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Sleep-Onset Insomniacs Have Delayed Temperature Rhythms

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Summary: It was predicted from free running and ultradian cycle studies that sleep-onset insomniacs would have endogenous circadian rhythms that were phase delayed compared to good sleepers. Thirteen sleep-onset insomniacs and nine good sleepers were selected to differ only in their sleep-onset latencies as confirmed by polysomnography. Their rectal temperatures were measured over a 26-h constant routine and analyzed with best-fit Fourier curves including 24-h fundamental and 12-h harmonic components. The temperature rhythm markers of the insomniacs' rhythms were approximately 2.5 h later than the respective phases of the good sleepers. The usual bedtimes of the insomniacs fell within the "wake maintenance zone" of their delayed temperature rhythm. The good sleepers had typical bedtimes several hours after their "wake maintenance zone" and closer to their body temperature minimum. It was suggested that manipulations to phase advance the insomniacs' rhythms would reduce their sleep-onset latencies. It was also predicted that early morning insomnia results from phase advanced circadian rhythms and that sleep maintenance insomnia results from an abnormal phase relationship between the 24-h temperature rhythm and 12-h sleep–alert rhythm. **Key Words:** Sleep onset—Insomnia—Circadian rhythms—Temperature rhythm—Wake maintenance zone.

Sleep-onset insomnia is typically characterized by frequent difficulty initiating sleep with little or no difficulty in maintaining sleep once initiated. Survey studies have shown that sleep-onset insomnia is the most common form of sleeping difficulty and is reported by approximately 7.5% of the general adult population (1,2). The typical treatment of this sleep disorder is the prescribed use of hypnotic drugs, which aims to provide symptomatic relief but fails to address the underlying cause. Hypnotic drug use can have attendant side effects of withdrawal rebound insomnia, drug dependence, and daytime sedation (3). A better understanding of the cause of sleep-onset insomnia may result in improved treatments.

It has been suggested that an important cause of insomnia is the disruption of the

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body's circadian rhythms (4,5). More recently Strogatz and Kronauer (6) identified what they termed "wake maintenance zones" (WMZ) or particular phases of the body's circadian temperature rhythm during which sleep onsets were not selected in free running studies. Strogatz analyzed the sleep–wake and temperature rhythm data of 15 subjects, contributed from various laboratories, whose sleep–wake cycles and deep body temperature rhythms were desynchronized (7). From the total of 355 sleep episodes in this group of subjects, sleep onsets should have occurred equally at all phases of the temperature rhythm. In fact, very few sleep onsets occurred in a zone about 6 to 9 h before mid-trough temperature and about 4 to 7 h after mid-trough temperature. These WMZs were consistent across a diversity of subjects. The free running studies established that subjects do not choose to go to bed during these WMZs. The question as to whether they could initiate sleep during these zones was answered by a number of ultradian sleep–wake cycle studies (e.g., "90 min-day, 3 hour-day") (8,9). In these studies, the shortest sleep onset latencies (SOLs) and maximum sleep times occurred when bedtime was around the body temperature minimum. In contrast, the least amount of sleep and longest SOLs occurred about 8 to 9 h before the body temperature minimum was reached. This latter period of minimum sleep tendency corresponds to the evening WMZ identified by Strogatz and Kronauer (6). Thus, during the WMZ, subjects rarely attempt to make this their bedtimes, but if they do, then sleep onset is unsuccessful or markedly delayed.

Strogatz (7) suggested that the relatively uncommon delayed sleep phase insomnia may result from a delayed temperature rhythm. If the temperature rhythm is delayed such that the body temperature minimum is, for example, at 8 a.m., then bedtimes before 2 a.m. would fall in the evening WMZ and would result in long SOLs. The regular experience of long SOLs would be perceived as sleep-onset insomnia. A delay of circadian rhythms has recently been reiterated as a cause of sleep-onset insomnia (10).

