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Complete suprachiasmatic lesions eliminate circadian rhythmicity of body temperature and locomotor activity in golden hamsters

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Abstract The effects of suprachiasmatic and control lesions on the circadian rhythms of locomotor activity and body temperature were studied in golden hamsters (*Mesocricetus auratus*) maintained in constant light as well as constant darkness. Large suprachiasmatic lesions, but not control lesions, eliminated circadian rhythmicity in locomotor activity as well as in body temperature. Analysis of the "robustness" of the rhythms of locomotor activity and body temperature in unlesioned and lesioned animals suggests that, because body temperature rhythmicity is more robust than locomotor rhythmicity, lesions that spare a small number of suprachiasmatic cells might abolish the latter but not the former. Our results do not support the hypothesis that the body temperature rhythm is controlled by a circadian pacemaker distinct from the main pacemaker located in the suprachiasmatic nuclei.

Key words Suprachiasmatic nucleus · Circadian rhythms Body temperature · Locomotor activity · Syrian hamster

Introduction

The existence of a daily oscillation in the physiology and behavior of mammals has been known for over a century (Davy 1845; Maurel 1884). Daily rhythmicity is the result of the combined action of an endogenous circadian pacemaker and environmental time cues (Aschoff 1960; Pittendrigh 1981).

In mammals, a circadian pacemaker responsible for the generation of several overt rhythms is located in the suprachiasmatic nuclei of the hypothalamus (Ralph et al. 1990; Sato and Kawamura 1984; Stephan and Zucker 1972; Stetson and Watson-Whitmyre 1976). Unsuccessful attempts to eliminate the circadian rhythm of body temperature (CRT) by ablation of the suprachiasmatic nuclei (SCN) have led several authors to suggest that the CRT may be controlled by a separate, extra-SCN pacemaker (Dunn et al. 1977; Fuller et al. 1981; Powell et al. 1980; Satinoff and Prosser 1988). The reports of persistence of the CRT after SCN lesions are in contrast with several studies in which large SCN lesions were found to eliminate the CRT as well as the circadian rhythms of other variables (Eastman et al. 1984; Honma et al. 1988; Ruby et al. 1989; Ruis et al. 1987; Stephan and Nunez 1977). The contradiction cannot be explained by species differences, as SCN lesions in the laboratory rat have been reported to either eliminate (Eastman et al. 1984; Ruis et al. 1987; Stephan and Nunez 1977) or spare (Dunn et al. 1977; Powell et al. 1980; Satinoff and Prosser 1988) the temperature rhythm. Regarding the exact location and extent of the lesions, it is conceivable that incomplete lesions might leave SCN tissue sufficient to generate the CRT but not other circadian rhythms, thus explaining the observations of spared CRT in lesioned animals. However, even if re-analysis of the published data could demonstrate that all cases of spared CRT were due to incomplete SCN lesions, there is no apparent reason why the CRT would require less SCN tissue than other rhythms.

Although advances in neuroanatomical research may eventually break the stalemate in the literature, we felt that approaching the issue from a new perspective might facilitate the resolution of the problem. Therefore, we decided to investigate the CRT in SCN-lesioned animals using a different animal species and a new analytical approach.

As the animal species, we chose the golden hamster (*Mesocricetus auratus*). Although the golden hamster is one of the most commonly used rodent species in studies of basic mechanisms of circadian organization, it has not been used in any of the previous studies of the effects of SCN lesions on the body temperature rhythm. These latter studies have utilized mostly – although not exclusively – laboratory rats.

The new approach we adopted consists of examining the "robustness" of the rhythms under study rather than

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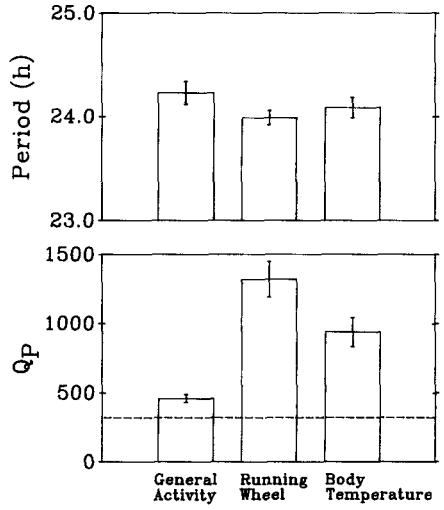


Fig. 1 Free-running period and rhythmicity index of golden hamsters maintained in constant light, as determined in three different variables. Each *bar* corresponds to the mean (±SE) of 12 animals. The *dashed line* in the lower panel indicates the 0.05 level of significance

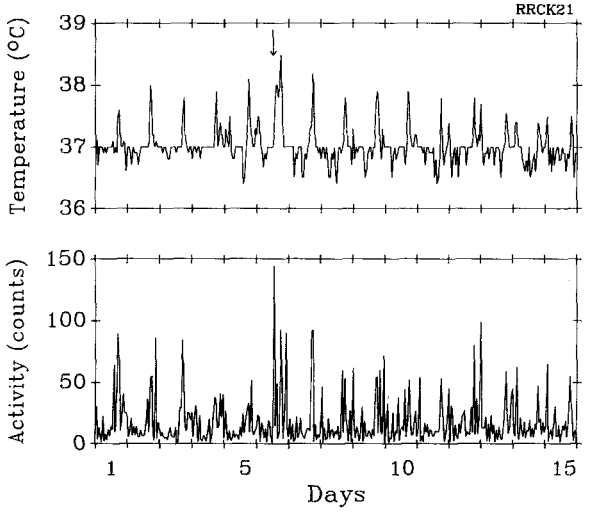


Fig. 2 Body temperature and locomotor activity records of a golden hamster that received an extra-SCN brain lesion at the time indicated by the *arrow*. The data were collected in 6-min bins and averaged hourly

