27.00 ± 5.54
REM	1.79 ± 0.74	0.01 ± 0.01	1.13 ± 0.38	0.00 ± 0.00	0.00 ± 0.00	0.11 ± 0.06	0.90 ± 0.37	0.00 ± 0.00	0.62 ± 0.18
TST	58.04 ± 8.20	3.06 ± 1.85[bd]	41.08 ± 6.26[a]	0.42 ± 0.28[f]	1.33 ± 0.81	14.17 ± 6.35[e]	29.23 ± 4.21	2.18 ± 1.30[bd]	27.62 ± 5.45
TST + Dr	65.10 ± 6.92	28.41 ± 12.49[bc]	49.89 ± 7.49	28.31 ± 9.50[e]	25.29 ± 10.68	35.19 ± 12.63	46.70 ± 7.26	26.80 ± 11.52[ac]	42.54 ± 9.66
SWA	112.83 ± 4.42	93.07 ± 3.05	115.50 ± 15.69	87.17 ± 4.42	95.90 ± 5.85	103.16 ± 13.30	100.00 ± 0.00	94.49 ± 4.45	109.33 ± 14.49

Values are means ± SE; $n = 5$ except EEG slow wave activity, 0.75–4.0 Hz (SWA; $n = 2$, no statistical comparisons). LD, 12:12-h light-dark cycle; LL, constant bright light; DD, constant dim red light; Subj, subjective night or day determined from body temperature (T_b) rhythm (see METHODS); NREM, non-rapid-eye-movement sleep; REM, rapid eye movement sleep; TST, total sleep time (NREM + REM); TST + Dr, TST + Drowsy. [a]$P < 0.05$, [b]$P < 0.01$, different from comparable period in LD; [c]$P < 0.05$, [d]$P < 0.01$, different from comparable period in DD; [e]$P < 0.05$, [f]$P < 0.01$, different from preceding 12-h period in same condition.

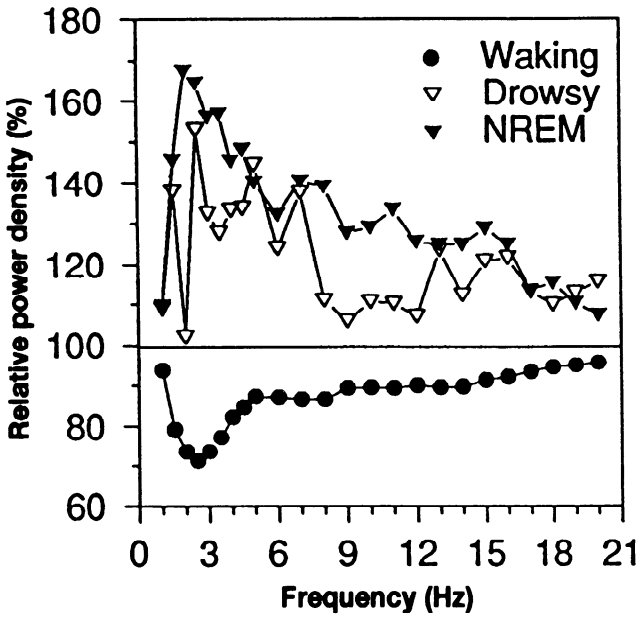

Fig. 2. Stage-specific relative EEG power density of a pigeon in 12:12 LD. Power density plotted in 0.5-Hz bands from 1 to 5 Hz, and 1-Hz bands from 6 to 20 Hz, as percentage of 24-h mean in each band.

included with NREM and REM in calculations of TST, TST + drowsy during the first 12 h of TDD did not differ from D in LD, despite a 59% reduction in 24-h TST + drowsy during LL compared with LD (Fig. 5, *top*; $P < 0.05$). Consistent with the delayed appearance of NREM after the transitions, SWA gradually increased during the first 5 h after the transition (Fig. 3*C*) but did not rise above prior D levels in LD, even though 24-h values were lower during LL than during LD (Table 1; Fig. 5, *bottom*).

Reappearance of circadian rhythms in DD. Free-running circadian rhythms of T_b, SWA, body weight, and ACT reappeared within a few days after LL-DD transitions and retained approximately the same phase relationships as in LD (Figs. 1 and 3*D*). Acrophase phase angles from T_b were 141.8° for body weight and 45.1° for ACT (Table 2). As in LD, there was a close correspondence between sleep and SWA (Fig. 3). The lag between T_b and SWA acrophases was 163.2°.