Two studies have reported differences in the body temperature rhythms of insomniacs compared to good sleepers (11,12). McFarlane and co-workers (11) measured the tympanic membrane temperature of eight chronic primary insomniacs over 37 consecutive hours, including two sleep periods and the intervening day. The type of insomnia (e.g., sleep onset, early morning awakening) was related to the pattern of temperature cycle disruption (phase delayed and phase advanced rhythms, respectively). A similar study by Lack and colleagues (12) measured rectal temperature every minute over a period of a week in a group of eight poor sleepers and eight good sleepers. Compared to the good sleepers the average temperature rhythm of the poor sleepers showed a higher mean temperature from 2 h before to 4 h after the time of sleep onset. The pattern was suggestive of a phase delay in the rhythms of the insomniacs. Both of these studies used a variety of types of insomnia, including sleep onset, sleep maintenance, early awakening, and mixed insomnias. Because the different types of insomniacs would be predicted to have different types of circadian rhythm abnormalities, sometimes in opposite directions, it is necessary to test the rhythms of each type separately.

The previously mentioned studies were both carried out on subjects who were ambulatory and maintained a normal sleeping and waking behavior. Under these conditions body temperature is affected by body activity (13), meals (14), ambient temperature (15), and sleep (16–20), all of which can mask the effect of the endogenous circadian pacemaker on body temperature. A procedure to eliminate or to hold constant these exogenous influences on body temperature was first described as a "constant

routine'' by Mills and co-workers (21) and was refined by Czeisler and associates (22). Therefore, the aim of this study was to use the constant routine to investigate the endogenous circadian temperature rhythm of sleep-onset insomniacs.

METHOD

Subjects

Subjects were volunteer recruits from the university student population and the general public. All were in self-reported good health with regular sleep–wake patterns. They were between 20 and 45 years of age with no evidence of hypnotic drug use or above average alcohol or caffeine consumption. From their responses to a sleep questionnaire the control group of subjects was selected using the criteria of self-reported good sleep with an SOL less than 15 min and the absence of nocturnal awakenings or early morning awakening. Sleep-onset insomniacs were selected using the criteria of reported SOL greater than 45 min with an absence of nocturnal or early morning awakenings and with a duration of this pattern of at least 5 years. On the basis of these criteria, nine control and 13 sleep-onset insomniac subjects were selected with mean ages of 31.2 years and 33.7 years, respectively. All subjects signed consent forms after being fully informed of the procedures of the study. There were three women in the control group and five in the insomniac group. Those who were ovulatory had laboratory sessions scheduled within 5 days after menstruation to avoid body temperature elevation following ovulation.

Apparatus

Rectal temperature and wrist activity were recorded using a Vitalog PMS-8 ambulatory recorder. Sampling rate was at 3-min intervals. Rectal temperature was measured with a Yellow Springs Series 400 indwelling thermistor probe inserted 8 to 10 cm deep. Wrist activity was measured with a wrist activity sensor containing omnidirectional mercury tilt switches. The Vitalog was interfaced with an Apple IIe computer for the purposes of Vitalog initialization, start-up, data readout, and data storage.

A Devices M-19 polygraph was used to record the polysomnographic (PSG) measures of electro-encephalogram (EEG), electro-oculogram (EOG), and respiration. The EEG channel recorded bipolar activity between Cz and O_2 electrode positions of the standard 10-20 system. EOG electrodes were placed 1 cm above and lateral to the outer canthus of the left eye and 1 cm below and lateral to the outer canthus of the right eye. The earth electrode was placed on the mastoid process of the right ear. Electrode impedances were all below 5K ohms. Respiration was measured with an elastic chest strap in series with a strain gauge transducer.

Procedure

Before the laboratory session, subjects kept a sleep–wake diary for 2 weeks on which they recorded their lights-out times (LOT), wake up times (WUT), sleep onset latencies (SOL), nocturnal awakenings (NA), and total awake time after sleep onset (WASO), as well as meal times, vigorous exercise, alcohol and caffeine ingestion, and taking of any medications. Female subjects were also asked to record the period of menses. The diaries served the purposes of determining the average LOT for each subject and of confirming the initial selection criteria of the two groups.

The laboratory session included one night of polysomnography followed by a 26-h

constant routine. Subjects arrived at 1800 h after their evening meal on the night of the laboratory session. They attended to any toileting requirements, fixed in place the Vitalog sensors, dressed in sleeping attire, went to bed about 1900 h, and engaged in quiet activity (e.g., reading or watching television). One hour before their habitual lights-out time, as determined by the average LOT over the 2-week sleep–wake diary keeping period, the PSG sensors were attached. Lights were then turned off and quiet conditions ensued from their normal LOT. The following morning subjects were aroused and lights were turned on at their normal wake-up time or no later than 0830 h. PSG sensors were removed; subjects attended to any toileting requirements and returned to bed to commence a 26-h constant routine. During this period, subjects remained awake in a near supine position with head and shoulders slightly elevated. Air conditioning kept ambient temperature constant at 20°C and kept humidity constant. Illumination by incandescent overhead light was kept constant at 40 lux as measured with a Gossen Lunasix light intensity meter incident at the subject's head position. This relatively dim ambient light was the minimum required for reading.