focusing merely on a dichotomous distinction between rhythmicity and arrhythmicity. Students of circadian rhythms have long known that expressed rhythms are much more "robust" in some species than others. For instance, the circadian rhythm of locomotor activity (CRA) is very "noisy" in guinea pigs (Kurumyia and Kawamura 1988) but quite robust in golden hamsters (Refinetti et al. 1992). Less attention has been given to the fact that different circadian rhythms in the same species have different degrees of robustness. If the CRT were more robust than the CRA, then one might expect that incomplete SCN lesions would have different effects on the two rhythms, even though only a single pacemaker were affected. The noise introduced by a partial lesion would have an equivalent effect on the generation of both the CRT and the CRA, but the expression of the latter would be more seriously disrupted than that of the former. The present experiment tested this hypothesis by comparing the robustness of the CRT and CRA in golden hamsters with and without SCN lesions.

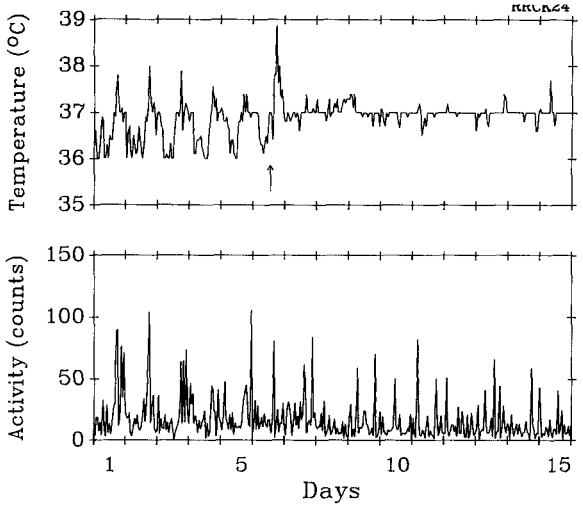


Fig. 3 Body temperature and locomotor activity records of a golden hamster that received an SCN lesion at the time indicated by the *arrow*. The data were collected in 6-min bins and averaged hourly. The temperature rhythm became almost flat after the lesion. Although enhanced by the hourly averaging, the frequent straight segments (at times longer than 12 h) are not artifactual and reflect true circadian arrhythmicity

Materials and methods

Animals

Male golden hamsters (*Mesocricetus auratus*) were purchased from Charles River Laboratories (Wilmington, MA) and housed under a 14:10 h light-dark cycle until the beginning of the experiment.

Experiment 1: Robustness of different circadian rhythms

In this first experiment, we compared the robustness of the rhythms of running wheel activity, general locomotor activity, and body temperature. Because the amplitude of the CRT is significantly larger in hamsters with access to running wheels (Refinetti and Menaker 1992), the robustness of the rhythm of running-wheel activity was investigated in one group of 12 hamsters whereas the robustness of the rhythms of general activity and body temperature were studied by telemetry in another group of 12 animals. In all cases, robustness was numerically defined as the value of the Sokolove-Bushell's Q_P statistic (Sokolove and Bushell 1978), as described below.

Running-wheel activity. At the age of 8–12 weeks, the animals were transferred to individual plastic cages (25×46×20 cm) lined with wood shavings and fitted with 17 cm diameter running wheels. Purina Lab Chow and water were available ad libitum. The cages were maintained in ventilated, light-tight boxes at 22°C. Microswitches attached to the wheels allowed recording of running-wheel activity in 6-min bins by a computerized data acquisition

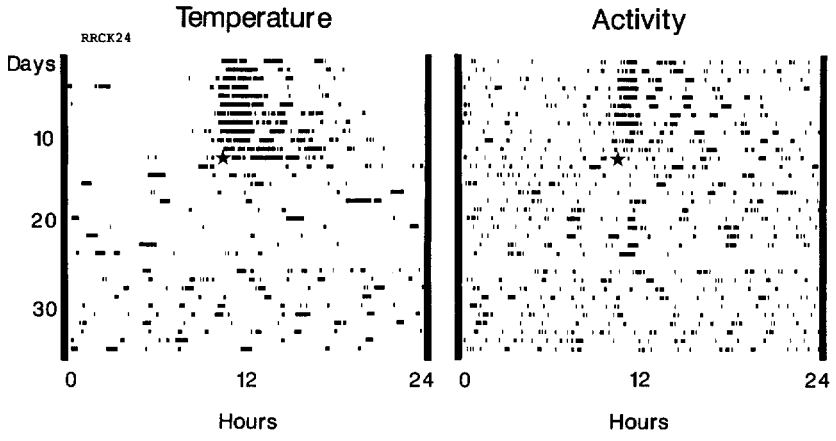


Fig. 4 Actograms of body temperature and locomotor activity of a golden hamster that received an SCN lesion at the time indicated by the *star*. Actograms were prepared by plotting a dark mark for each 6-min bin in which body temperature exceeded the daily mean by more than 2% and locomotor activity exceeded the daily mean by more than 80%. Ambient temperature was 22°C from day 1 to day 23 and 12°C from day 24 to day 34. The data from days 7 through 21 are shown in Cartesian format in Fig. 3

