highly motivated students were tested. Are the SMPY results indicative of the general population? Lowering qualifications for the talent search did not result in more high-scoring individuals (except in 1972, which was a small and not widely known search), suggesting that the same results in the high range would be observed even if a broader population were tested. In addition, most of the concern about the lack of participation of females in mathematics expressed by Ernest (*11*) and others has been about intellectually able girls, rather than those of average or below average intellectual ability.

To what extent do girls with high mathematical reasoning ability opt out of the SMPY talent searches? More boys than girls (57 percent versus 43 percent) enter the talent search each year. For this to change our conclusions, however, it would be necessary to postulate that the most highly talented girls were the least likely to enter each search. On both empirical and logical grounds this seems improbable.

It is hard to dissect out the influences of societal expectations and attitudes on mathematical reasoning ability. For example, rated liking of mathematics and rated importance of mathematics in future careers had no substantial relationship with SAT-M scores (*6*). Our results suggest that these environmental influences are more significant for achievement in mathematics than for mathematical aptitude.

We favor the hypothesis that sex differences in achievement in and attitude toward mathematics result from superior male mathematical ability, which may in turn be related to greater male ability in spatial tasks (*12*). This male superiority is probably an expression of a combination of both endogenous and exogenous variables. We recognize, however, that our data are consistent with numerous alternative hypotheses. Nonetheless, the hypothesis of differential course-taking was not supported. It also seems likely that putting one's faith in boy-versus-girl socialization processes as the only permissible explanation of the sex difference in mathematics is premature.

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21 March 1980; revised 14 August 1980

Human Sleep: Its Duration and Organization Depend on Its Circadian Phase

Abstract. *Two- to threefold variations in sleep length were observed in 12 subjects living on self-selected schedules in an environment free of time cues. The duration of polygraphically recorded sleep episodes was highly correlated with the circadian phase of the body temperature rhythm at bedtime and not with the length of prior wakefulness. Furthermore, the rate of REM (rapid eye movement) sleep accumulation, REM latency, bedtime selection, and self-rated alertness assessments were also correlated with the body temperature rhythm.*

Forty years ago, Kleitman wrote that "the time between going to bed at night and getting up in the morning is one of the easiest characteristics of sleep to study" (*1*). Despite this ease of measurement, the processes that control the length of "ad libitum sleep" (that is, sleep not truncated by an alarm clock or disturbance) have remained undefined. A number of studies have contradicted the intuitive assumption that the length of sleep is determined by the length of prior wakefulness. "Recovery" sleep after 3 to 10 days of total sleep deprivation rarely exceeds 11 to 16 hours (*2*), while both longer (15 to 20 hours) and shorter (6 to 10 hours) sleep episodes have been observed in subjects not deprived of sleep who lived on a self-scheduled routine (*3-6*). In fact, the wide variation in sleep duration reported in such "free-running" subjects has been characterized as random and irregular. We now report that such variations in sleep duration occur in a consistent and predictable manner which depends on when subjects go to sleep, rather than how long they have been awake beforehand.

We polygraphically recorded the sleep of 12 male subjects (21 to 53 years old), each living separately for 16 to 189 days (total of 562 days) on a self-scheduled routine in an environment free of time cues (*3, 4*). These subjects developed free-running, non-24-hour sleep-wake, body temperature, and neuroendocrine cycles. In one group of subjects, all those free-running rhythms remained internally synchronized with nearly identical periods, although their waveshapes and phase relationships were different from those during entrainment to a 24-hour day. For example, the decrease in body temperature that has long been associated with the daily sleep episode (*1*) began several hours before sleep in those free-running subjects, reaching its nadir near sleep onset and then rising throughout the rest of the sleep episode (*3-5*).

However, six of our subjects had a number of sleep-wake cycles of extraordinary duration—up to 50 hours in length—with a persisting near-24-hour rhythm in body temperature. This state, which several others have observed (*5, 6*), has been termed "internal desynchronization" by Aschoff and Wever, a concept emphasizing the uncoupling of rhythms that are normally linked in close temporal order.