Subjects' normal 24-h caloric intake was evenly distributed across the constant routine period with iso-energetic snacks given at 2-h intervals. The snacks consisted of approximately half of a thin sandwich, a quarter piece of a fruit (e.g., apple or pear), and a plain oatmeal cookie. A 250-ml drink of water was given each hour to ensure adequate hydration and maintain a regular fluid intake. Urine was collected regularly with bedpans.

Subjective arousal was kept as low as possible while ensuring wakefulness. Subjects could read, write, listen to music, or watch television or video tapes. Experimenters were in attendance during the entire routine to ensure that wakefulness was maintained. Although subjects experienced greatest feelings of sleepiness sometime between 0200 h and 0800 h, quiet verbal interaction with the experimenter was sufficient to maintain the subjects' wakefulness during these sleepy periods.

RESULTS

Analysis

A common way of examining circadian variation is to assume a 24-h period and calculate a best-fit cosine function to obtain a phase and amplitude estimate. However, because there is evidence for a 12-h rhythm of sleepiness, which may be evident in the 24-h temperature data (23), we decided to use a periodic regression analysis as described by Bliss (24) and Walters and Curtis (25). This analysis uses the first two components in a Fourier series (the 24-h fundamental and 12-h harmonic) to obtain a best-fit periodic function. The analysis allows the phases and amplitudes to be calculated for each component and the significance of each component independently to be tested using an analysis of variance (ANOVA) format. It also allows the phase and amplitude estimates to be compared between two different curves.

Group Differences

The Fourier ANOVA was applied to the last 24 h of temperature data from each subject in the constant routine. All subjects in both groups had significant ($p < 0.05$) 24-h fundamental rhythms and 12-h harmonic rhythms. The rhythm and sleep data means are shown for both groups as well as *t* test comparisons between the group

means in Table 1. There were no significant differences between the groups in mean temperature over the 24-h period, in mean amplitude of the 24-h rhythm, in the 12-h rhythm amplitudes, or in the percentage of variance of the temperature curve accounted for by the 24-h rhythm and the 12-h harmonic.

The only rhythm difference between the groups was in phase position of the rhythms. The phase position, which can be represented by the acrophase or by the fitted minimum value, was later in the sleep-onset insomnia group than in the control group. The mean acrophase of the insomnia group 24-h rhythm (1920 h) was significantly later ($p < 0.005$) than that of the control group (1556 h). The mean 12-h rhythm acrophase for the insomnia group (2021 h) was significantly later ($p < 0.005$) than that for the control group (1726 h). The combined components Fourier fitted curve of the insomnia group also had a mean minimum time (0718 h), which was significantly later ($p < 0.005$) than the mean minimum time (0315 h) of the control group. Thus, all measures of rhythm phase position show the rhythms of this group of sleep-onset insomniacs to be delayed about 3 to 4 h compared with good sleeping control subjects. All other rhythm characteristics of the two groups were similar.

Table 1 also shows the group means of the sleep parameters LOT, SOL, and WUT as derived from the PSG recording the night prior to the constant routine. The insomnia group mean SOL (42 min) was significantly longer ($p < 0.005$) than the mean SOL of the control group (11 min). No subject had nocturnal arousals. The insomnia group had a longer SOL mean despite their later LOT (2402 h versus 2306 h). However, the insomnia group mean LOT was 7 h before their mean temperature minimum and therefore within their evening WMZ (6 to 9 h before the fitted temperature minimum). The control group mean LOT was 4 h 16 min before their mean temperature minimum and significantly closer to the minimum than the insomniac group ($t = 4.78$, $df = 20$, $p < 0.001$). No control subject had a LOT within his or her WMZ estimated from the constant routine, whereas eight of 13 insomniac subjects had LOTs within their individually estimated WMZs.