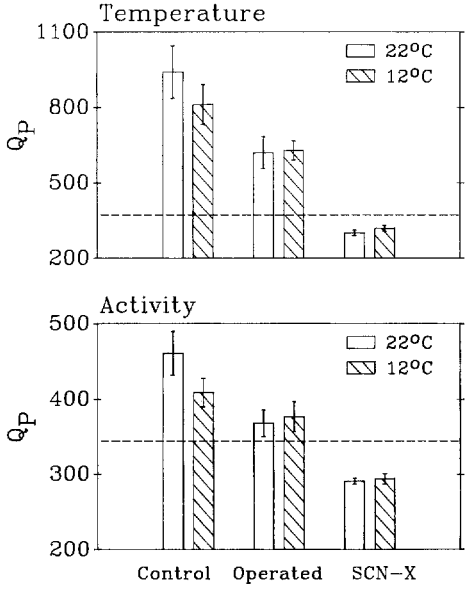


Fig. 5 Rhythmicity index (Q_P) for body temperature and locomotor activity in unoperated control (*Control*), operated control (*Operated*), and SCN-lesioned (*SCN-X*) hamsters maintained at two different ambient temperatures. Each bar corresponds to the mean (±SE) of 12 animals. The *dashed lines* indicate the 0.05 level of significance

Table 1 Parameters of the body temperature rhythm[a]

	Control	Operated	SCN-X
Period (h)			
22°C	24.09 (0.10)	23.91 (0.14)	#
12°C	24.29 (0.07)	24.24 (0.10)	#
Amplitude (°C)			
22°C	2.09 (0.07)	2.12 (0.16)	1.70 (0.06)
12°C	2.34 (0.11)	2.53 (0.09)	2.09 (0.11)
Mean level (°C)			
22°C	36.53 (0.11)	36.74 (0.12)	36.83 (0.09)
12°C	36.33 (0.08)	36.31 (0.14)	36.48 (0.11)

[a] All values are means (±SE) of 12 animals
The mean periods for SCN-X animals have no physiological meaning, as none of the Q_P values reached statistical significance. The calculated periods oscillated randomly within the investigated range of 23.0 to 25.0 h

system (Dataquest III, Data Sciences, St. Paul, MN). The animals were left undisturbed for three or more weeks under constant light (LL, 150 lux).

Telemetry. At the age of 8–12 weeks, the animals were transferred to individual plastic cages (21×30×20 cm) lined with wood shavings and were fed Purina Lab Chow and water ad libitum. Radio transmitters for the monitoring of body temperature and locomotor activity (Model VM-FH, Mini-Mitter Co., Sunriver, OR) were implanted intraperitoneally under sodium pentobarbital anesthesia (80 mg/kg i.p.). The animal cages were placed on top of radio receivers (Model RA-1010, Mini-Mitter Co., Sunriver, OR) located in a large temperature-controlled room. The radio receivers were attached to the computerized data acquisition system, so that body temperature and activity could be recorded in 6-min bins for three or more weeks. Ambient temperature (22°C) and illumination (LL, 150 lux) were maintained constant throughout the experiment.

Experiment 2: Brain lesions in animals maintained in constant light (LL)

In this experiment, we investigated the effects of suprachiasmatic lesions on the rhythms of body temperature and locomotor activity. Because the amplitude of the CRT is significantly larger in hamsters with access to running wheels, we felt that the effects of SCN lesions on the CRT might be artificially exaggerated by the reduction in activity that follows the lesions. To avoid this complication, the animals in this experiment did not have access to running wheels, and their general locomotor activity was measured by telemetry as described above. Twelve animals received suprachiasmatic lesions and 12 received control lesions. The 12 animals from experiment 1 served as a third group of unoperated controls. In order to assess possible thermoregulatory effects of the brain lesions, ambient temperature was maintained at 22°C for a 7–12 day baseline period, again at 22°C for 12–14 experimental days, and at 12°C for 12–14 days. Four animals were maintained at 22°C or 2°C for 10–20 additional days. Constant illumination (LL, 150 lux) was maintained throughout the experiment, and all animals had free access to food and water.

Electrolytic (anodal) brain lesions were performed under sodium pentobarbital anesthesia (80 mg/kg i.p.) at the end of the 7–12 day baseline period. Control lesions (2–3 mA, 10 s) were placed bilaterally in proximity to the preoptic/anterior hypothalamic area (AP +1.0, V −7.8, L +0.7 and −0.7 from bregma; tooth bar −2.0 mm). Suprachiasmatic lesions (4 mA, 15 s) were placed along the midline between the base of the third ventricle and the optic chiasm (AP +0.6, V −8.3, L 0.0). The electrode was made of platinum/iridium (90/10) and insulated except for 0.3 mm at the tip.

At the end of the experiment, the animals were deeply anesthetized with halothane and intracardially perfused with 4% formaldehyde in 0.1 *M* sodium phosphate buffer containing 15% picric acid (pH 7.4). Serial brain sections 50 µm thick were cut with a