Examination of the bedrest-activity pattern, when plotted in a raster format (*7*), led us to recognize—even in such "desynchronized" subjects—regularly recurring clusters of short (6 to 10 hours) sleep episodes, interrupted by long episodes of sleep which were not "in phase" with those clusters (triple plotted example from subject CA shown in Fig. 1 on experimental days 35 to 83). The visible line along which those clusters recurred had a consistent phase relationship to the ongoing near-24-hour cycle of body temperature (stippled area in Fig. 1); that is, the short sleep episodes usually began just at or after the mid-trough of the temperature cycle. This phase relationship was very similar to that already

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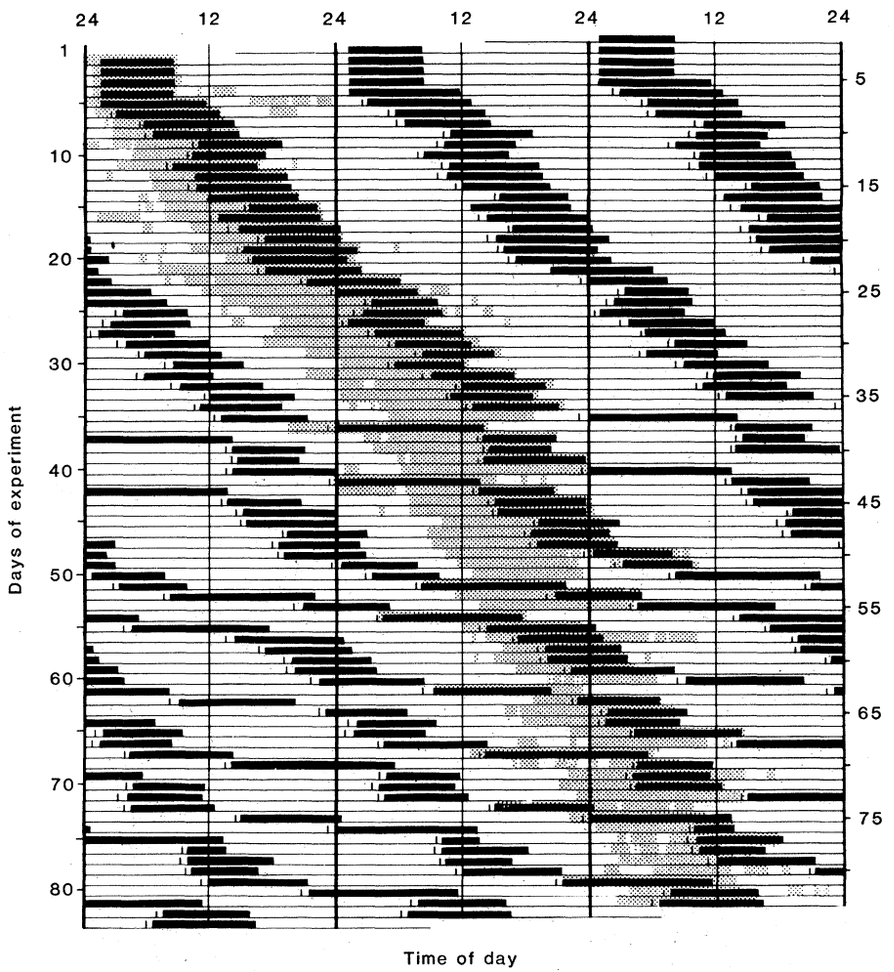
described for the "internally synchronized" free-running subjects. Furthermore, in our long-term studies (subject CA: 3½ months; DC: 6 months), the independently computed (*3*) period lengths of the body temperature cycle and that cluster line of short sleep episodes were exactly the same (both 24.5 hours in subject CA and both 25.1 hours in DC).

These relationships are more extensively demonstrated for subject CA in Fig. 2. The duration of sleep (Fig. 2A) varied with the phase of the educed temperature waveform (Fig. 2E) at bedtime. When the subject's chosen bedtime was near the temperature cycle minimum (−30° to +30°), where 0° is equal to the mid-trough of the average temperature cycle waveform (see legend to Fig. 1), the average sleep duration was 7.8 ± 0.3 hours (S.E.M.); whereas if he chose to go to sleep at the temperature cycle maximum (150° to 210°), the sleep length averaged 14.4 ± 0.35 hours ($P < .001$). This relationship persisted even when the chosen bedtimes followed more than 20 hours of prior wakefulness (closed circles in Fig. 2A). No significant correlation ($r = +.41$) was observed in these experiments between sleep duration and the length of prior wakefulness, provided that the subjects had been awake for at least 14 hours (*8, 9*).