TABLE 1. *Means of temperature rhythm data and sleep parameters of the control group and sleep-onset insomnia group and t test comparison between the group means*

Variable	Controls	Insomniacs	*t* value	p value
Temperature data				
Mean rectal temperature amplitude	36.91°C	36.90°C	0.07	ns
24-h fundamental	0.212°C	0.267°C	1.33	ns
12-h harmonic	0.105°C	0.100°C	0.22	ns
Percentage variance from				
24-h fundamental	67%	66%	0.10	ns
12-h harmonic	12%	13%	0.18	ns
Acrophase				
24-h fundamental	1556 h	1920 h	3.20	0.005
12-h harmonic	1726 h	2021 h	2.88	0.005
Time of minimum temperature in fitted Fourier curve	0315 h	0718 h	7.31	0.005
Wake maintenance zone (est.)	1815 h–2115 h	2218 h–0118 h		
Polysomnographic data				
Lights-out time	2306 h	2402 h	2.40	0.025
Sleep onset latency	11 min	42 min	6.30	0.005
Wake up time	0821 h	0806 h	1.12	ns

ns, not significant.

Sleep Adjusted Group Rhythms

We have shown that the phase positions of the temperature rhythms as well as the LOTs for the insomnia group were later than those of the control group. It could be argued that the insomniacs simply have a later lifestyle and perhaps are extreme evening types (26). We wanted to determine if their chronic sleep onset difficulty was due to their chosen bedtimes in relation to their endogenous temperature rhythms rather than simply their later endogenous rhythms. We have already shown that the control subjects' bedtimes are significantly closer to their times of minimum body temperature. To illustrate this, we decided to combine the temperature rhythms of subjects within each group to obtain average group curves. However, because within each group, the subjects' bedtimes and sleep onset times varied, averaging by clock times would include temperatures from some subjects asleep and some awake at clock times around the average bedtime. Such an average by clock time would not illustrate the typical temperature curve with respect to the significant state transition of sleep onset. Therefore, we averaged body temperatures with respect to the time of sleep onset as determined by PSG in the night prior to the constant routine. The average body temperatures for each group were obtained from 14 h before to 10 h after this sleep onset time.

Figure 1 shows the best-fit Fourier curves for the sleep-onset insomniac group and control group. The percentages of the control and insomniac mean temperature curves accounted for by the fitted Fourier curves were 90% and 95%, respectively. Thus, the fitted curves accurately represent the 480 data points from each mean curve. The acrophase of insomniac group was 2 h 40 min later than that of the control group, which was a significant difference [$F(1,959) = 1{,}176.2$, $p < 0.001$]. Included in Fig. 1, for illustrative purposes, are the evening WMZs and the mean LOTs for each group. It can be seen that the mean LOTs for the insomniac group fall within their WMZs whereas

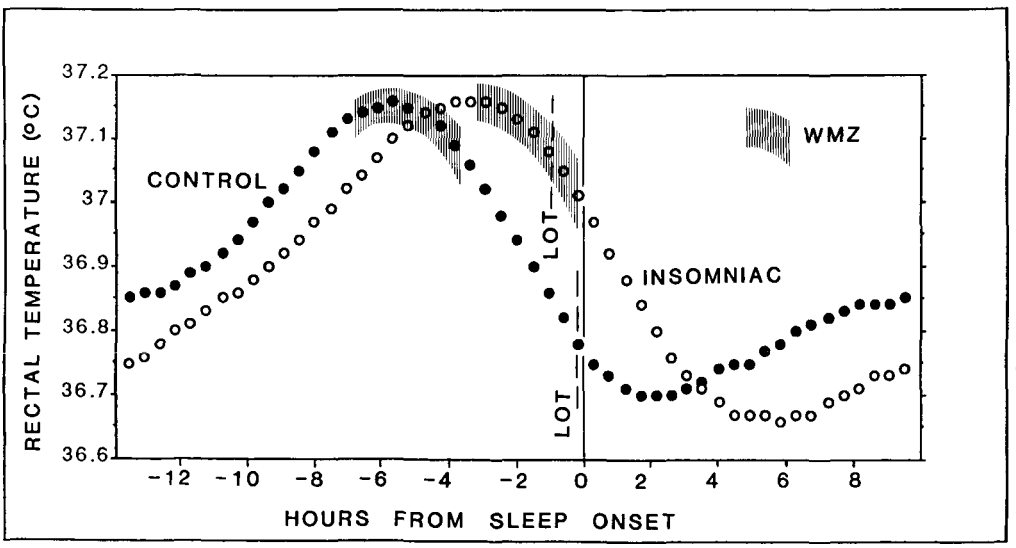

FIG. 1. Fitted Fourier curves to the control group and insomniac group mean 24-h temperature data in the constant routine relative to subjects' usual sleep onset times (vertical solid line). The usual mean lights out times (LOT) for each group are indicated as vertical dashed lines. The estimated mean wake maintenance zone (WMZ) for each group is indicated as a shaded area.