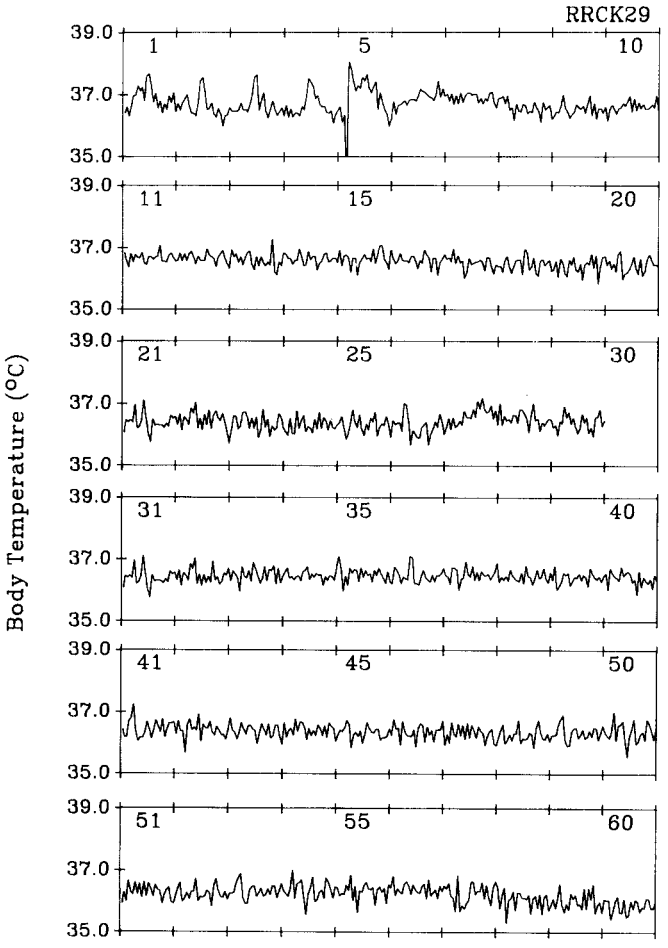


Fig. 6 Body temperature records of a golden hamster maintained in constant light for 60 days. The SCN was lesioned electrolytically on day 5. The reduction in the mean level of body temperature during days 51–60 was due to the lowering of ambient temperature from 22°C to 2°C

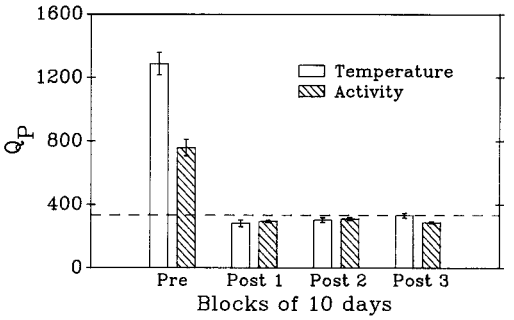


Fig. 8 Rhythmicity index (Q_P) for body temperature and locomotor activity in hamsters maintained in constant darkness. Each *bar* corresponds to the mean (±SE) of 11 animals. The *dashed line* indicates the 0.05 level of significance. *Pre*: 10 days before SCN lesion; *Post 1*, *Post 2*, and *Post 3*: consecutive blocks of 10 days post lesion

Fig. 7A,B Photomicrographs of two brains sectioned coronally through the region that normally contains the SCN and Nissl stained. The brain on the left (**A**) sustained a lesion of the anterior hypothalamic area (not shown) that did not extend to the SCN, whereas the one on the right (**B**) sustained a complete SCN lesion. *Arrows* point to the base of the SCN. Abbreviations: *3V* third ventricle; *OC* optic chiasm. Scale bar: 100 µm. Activity and body temperature data of the animal whose brain is shown in **A** are shown in Fig. 2. Data for the animal whose brain is shown in **B** are shown in Fig. 3

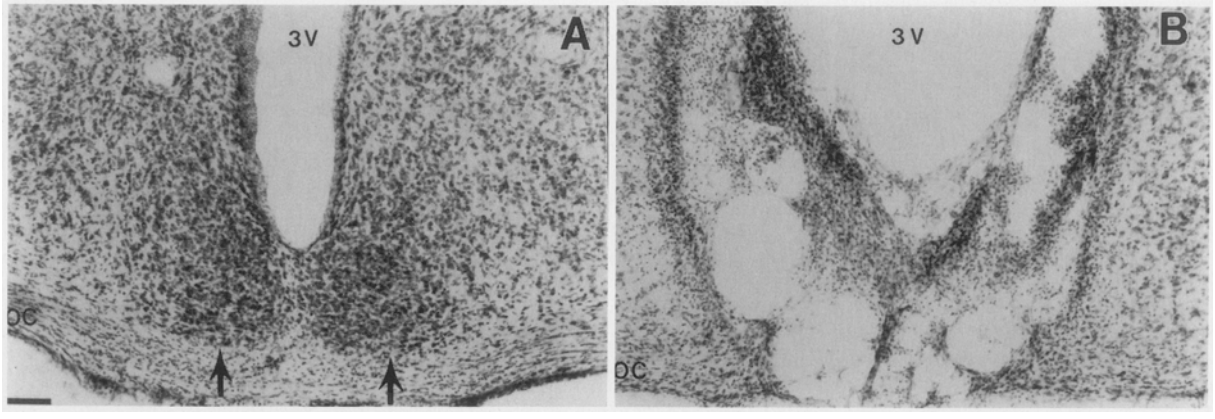
cryostat, mounted onto gelatin coated slides, dried overnight at room temperature, and Nissl stained.

Experiment 3: Brain lesions in animals maintained in constant darkness (DD)

Because the activity rhythm of golden hamsters maintained in constant darkness for several weeks tends to become less robust even without brain lesions (Refinetti et al. 1992), the animals in experiment 2 were maintained in constant light. However, constant illumination for several weeks may supress activity levels and has been reported to cause arrhythmicity in rats (Eastman and Rechtschaffen 1983; Summer et al. 1984) and splitting of circadian rhythms into two components in golden hamsters (Pickard et al. 1984; Swann and Turek 1985). Consequently, we replicated experiment 2 under conditions of constant darkness. The same equipment and procedures were employed, except that only one group of 11 animals was used. Body temperature and locomotor activity were recorded for two weeks before and four weeks after electrolytic lesions of the SCN. Ambient temperature was 22°C throughout. Histological procedures were similar to those of experiment 2 except that brain slices were stained with antibodies to vasoactive intestinal peptide (VIP, RIN 7161, Peninsula Laboratories, Belmont, CA) and glial fibrillary acidic protein (GFAP, G-A-5MAb, ICN Bio-Medicals, Costa Mesa, CA) instead of thionin. Sites of antibody:antigen binding were tagged by an aviden-biotin peroxidase procedure (Elite ABC Kit, Vector Laboratories, Burlington, CA).