Not only was the sleep length primarily dependent on its phase within the averaged temperature cycle, but we also found that all free-running subjects chose to go to bed at certain phases of that temperature cycle much more frequently than at others (*3, 10*). The greatest bedtime frequency was just after the circadian temperature cycle minimum (0°



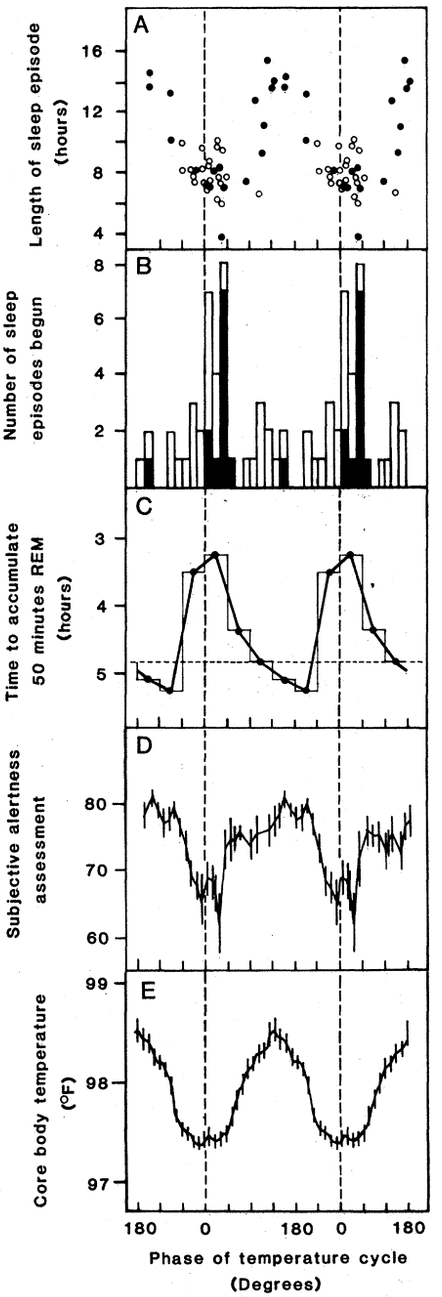


Fig. 1 (left). Scheduled (days 1 to 5) and self-selected (days 5 to 83) rest-activity pattern of a normal 23-year-old male subject (CA) over 83 days while living without the knowledge of time. Data are triple-plotted in a raster format, with successive days plotted both next to and beneath each other. Thin horizontal lines indicate waking hours; rest time is indicated by heavy black horizontal bars. Thin vertical hatch marks indicate the beginning of bedtime preparations on unscheduled "nights." A single plot of the times that the body temperature was below the normal entrained mean (98.02°F; 36.68°C) is overlaid with stippling. Fig. 2 (right). (A) The self-selected duration of sleep as a function of the phase of the circadian body temperature cycle at bedtime, from days 35 to 83 in subject CA (Fig. 1), when the average period of the rest-activity cycle (29.3 hours) was different from that of the body temperature cycle (24.5 hours). Closed circles indicate bedtimes after more than 20 hours of prior wakefulness. (B) Histogram showing the number of bedtimes selected at different circadian phases. Those sleep episodes in which REM sleep occurred within 10 minutes after bedtime are indicated by solid vertical bars. (C) The average number of hours after bedtime required to accumulate 50 minutes of REM sleep at different circadian phases. Average baseline value (from scheduled days 1 to 5) indicated with horizontal dashed line. (D) Average subjective alertness assessments at different circadian phases. Self-reports were taken at frequent intervals on a nonnumeric, continuous vertical scale, marked only "very alert" at the top (= 100) and "very sleepy" at the bottom (= 0). (E) Educed waveform of core body temperature cycle at period length (24.5 hours) estimated for this section of data by the technique of minimum variance (*3*); 0° is equal to the mid-trough of educed temperature waveform.

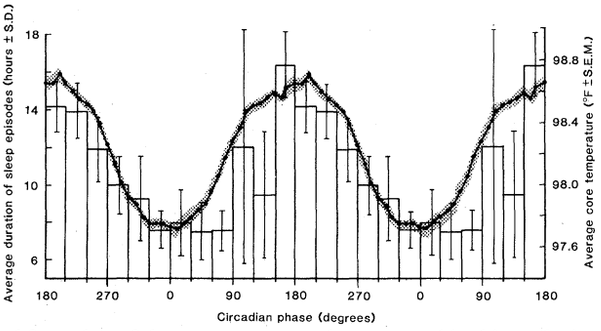


Fig. 3. Relation of average self-selected sleep episode duration to the educed body temperature waveform in data from 192 days of recordings in five subjects (AC, AD, AJ, CA, and DC). (Solid line) Mean core temperature; (open bars) average sleep length for sleep episodes begun within each phase interval. Only those sections of data in which the average period of the rest-activity cycle differed from that of the temperature cycle were used in this analysis; 0° is equal to the mid-trough of educed temperature waveform.

to 60°, illustrated in Fig. 2B for subject CA, corresponding to the nadir of the subjective alertness assessment curve shown in Fig. 2D. (Alertness was measured only during self-selected wake times, without the confounding influence of sleep interruption or sleep deprivation.)