Although the amplitude of circadian rhythms was reduced in DD compared with LD (Fig. 1; Table 1), T_b was 0.6°C higher during the subjective day than during the subjective night ($P < 0.05$), and NREM was higher during the subjective night than during the subjective day ($P < 0.05$). SWA was 12% higher during the subjective night than during the subjective day. Waking was negatively correlated with SWA during both subjective night ($r = -0.77, P < 0.01$) and day ($r = -0.48, P < 0.05$). NREM was positively correlated with SWA ($r = 0.74$, $P < 0.01$) during the subjective night but not during the subjective day.

DISCUSSION

Entrained rhythms in LD. Arousal state and PSD patterns in LD were similar to those previously reported in the pigeon (25). SWA was lowest during daytime, increased during drowsy, and reached maximal levels during NREM, so that it rose to high levels at night when NREM was maximal and fell during the day when NREM was minimal. Correspondingly, 24-h rhythms of PSD, T_b, ACT, and body weight were also entrained by the LD cycles.

In LD, TST/24 h (29%) was confined almost entirely to the 12-h D period and was lower than in previous studies in the same laboratory [45% (30); 44% (18)] and in others [44% (29); 60% (25)]. However, before the study of Phillips and Berger (18), drowsy either was not scored as a separate state (25, 30) or its amount was not reported (29). Tobler and Borbély (25) reported a much higher frequency of EOG activity during NREM in L (32.6/min) than during D (1.7/min). Therefore, some or all of their daytime NREM (37.7%) would be scored as drowsy according to the sleep stage criteria of the present study. In the case of the study by Walker and Berger (30), much of the daytime NREM (12%) probably would also have been scored as drowsy if the criterion of a maximum EOG rate of $< 1/5$ s had been applied to the scoring of NREM. Because Van Twyver and Allison (29) did not report the day-to-night distribution of arousal states, their results cannot be included in specific comparisons between studies of diurnal and nocturnal arousal states. If drowsy is included in TST in the current study, then the daytime differences between studies disappear (mean of other studies, 27%; current study, 28%). Nevertheless, nighttime differences remain (83% vs. 65%).

Although there were a number of methodological differences among these studies that could have affected sleep amounts (see Ref. 18), a major difference was in the provision of dim red light during the D portion of LD in the present study vs. total darkness in the other studies (except Ref. 29). Suppression of pineal and

Table 2. *Results of cosinor analysis of data from 12:12-h light-dark cycle and constant dim red light*

Condition	Variable	n	Mean	Amplitude	Acrophase, °	Lag from T_b Acrophase, °
LD	T_b, °C	5	40.64 ± 0.12	0.76 ± 0.14	209.4 ± 8.24	0.0 ± 0.00
	Body wt, g	3	435.88 ± 25.64	6.19 ± 1.99	350.9 ± 7.50	138.4 ± 14.95
	Activity, events	3	11.60 ± 2.39	9.61 ± 1.92	207.3 ± 8.41	354.7 ± 13.22
	SWA, %	2	84.36 ± 33.08	13.17 ± 9.43	24.6 ± 3.42	174.6 ± 15.61
DD	T_b, °C	5	40.61 ± 0.23	0.43 ± 0.17	NA	0.0 ± 0.00
	Body wt, g	3	477.84 ± 47.12	4.98 ± 0.95	NA	141.8 ± 26.70
	Activity, events	3	8.54 ± 1.90	2.79 ± 1.17	NA	45.1 ± 23.38
	SWA, %	2	86.80 ± 23.93	6.70 ± 2.81	NA	163.2 ± 6.00

Values are means ± SE; n, no. of pigeons. NA, nonapplicable owing to absence of zeitgeber in DD. (Lags from T_b acrophase for each variable are not equal to algebraic differences in acrophase because of different nos. of pigeons.)