Data analysis

For each animal, the mean level of body temperature was calculated as the arithmetic mean of all temperature readings during



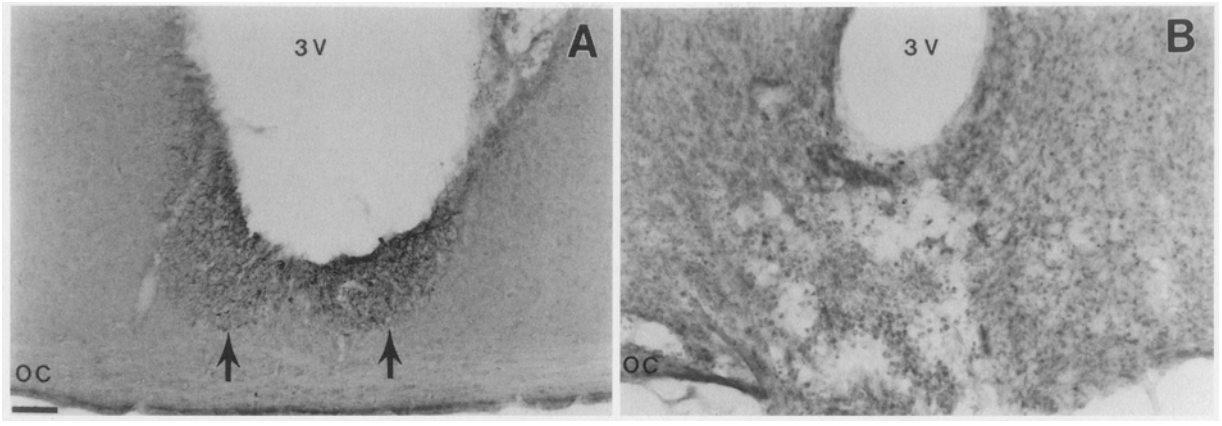


Fig. 9A,B Photomicrographs of two brains sectioned coronally through the region that normally contains the SCN and stained with VIP. The brain on the left (**A**) sustained an incomplete lesion that spared approximately 30% of the SCN, whereas the one on the right (**B**) sustained a complete SCN lesion. *Arrows* point to the base of the SCN. Abbreviations: *3V* third ventricle; *OC* optic chiasm. Scale bar: 100 μm. Activity and body temperature data for these animals are shown in Figs. 10 and 11

blocks of 10 days ($N = 2400$ readings). To calculate the amplitude of the temperature rhythm, we first tabulated the number of values recorded for each temperature between 35.0 and 39.0°C in intervals of 0.1°C. The lowest temperature value that was recorded at least 10 times during the 10 days was considered the lower limit of the oscillation, and the highest value was considered the upper limit. The amplitude of the rhythm was calculated as the difference between the upper and lower limits. It should be noted that this measure of amplitude is independent of the presence or absence of a circadian oscillation. To determine the presence of circadian oscillation, as well as to calculate the period of the oscillation, we used the chi-square periodogram procedure (Refinetti 1992; Sokolove and Bushell 1978).

Because it has been claimed that the chi-square periodogram procedure may not be suited for the determination of periodicity in data with low signal-to-noise ratios (Dowse and Ringo 1991; Kittrell 1991), we first evaluated the effects of increasing noise on the value of the Q_P statistic. The results, which are reported elsewhere (Refinetti 1993), indicated that Q_P is linearly related to the amount of noise present in the data and that the periodogram procedure is capable of detecting statistically significant periodicity in data sets containing up to about 95% noise. In the combined analysis of accuracy and tolerance to noise, the chi square periodogram was superior to autocorrelation, Fourier analysis, and visual observation of actograms (Refinetti 1993).

We used the value of Q_P as an index of rhythmicity in the body temperature and activity data. For each animal, the free-running period (in LL or DD) was calculated as the period yielding the largest Q_P in the interval from 230 to 250 bins (i.e., from 23.0 to 25.0 h, with 0.1 h resolution), and the value of this Q_P was used as the index of rhythmicity. Decisions about the significance ($P < 0.05$) of Q_P values were made in reference to the chi-square distribution (Refinetti 1992). To ascertain that unusual periodicities outside the 23–25 h range were not present in the data, periodograms were also calculated in the range from 14 to 34 h with 1 h resolution. No significant periodicities were found outside the 23–25 h range at any time.

Results

Experiment 1: Robustness of different circadian rhythms

Figure 1 shows the free-running period and the corresponding index of rhythmicity of hamsters maintained in constant light at 22°C for 10 or more days. Ten days of data were used for all calculations. The mean free-running period for all animals was 24.1 h and was independent of the variable used for the computations. Because general activity and body temperature were recorded from the same animals, whereas running-wheel activity was recorded from a different group of animals, we evaluated the statistical significance of differences between the means with *t*-tests for matched or unmatched samples, as appropriate, with the necessary corrections for differences in variance. Even without correction of the level of significance to account for the multiple tests, none of the *t* values was associated with a probability smaller than 0.10.

Significant rhythmicity was found in all three variables, as the critical Q_P at the 0.05 level is approximately 330. However, the index of rhythmicity was significantly lower for general activity than for the other two variables. Even with correction for multiple *t*-tests, the probabilities associated with the *t* values when comparing the mean Q_P for general activity with the Q_Ps for running-wheel activity or body temperature were always lower than 0.005. This indicates that the rhythm of general activity is significantly less robust than the rhythm of body temperature. Running-wheel activity yielded a significantly higher Q_P than body temperature before ($P = 0.03$) but not after ($P = 0.09$) correction for multiple *t*-tests. Also, it is important to reiterate that in this study body temperature was measured in animals without access to running wheels. In hamsters with free access to wheels, the robustness of the body temperature rhythm may even exceed that of running-wheel activity (Refinetti and Menaker 1992). In any event, general activity, rather than running-wheel activity, was the variable monitored in all previous studies that investigated the relationship between activity and temperature rhythms in SCN-lesioned animals.