The internal structure of sleep also varied with the phase of the temperature cycle (Fig. 2C). The first 50 minutes of REM (rapid eye movement) sleep were accumulated an average of 2 hours earlier when sleep began just after the trough of the temperature cycle as compared to sleep beginning just after the temperature cycle maximum, although this was not the case for stages 3 and 4 sleep (*3*). We conclude that the long-recognized variation in REM sleep with the time of day (*11*) is based on a close relation between a rhythm in REM sleep propensity and the body temperature rhythm and that both can oscillate together at a period different from that of the sleep-wake cycle itself (*10*). This result is also consistent with our report of an internal phase advance of both temperature and REM sleep, but not slow-wave sleep, under free-running conditions (*3, 4*). Recognition of this REM sleep propensity rhythm also explains, in part, the sporadic occurrence of sleep onset REM (SOREM) episodes in normal free-running subjects (*3, 6, 12*), a very rare phenomenon normally diagnostic of patients with narcolepsy (*13*). Yet in our normal subjects, we found that SOREM's reliably occurred (dark bars in Fig. 2B for subject CA) when they chose to go to bed just after the temperature cycle trough, at the peak of the REM accumulation (Fig. 2C) and sleepiness (Fig. 2D) curves (six of seven sleep episodes begun between 40° and 60° in subject CA were SOREM's).

Not all rhythms remained synchronized to the very stable circadian body temperature rhythm in these subjects. We have reported that the timing of growth-hormone secretion, peripheral skin temperature, and sleep stages 3 and 4 were more closely linked to the sleep-wake cycle itself than to the core temperature rhythm. In addition, a component of the cortisol secretion rhythm (like the rhythm of temperature itself) has been shown to be related to the sleep-wake pattern, whereas another component of the cortisol rhythm remained coupled to the body temperature rhythm (*3, 4*).

Using the circadian temperature rhythm as a convenient rhythmic marker for the group of mutually coupled circadian rhythms that have a stable period, we analyzed the relation of sleep duration to circadian phase across subjects. The average sleep durations from 192 days of experimental data obtained from five of our subjects who had recurrent rest-activity cycles of extraordinary duration are shown in Fig. 3. Sleep episode durations were averaged separately for all sleep episodes begun at each phase of the body temperature cycle. The dependence of the average sleep length on the circadian phase of the core temperature rhythm—and therefore to the oscillations coupled to it—is evident. The body temperature waveform averaged over many cycles was used to determine these phase positions, not the actual temperature recorded on a given day.

When bedtimes occurred at the trough of the averaged temperature cycle (that is, near the peak of the averaged sleepiness curve), sleep episodes were short, with wake times occurring on the rising phase of the temperature cycle. When bedtimes occurred at or after the peak of the temperature (and alertness) cycles, the duration of sleep was extended, such that wake times occurred on the next upslope of the temperature curve. In fact, 86.1 percent of all 151 wake times in these five subjects occurred on the rising phase of the temperature rhythm (0° to 180°). There was considerable variability in sleep duration when bedtimes occurred between 90° and 150° which appears to represent a breakpoint in the curve (Fig. 2A).

We obtained further support for a relation between the mean core body temperature cycle, sleep duration, sleep tendency, and REM sleep propensity by reanalyzing published data from several laboratories. Although not previously recognized, other subjects studied in free-running conditions, whether in caves or in isolation facilities (*5, 6*), have shown the same dependence of reported sleep duration on circadian temperature phase described here (*3, 9*). Furthermore, we have noted that all such free-running subjects living for more than 2 months without the knowledge of time have eventually shown this alternating pattern of long and short sleep episodes (*3*). Reanalysis of the published data of Weitzman *et al.* (*14*), who studied subjects living on an imposed ultradian sleep-wake schedule (two wake-time hours and one bedtime hour per cycle) for 10 days, demonstrates the same approximate relationship between the circadian REM sleep propensity rhythm and that of the average body temperature cycle (*3, 10*), even on that potentially disruptive schedule.