the LOTs for the control group are considerably after their evening WMZs and about 3 h before their fitted temperature minimum.

Fundamental and 12-h Harmonic Components

Figure 2 shows both Fourier components of the curve fitted to the sleep adjusted mean curves of each group. For reference purposes, the fitted temperature minimums have been indicated by vertical dashed lines on each figure. The phase relationships between the components are the same in each figure. In both figures the acrophases of the 12-h harmonic are just over 1 h later than the acrophase of the 24-h fundamental. There was no significant difference in this phase relationship between the insomniac and the control group. Both the acrophases in the insomniac group curves were about 2.5 h later than their respective acrophases in the control group curves. The phase relationships of both components to the fitted minimum phase were also similar in both groups. The fitted minimums occurred about 1 h after the 12-h harmonic minimums and about 3 h before the 24-h fundamental minimums in both groups. Thus, the phase relationships between Fourier components and between the components and the fitted temperature minimum are consistent in both groups. The whole circadian system relative to sleep onset is simply delayed by about 2.5 h in the insomnia group compared to the control group.

Figure 2 also indicate the four periods of high and low sleep probability relative to the temperature minimum that have been identified in earlier studies. The evening WMZ coincides with the first 12-h peak in both groups. The morning WMZ coincides with the second 12-h peak. The "post-lunch dip" or sleep-onset zone 1 (SOZ1) coincides with the 12-h trough 1, and the maximum sleepiness time (SOZ2) coincides more closely with the second 12-h trough than it does to the 24-h trough. Therefore, the periods of lowest and highest probability of falling asleep coincide with the two peaks and troughs, respectively, of the temperature rhythm 12-h harmonic rather than any obvious phases of the 24-h fundamental rhythm.

DISCUSSION

The main finding of the study is that the body temperature rhythm of sleep-onset insomniacs is phase delayed compared with good sleepers. The phase difference between the groups of fitted temperature minimums was in the order of 4 h of clock time (0315 h versus 0718 h). Because the insomniacs generally had later LOTs, body temperature values were adjusted to each subject's typical sleep-onset time before being averaged for the two groups. Thus, after controlling for differences of habitual sleep-onset times, the insomniac average temperature rhythm was still approximately 2.5 h later than the rhythm of the good sleepers. A majority of the insomniacs had typical LOTs within their evening WMZs as estimated from their endogenous temperature rhythms and Strogatz's predictions (7). None of the good sleepers had typical bedtimes near their estimated WMZs. These findings suggest that the sleep-onset difficulties and the long SOLs of the insomniacs result from phase delayed temperature rhythms which place the WMZs of the rhythm across the typical bedtimes of the insomniacs. It follows from this logic that if the insomniacs delayed their bedtimes sufficiently (2 to 3 h), they would have less or little trouble falling asleep. However, most people usually have constraints on their schedules that necessarily require wakening by 0700 to 0800 h most mornings. A delayed bedtime—say to 0300 h or 0400 h, would result in even less sleep