Experiment 2: Brain lesions in animals maintained in constant light

None of the animals, in any of the three groups, showed signs of splitting. Records of body temperature and locomotor activity of a hamster that received an extra-SCN

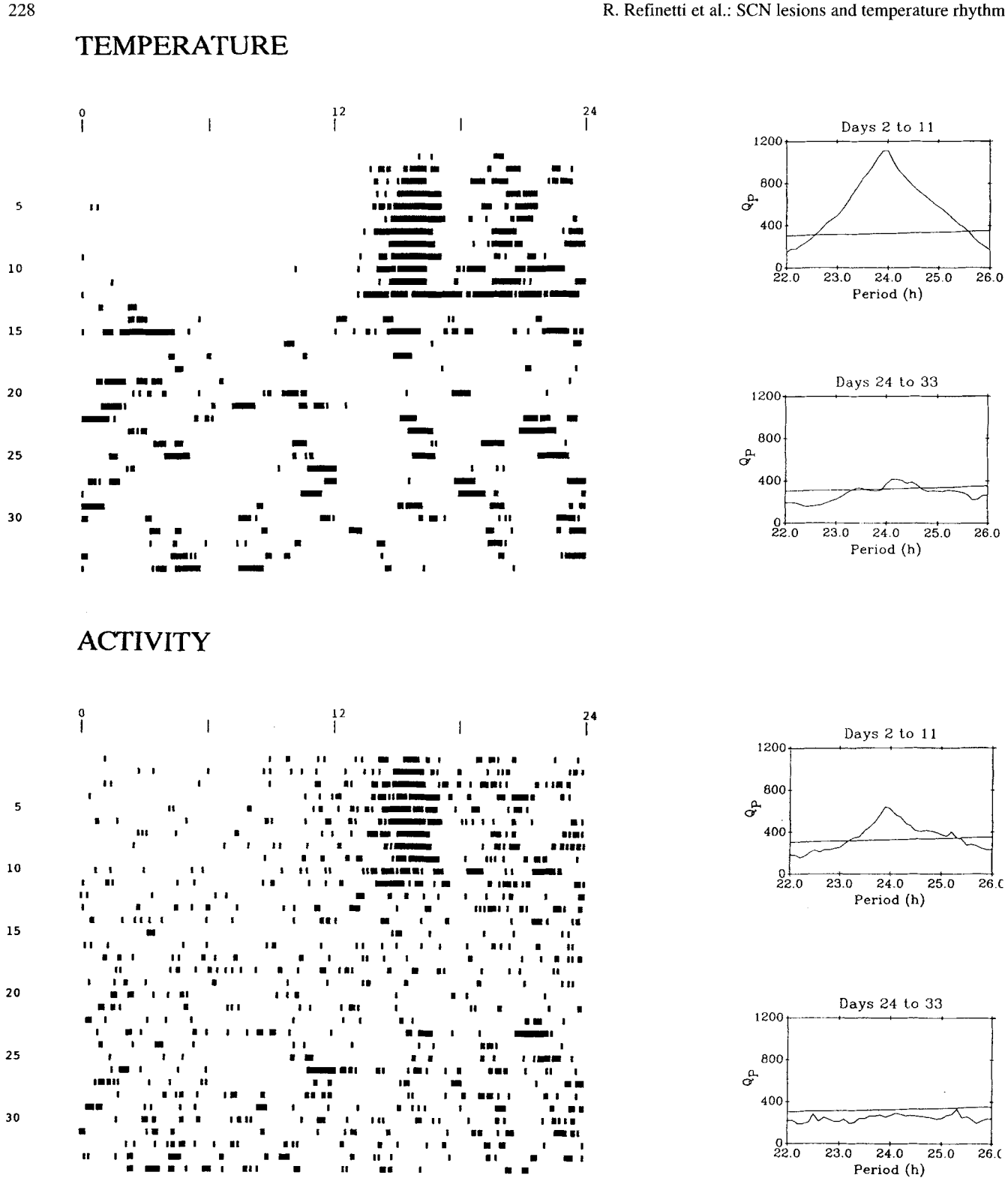


Fig. 10 Actogram and periodograms of the body temperature and activity rhythms of an animal that received an incomplete lesion that spared approximately 30% of the SCN. The *straight line* in the periodograms indicates the 0.05 level of significance with correction for multiple testing. A photomicrograph of the brain of this animal is shown in Fig. 9A

brain lesion are shown in Fig. 2. To facilitate visualization of circadian oscillations, short ultradian oscillations were removed by use of hourly means (means of ten 6-min bins). Clear circadian rhythmicity can be seen both before and after the lesion. The temperature rhythm was practically unaffected by the lesion. The activity rhythm was less clear after the lesion but was still quite robust. In contrast, rhythmicity was drastically reduced or elimi-