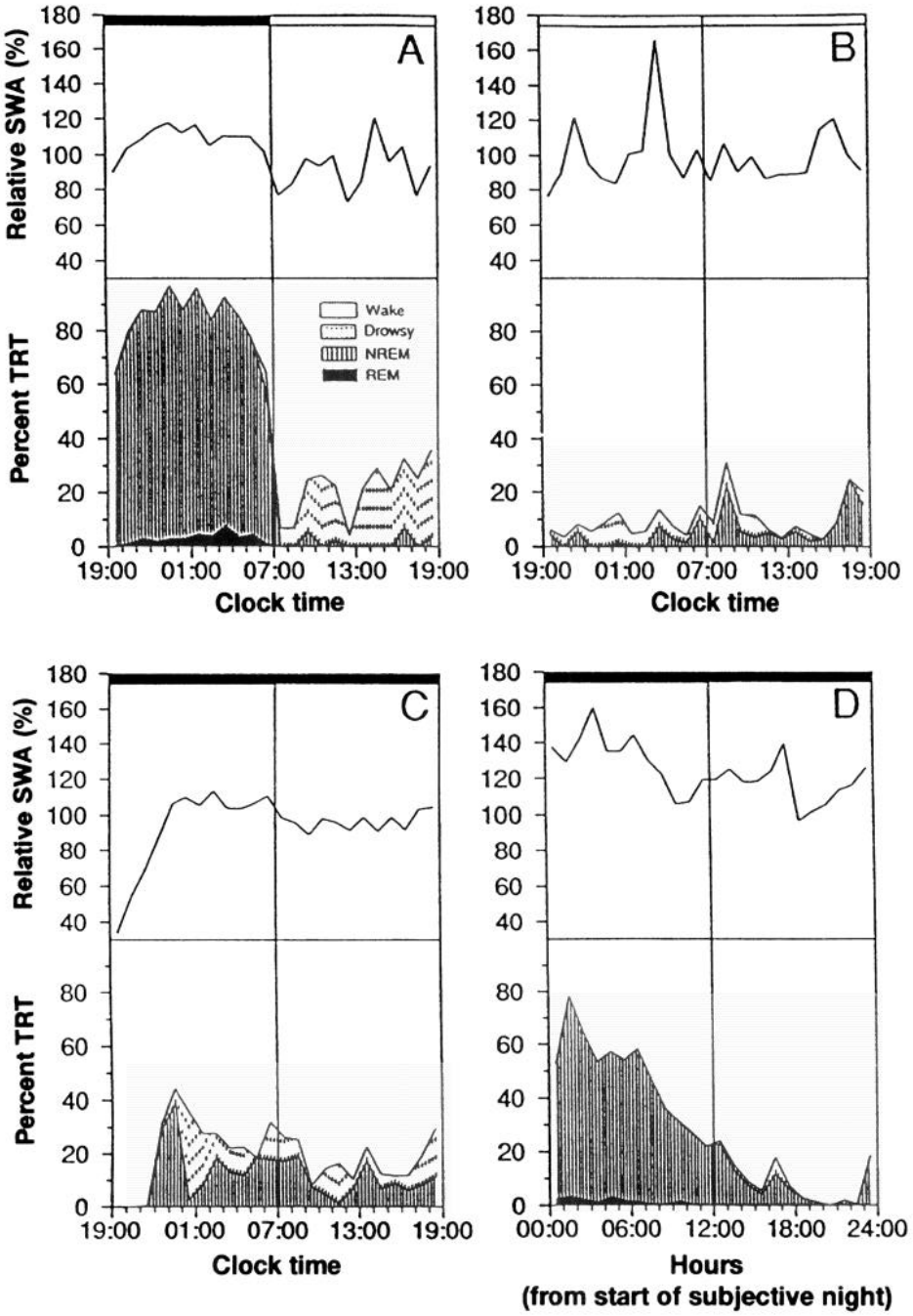

Fig. 3. Sixty-minute mean relative EEG percent slow wave activity (SWA, 0.75–4.0 Hz as percentage of 24-h LD mean; *top*) and percent total recording time (TRT) of waking, drowsy, non-rapid eye movement (NREM), and rapid eye movement (REM) (*bottom*) of a pigeon in LD (*A*), LL (*B*), the first 24 h in DD after LL (*C*), and several weeks later in DD (*D*).

serum melatonin in rats by red light intensities as low as 2 lx (14, 24) was reported after the completion of the current study. Even though maximal inhibition of pineal melatonin secretion is induced by blue-green wavelengths (5) and is generally greater in nocturnal than in diurnal animals (22), it is likely that dim red light partially suppresses melatonin secretion and sleep in pigeons. NREM during dim red light (D) in the current study was 56%, compared with 72% during total darkness in an earlier study conducted in the same laboratory and in which the same scoring criteria were applied (18). On the other hand, Van Twyver and Allison (29) reported total 24-h NREM (not including drowsiness) as 44% in pigeons which were exposed at night to dim red light of unspecified intensity (29), a value similar to the 36% of the study by Phillips and Berger (18) conducted in nocturnal darkness. However, because there probably were greater differences in methods and procedures, including scoring criteria, between studies conducted by different investigators in different laboratories than by the same investigators in a single laboratory, greatest weight should be placed on the reduction in nocturnal NREM during red light in the current study compared with total darkness in the earlier study in the same laboratory (18).