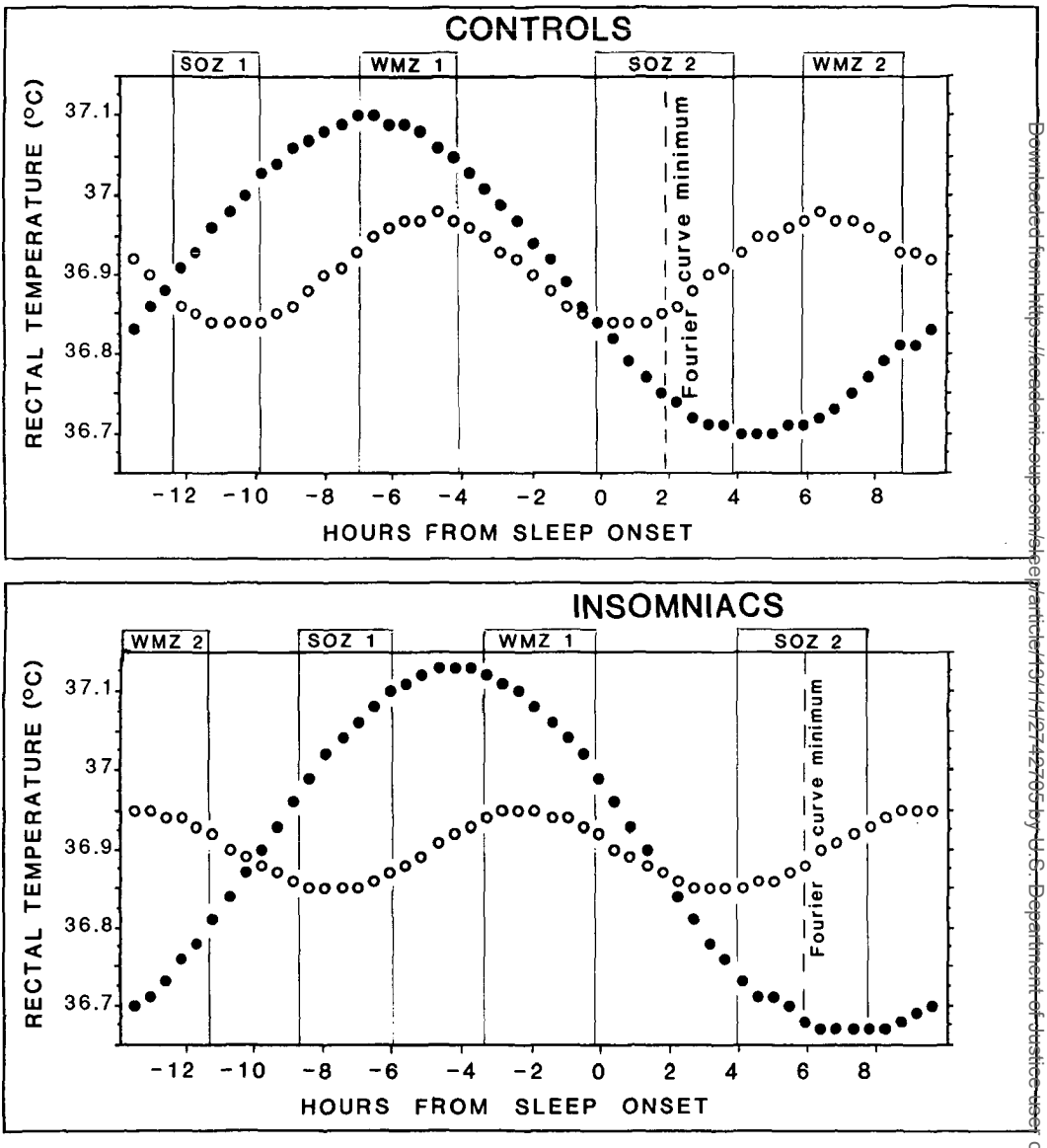

FIG. 2. The 24-h fundamental (filled circles) and 12-h harmonic (open circles) components of the Fourier curve fitted to the mean temperature data of the control group (**top panel**) and insomniacs (**bottom panel**) relative to usual sleep onset time. The Fourier curve minimum temperature for each group is indicated by a vertical dashed line. With respect to these minimums the estimated evening and morning wake maintenance zones (WMZ 1 and WMZ 2, respectively) and afternoon ("post-lunch dip") and nocturnal sleep onset zones (SOZ 1 and SOZ 2, respectively) are indicated for each group.

than usual and be less desirable than suffering a delayed sleep onset at an earlier bedtime. Thus, in practice, sleep-onset insomnia is associated with a delayed circadian temperature rhythm.

Although Strogatz's predictions regarding ease of sleep onset were with respect to the body temperature rhythm (7), the ease of sleep onset may be strongly associated

with the rhythms of other variables that are correlated with body temperature rhythm. Other variables in addition to core temperature that are included in the total circadian system are melatonin, cortisol, and urine volume (27,28). It would, therefore, be appropriate to investigate other circadian rhythms in sleep-onset insomniacs to confirm the delay of the circadian system in general.