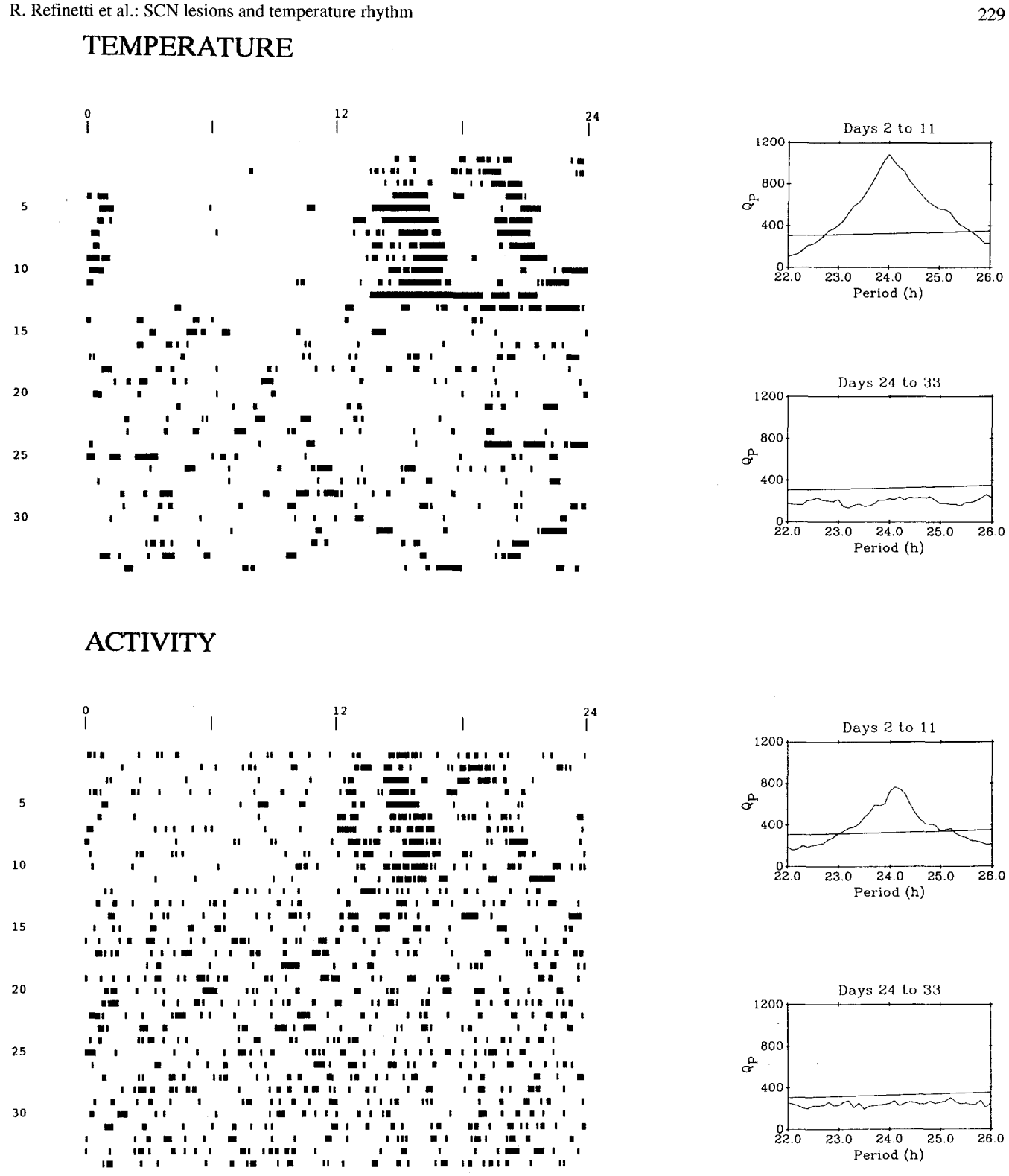


Fig. 11 Actogram and periodograms of the body temperature and activity rhythms of an animal that received a complete SCN lesion. The *straight line* in the periodograms indicates the 0.05 level of significance with correction for multiple testing. A photomicrograph of the brain of this animal is shown in Fig. 9B

nated by SCN lesions. As shown in Fig. 3, SCN lesion resulted in an almost flat temperature profile, with no pattern of circadian oscillation. Circadian rhythmicity in locomotor activity was also eliminated, although some ultradian variability persisted, as is commonly reported.

The full 34 days of data for the SCN-lesioned animal whose rhythms were illustrated in Fig. 3 are shown in the

form of actograms in Fig. 4. Body temperature shows robust circadian rhythmicity during the 11 days preceding the lesion ($Q_P = 866$, $P < 0.01$) but not during the following 22 days ($Q_P = 270$, $P > 0.05$). The locomotor activity rhythm, which was less robust than the temperature rhythm before the lesion ($Q_P = 445$, $P < 0.01$), was also eliminated by the lesion ($Q_P = 301$, $P > 0.05$).

The mean circadian rhythmicity indexes for all animals under both ambient temperatures are shown in Fig. 5. Regarding body temperature, analysis of variance indicated a significant effect of surgical treatment ($F(2,33) = 27.17$, $P < 0.001$) but not of ambient temperature ($F(1,33) = 1.03$, $P > 0.10$). Post-hoc pairwise comparisons by Tukey's HSD test revealed that, at either ambient temperature, the mean rhythmicity index was significantly ($P < 0.01$) smaller in SCN-lesioned animals than in either of the other groups. Although the index for operated controls was significantly smaller than that of unoperated controls, it was significantly larger than that of SCN-lesioned animals. Equivalent results were obtained in the analysis of the locomotor activity data.

The mean results for the period, amplitude, and mean level of the temperature rhythm in the three groups of animals are shown in Table 1. Analysis of variance and post hoc comparisons showed no significant effect ($P > 0.05$) of control lesions or ambient temperature on the free-running period. The amplitude of the body temperature rhythm was significantly affected by SCN lesions ($P < 0.005$) as well as by ambient temperature ($P < 0.001$). In all three groups of animals, amplitude was approximately 0.4°C larger at 12°C than at 22°C. At either ambient temperature, amplitude was significantly smaller in SCN-lesioned animals than in animals of either of the other groups. Finally, only ambient temperature had a significant effect on the mean level of body temperature ($P < 0.001$). In all three groups, body temperature was 0.3°C lower at 12°C than at 22°C.