The percent REM/TST of 3% was also much lower than the amounts ranging from 7 to 18% reported in the previous studies. Because REM usually occurs only

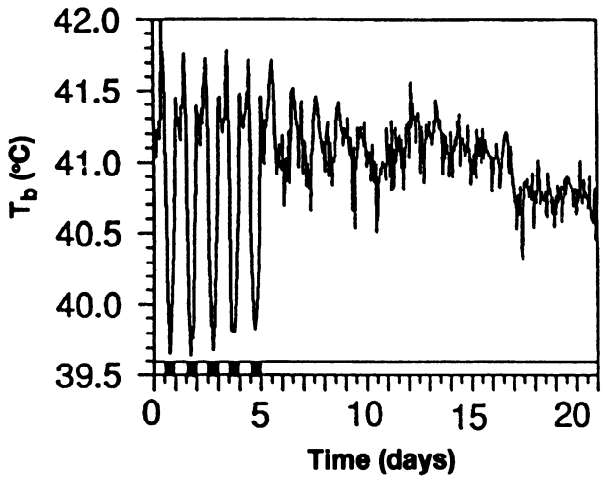

Fig. 4. Disappearance of 24-h T_b rhythm in a pigeon after transition from LD to LL. Solid bars, dark periods.

during extended episodes of NREM, suppression of NREM by dim light would also result in a suppression of REM.

Suppression of sleep and circadian rhythms by LL. LL sharply suppressed sleep and eliminated circadian rhythms of PSD, T_b, and behavioral measures as in earlier studies (11, 19, 31). The suppression of circadian rhythms of melatonin by LL (31) can be assumed to mediate the suppression of the other circadian rhythms, inasmuch as sleep is restored in LL by daily infusions of melatonin (19), as are circadian rhythms of activity in pinealectomized pigeons by daily injections (17) or physiological infusions (8) of melatonin.

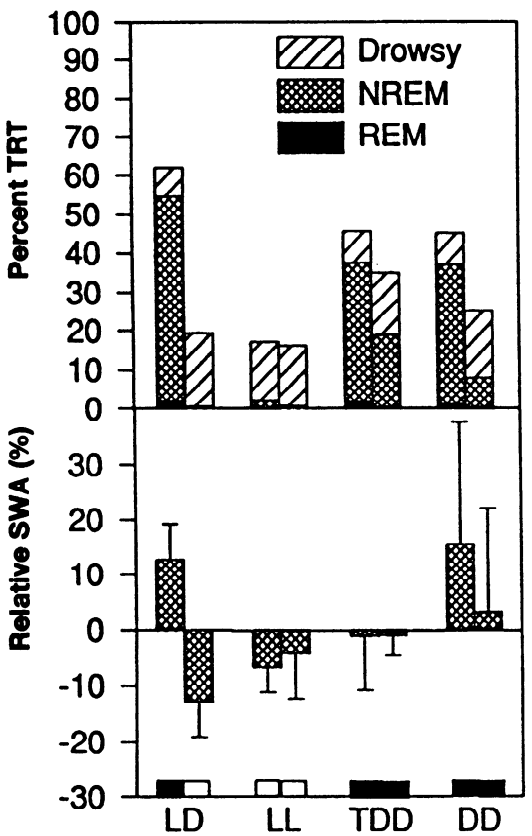

Fig. 5. Percent TRT of drowsy, NREM, and REM (*top*; n = 4) and EEG SWA (*bottom*; n = 2) during LD, LL, the 1st 24 h after transition from LL to DD (TDD), and after adaptation to DD. Each pair of bars represents consecutive 12-h periods. Relative SWA is percent change (means ± SD) from 24-h LD mean power density in 0.75- to 4-Hz band. Bars at very bottom: solid, dark periods; open, light periods.

Table 3. *Sleep onset latency after L to D in L-D and LL-DD transitions*

Pigeon No.	L-D	LL-DD
22	4.6	38.3
24	24.6	40.4
25	35.0	303.3
26	24.6	57.1
31	0.8	193.3
32	25.4	43.8
Mean ± SD	19.2 ± 13.4	112.7 ± 110.8*

Values are in minutes. *$P < 0.05$ vs. L-D, Wilcoxon signed-rank test.