That these relations, revealed under free-running conditions, are relevant to the normal 24-hour scheduled sleep-wake cycle is shown by the data of Åkerstedt and Gillberg (*15*). They studied unrestricted sleep in normal subjects who were kept awake for a variable number of hours, such that their scheduled bedtimes occurred at systematically varied clock hours throughout the day and night. Despite the potential confounding influence of sleep deprivation inherent in such a design, they found a similar relation between the sleep length, REM tendency, sleepiness, and core body temperature at different times of day in the entrained state, as we have described here among those rhythms in the free-running state.

Our results in free-running subjects demonstrate unequivocally that the phase of the endogenous circadian system, rather than the duration of prior wakefulness, is the major determinant of the length of sleep in normal man. Furthermore, they refute the notion that the timing and organization of sleep in man are ever free from the influence of the group of rhythms marked by the circadian rhythm of core body temperature. Our earlier observation (*3*) that all human subjects studied to date living without the knowledge of time in an unscheduled environment for more than 2 months have progressively developed these same consistent bedrest-activity

patterns indicates the generality of these findings. This has formed the basis for the development by Kronauer *et al.* of a mathematical model of the human circadian timing system, using two interacting oscillators (*16*), that may correspond to specific anatomical structures within the human brain (*17*). The success of that mathematical model in reproducing the patterns observed (Fig. 1) supports our analysis of the data. These findings have major implications for understanding the timing of human sleep and may also help explain the sleep-wake patterns in shift workers and in certain clinical sleep disorders (*18*).

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8. When the length of the rest-activity cycle period was << 24 hours (*5*), the length of the waking episodes was also much shorter. Nonetheless, a cluster line of 6- to 10-hour sleep episodes continued to occur with their usual phase relation to the temperature rhythm. However, sleep episodes begun out of phase with the cluster line were now very short (< 4 hours) instead of very long (> 12 hours). Thus, prior wakefulness was only a factor in determining whether sleep episodes of abnormal length were very short or very long (*9*).
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24 March 1980; revised 11 September 1980

Light Suppresses Melatonin Secretion in Humans

Abstract. *Bright artificial light suppressed nocturnal secretion of melatonin in six normal human subjects. Room light of less intensity, which is sufficient to suppress melatonin secretion in other mammals, failed to do so in humans. In contrast to the results of previous experiments in which ordinary room light was used, these findings establish that the human response to light is qualitatively similar to that of other mammals.*

The physiology of the pineal gland and its hormone melatonin have been studied extensively in mammals (*1*). Both nocturnal and diurnal animals synthesize and secrete melatonin almost exclusively during nighttime darkness (*2*), a pattern consistent with the suppression of melatonin synthesis by environmental light (*3*). Even in constant darkness this 24-hour secretory rhythm persists (*4*), driven by an endogenous circadian oscillator in the suprachiasmatic nucleus (SCN) of the hypothalamus (*5*). A neuronal pathway links the SCN to spinal nuclei of sympathetic neurons that innervate the pineal gland (*6*). Neuronal input from the retina to the SCN (via the retinohypothalamic tract) mediates the suppressant effect of light and the entrainment of the melatonin secretory rhythm to the light-dark cycle (*5*).

Although in humans there is a nocturnal increase in melatonin secretion which appears to be mediated by sympathetic neurons (*7*), previous studies have failed to demonstrate a pronounced suppressant effect of light (*8–11*). Consequently, some investigators have proposed that the regulation of melatonin secretion in humans is substantially different from that of all other mammals (including nonhuman primates) and have speculated that escape from direct control by the environmental light-dark cycle has conferred on humans an evolutionary advantage (*12*). We examined this apparent difference and report that light of higher intensity than that used in previous studies unequivocally suppresses melatonin secretion in humans.

Six normal subjects (four females and two males), who gave written informed consent, were each studied on two separate occasions. Blood was sampled at intervals through an indwelling catheter. Between 11 p.m. and midnight on each night of the study the subjects retired to a dark room to sleep; at 2 a.m. they were awakened and exposed to light for 2 hours. On one night fluorescent light was used (Vita-Lite, ≃ 500 lux at eye level—the approximate intensity used in home or industrial conditions), and on another night incandescent light was used (150-W flood lamps, ≃ 2500 lux at eye level—the approximate intensity of indirect sunlight measured 1 inch from a window on a clear spring day). At 4 a.m. the subjects resumed sleeping in the dark. The two male subjects were studied under two additional conditions: on a third night they were exposed to approximately 1500 lux of incandescent light between 2 a.m. and 4 a.m., and on a fourth occasion they slept in the dark throughout the night (*13*). The concentration of melatonin in the plasma was assayed by gas chromatography–negative chemical ioni-

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