To verify that the elimination of circadian rhythmicity by SCN lesions was not a transient phenomenon, 4 animals were studied for longer than the standard 31 days. Body temperature records of an animal that was studied for 60 days are shown in Fig. 6. Circadian rhythmicity, which was present during the 4 days prior to the lesion, was eliminated for the remainder of the experiment.

In the histological analysis, 3 out of the 12 operated control hamsters showed no sign of damage to the SCN (e.g., Fig. 7A). The remaining 9 animals showed very small partial SCN lesions (about 2–8%), with 8 of these being in the far rostral region and one in the caudal end of the SCN. Examination of the brains of hamsters in the SCN-lesion group indicated that 11 out of the 12 animals had complete bilateral ablation of the SCN (e.g., Fig. 7B), while one animal had unilateral destruction of one nucleus and approximately 60% destruction of the other.

Experiment 3: Brain lesions in animals maintained in constant darkness

The robustness of the rhythms of body temperature and locomotor activity was drastically reduced after the SCN lesions in all animals. The mean rhythmicity index for 10 days preceding the lesions and three blocks of 10 days after the lesions is shown in Fig. 8. Rhythmicity was reduced below the significance level immediately after the lesions and was not recovered for the duration of the experiment. Factorial analysis of variance revealed significant effects of both time ($F(3,70) = 264.3$, $P < 0.001$) and variable ($F(1,70) = 38.5$, $P < 0.001$). Post-hoc pairwise comparisons of means indicated significant ($P < 0.01$) differences between the pre-lesion rhythmicity of temperature and activity and between pre-lesion and post-lesion values in both variables. Although a small increase in the rhythmicity of body temperature along the three post-lesion blocks can be observed in Fig. 8, no significant differences ($P > 0.10$) were found for any pairs of post-lesion means.

Histological examination of brain slices (VIP- and GFAP-stained sections) confirmed the complete bilateral destruction of the SCN in 9 of the 11 animals. The two remaining animals sustained incomplete lesions that spared 10% and 30% of the SCN. Photomicrographs of VIP-stained sections of the brains of two animals, one with an incomplete lesion and the other with a complete lesion, are shown in Fig. 9.

Although visual inspection of actograms of the two animals with incomplete SCN lesions did not indicate the presence of circadian rhythmicity, weak albeit significant rhythmicity was detected by periodogram analysis. Actograms and periodograms of one of the animals sustaining incomplete lesion of the SCN are shown in Fig. 10. The post-lesion robustness of the activity rhythm was reduced below the significance line but the robustness of the temperature rhythm remained slightly above the significance line. In none of the animals sustaining complete SCN lesion (Fig. 11), did post-lesion robustness of rhythmicity stay above the significance line.

Discussion

Visual observation and periodogram analysis of our results indicate that circadian rhythmicity of both body temperature and locomotor activity are eliminated in hamsters with complete SCN lesions. This is in agreement with a number of previous studies in other rodent species (Eastman et al. 1984; Honma et al. 1988; Ruby et al. 1989; Ruis et al. 1987; Stephan and Nunez 1977) but is in contrast with several other studies (Dunn et al. 1977; Fuller et al. 1981; Powell et al. 1980; Satinoff and Prosser 1988). The interpretation of the results in the latter studies may have been predicated on the assumption that the temperature and activity rhythms are equally robust. Our comparison of the robustness of the temperature and activity rhythms shows that this is not the case in untreated

hamsters. Inspection of the lower panel in Fig. 1 indicates clearly that the rhythm of general locomotor activity is significantly less robust than the rhythm of body temperature. Even without brain lesions, a displacement of the significance line up by a small amount (200 points) would support the interpretation that the temperature rhythm persists while the activity rhythm is eliminated. In animals with partial SCN lesions, a similar interpretation could result from a proportional reduction in the robustness of both rhythms. In experiment 2, we observed a reduction in the robustness of the temperature and activity rhythms as a result of extra-SCN lesions, most of which extended slightly into the SCN (Fig. 5). Had we used a somewhat lower significance level (i.e., a slightly higher critical value of Q_P [e.g., 400]), we would have seen elimination of rhythmicity in activity but not in body temperature. In experiment 3, we observed elimination of rhythmicity in activity but not in body temperature in two animals with incomplete SCN lesions (e.g., Fig. 10). On the basis of our results, which show that partial lesions have differential effects on the expression of the two rhythms, we suggest that the persistence of the body temperature rhythm in SCN-lesioned animals in previous studies was due to the differential effect of partial lesions on the activity and temperature rhythms. However, since a number of investigators have observed persistence of the CRT in animals that, as determined by currently available histological techniques, had complete SCN lesions (Kittrell 1991), full confirmation of our suggestion must await new advances in neuroanatomical research.

The interpretation that complete SCN lesions may spare the temperature rhythm while eliminating the activity rhythm is weakened by the facts that in some of the studies body temperature was the only variable actually recorded (Dunn et al. 1977; Powell et al. 1980) and that in others rhythmicity in locomotor activity was occasionally observed in animals considered to have complete SCN lesions (Fuller et al. 1981; Satinoff and Prosser 1988). From this and from our own results we conclude that the available evidence does not support the hypothesis that the body temperature rhythm is controlled by a pacemaker distinct from the main circadian pacemaker located in the suprachiasmatic nuclei. Naturally, our conclusion is based on the assumption that the golden hamster is as adequate a model of circadian rhythmicity as the laboratory rat or the squirrel monkey. To avoid precipitous generalizations from one species to another, it would be appropriate to repeat the kind of studies that we have reported here using laboratory rats and other mammalian species.

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