The effect of LL on arousal state patterns was to eliminate previous changes induced by darkness in LD, thus prolonging the patterns of arousal during the preceding L portion of the LD condition throughout LL. Despite the prolonged suppression of sleep by LL for up to 74 days, there were no progressive signs of increased sleepiness in SWA. The physical appearance and behavior of the pigeons also remained normal throughout prolonged LL-induced insomnia, in contrast to the debilitated appearance before death of rats instrumentally sleep deprived for a mean duration of only 21 days (12, 21). Unlike the initial rise and subsequent fall in T_b of sleep-deprived rats (1), T_b stabilized at a relatively constant level within a few days after the initial suppression of its circadian rhythm by LL. Finally, body weight and activity also stabilized within a few days after the LD-LL transition, in contrast to increased activity (1), increased feeding, and decreased body weight (12) produced by total sleep deprivation in rats. The insomnia of LL-exposed pigeons is due to the suppression of melatonin, which acts as a potent sleep-inducing hormone in pigeons (19). Therefore, it is not equivalent to instrumental sleep deprivation of mammals, during which incipient sleep episodes are constantly interrupted and sleep factors presumably accumulate.

Drowsiness is an arousal state intermediate between waking and NREM that may partially fulfill the function(s) of NREM. If LL simply caused an increase in the frequency of EOG activity, NREM in LD would have been replaced by drowsy in LL. However, there were no significant differences in drowsy between LD and LL (although it did increase slightly during LL to levels similar to those during L of LD) despite significant decreases in NREM. Furthermore, even if drowsy is included with NREM and REM in TST/24 h, the latter was still significantly suppressed by LL compared with LD (Table 1).

Absence of compensatory recovery of sleep after LL-DD transitions. Arousal states after LL-DD transitions did not exhibit a pattern of increased compensatory recovery of sleep that is often seen after instrumental sleep deprivation. Sleep latency after the LL-DD transition

was significantly longer than that after the prior L-D transition in LD. Moreover, total amounts of drowsy, NREM, REM, and SWA during the first 12-h period in DD did not differ from those during D of LD (Fig. 5).

These results confirm those of Tobler and Borbély (25) after 24-h deprivation of sleep and drowsiness in pigeons by continuous light, human presence, and acoustic and tactile stimuli. Their birds also displayed increased initial wakefulness, without increased NREM or increased NREM SWA during the 12-h recovery period in D, compared with 12-h baseline D values in LD. On the other hand, wakefulness decreased and REM increased during D after sleep deprivation. The decrease in waking can be attributed to the increased REM, because no changes in NREM were reported. The low and variable amounts of REM in D and DD did not affect corresponding amounts of wakefulness in the present study.

The reappearance of free-running circadian rhythms of sleep, T_b, and behavioral variables within a few days after LL-DD transitions, retaining similar phase relationships to sleep as in LD, demonstrated that LL exposure did not cause any permanent effects on the circadian system.

Perspectives

The remarkable absence of any buildup of sleep pressure during LL, or of subsequent compensatory recovery of sleep, regardless of the duration of LL-induced insomnia, is in contrast to increases in SWA and/or amounts of NREM in mammals (e.g., Refs. 4, 15, and 27) after they were instrumentally deprived of sleep. However, compensatory recovery of sleep or SWA in the hamster occurred after sleep deprivation during the first 3 h of the 12-h light period but not after 24-h sleep deprivation (26). Therefore it is possible that the time and duration of instrumental sleep deprivation relative to the phase of the circadian system may modulate subsequent arousal state patterns. As noted in METHODS, birds display more drowsiness and SWA during their major active period than mammals. However, drowsy and SWA remained at about the same levels throughout LL as during the prior L of LD (Fig. 1, Table 1). Therefore the pigeons did not compensate for the suppression of NREM SWA by subsequent increased SWA during waking or drowsy. Pigeons, unlike chickens (28, 32) and most mammals, do not exhibit a decreasing trend in SWA during their major sleep period (see Fig. 1 and Ref. 25). However, the cat, which lacks a strong diel rhythm of sleep, also does not manifest a trend in SWA during either the day or night but does display increased SWA after sleep deprivation (15, 27). Therefore the presence or absence of decreasing trends of SWA during sleep does not signify a corresponding presence or absence of compensatory recovery of sleep after sleep deprivation.

In birds, the pineal gland can act as an autonomous circadian clock, whereas in mammals the SCN is the master circadian oscillator (7). The suppression of melatonin synthesis and sleep by LL and the restoration of sleep by melatonin infusions in pigeons indicate that melatonin is a potent avian sleep factor (19). Prolonged suppression of melatonin secretion and sleep by LL in pigeons is functionally different from instrumental sleep deprivation, during which the accumulation of sleep factors could be responsible for increasing sleepiness and subsequent compensatory recovery of sleep. In contrast, the increased sleep latency and absence of homeostatic compensatory recovery of sleep after LL-DD transitions in pigeons probably reflects a delay in the synthesis and secretion of melatonin after prolonged LL exposure. Therefore, current evidence indicates that it is more apt to describe the LL-induced suppression of melatonin and sleep as a condition of chronic insomnia than of sleep deprivation.

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