The results of this study also raise the question of the circadian rhythm parameters of other types of insomniacs besides sleep-onset insomniacs. Early morning awakening insomnia is characterized by relatively rapid sleep onset, reasonably undisturbed sleep, but premature early awakening (e.g., 0300 h) with an inability to reinitiate sleep. This can result in insufficient sleep and partial sleep loss as well as the frustration and boredom of trying to return to sleep without success. Early morning insomnia may be the opposite of sleep-onset insomnia and result from an advanced instead of delayed circadian rhythm phase. Strogatz showed that the frequency of wake-ups in free running studies had a single broad peak in the 24-h period around 6 h after the body temperature trough coinciding approximately with the morning WMZ (7). Zulley and co-workers have also identified a "wake-up zone" 3 to 7 h after the body temperature minimum (29). If the circadian system were advanced relative to sleep onset, this wake-up period, instead of beginning 7 h after sleep onset, as in our control subjects, may arrive only 3 to 4 h after sleep onset and terminate the sleep period before sufficient sleep is obtained. If sleep onset is at 2200 to 2300 h, the early morning awakening will occur around 0200 to 0300 h, the typical pattern in early morning insomnia.

A secondary finding of this study, with considerable heuristic value, derives from the use of the Fourier analysis to establish the amplitude and phase of a 12-h harmonic rhythm embedded in the average temperature curves. We mentioned earlier that the free running studies of desynchronized subjects and the ultradian studies identified two phases of the body temperature rhythm that were sleepy or sleep onset zones (SOZ) and two phases that were alert periods, wake-up periods or WMZs. Several other studies corroborate the existence and timing of these four zones. Carskadon and Dement studied objective sleepiness as measured with multiple sleep latency tests across a constant routine, and their observation of unintended microsleeps shows all four zones (30,31). In a "disentrained" free running environment, Campbell and Zulley showed peaks and troughs of sleep onsets corresponding to the two WMZs and SOZs, respectively (32). In a traditional free running study, in which naps did occur contrary to instructions, the naps occurred in the midday SOZ (33). Studies of extended (>12 h) sleep have shown a return of slow wave sleep about 11 to 12 h after the first peak of SWS, also suggesting a 12-h rhythm of sleepiness (34–36). The four points of maximum and minimum sleepiness shown in past studies show a remarkable coincidence with the peaks and troughs of the 12-h harmonic in the present study. In the 24-h fundamental component, only the trough phase corresponds closely to the maximum sleepiness period. The two WMZs correspond, however, to the beginning of the descending phase and the beginning of the ascending phase of the 24-h rhythm. The afternoon sleepy period ("post-lunch dip") corresponds approximately to the latter part of the ascending temperature phase of the 24-h rhythm. The placement of these latter three periods with respect to the 24-h rhythm does not have high face validity. On the other hand, the correspondence of these four periods to the peaks and troughs of the 12-h harmonic component makes greater intuitive sense. It may indicate an independent 12-h sleepiness rhythm that influences body temperature and generates the 12-h component in the body temperature rhythm.

In this study, the 12-h component had a consistent relationship to the 24-h component in both groups. It was also the case that all subjects, once they initiated sleep, slept without interruption for at least 7 h. This absence of sleep interruptions may be facilitated by the particular phase relationships between the 12-h sleepiness rhythm and the 24-h temperature rhythm such that nocturnal sleep is of best quality and length when a 6-h trough phase of the 12-h rhythm coincides with the descending and trough phase of the 24-h rhythm.

Not all persons may have this phase relationship between 12-h and 24-h components. If the 12-h rhythm is shifted about 3 h in the delay direction with respect to the 24-h rhythm, the troughs of both components would coincide. If it is assumed that the 12-h rhythm represents a sleep-alert rhythm and the 24-h rhythm is the temperature rhythm, which also affects sleepiness independently, the coincidence of both troughs should result in maximum sleepiness but over a reduced duration. This phase relationship may be predicted to exist in good short sleepers who appear to obtain sufficient sleep over 4 to 6 h and who show high sleep efficiency with reduced wakefulness, stage 1 and stage 2 sleep, but a normal amount of slow wave sleep (37–39). In other words, this coincidence of trough phases may strip away "optional" sleep and leave what Horne refers to as "core" sleep (40).

Alternatively, if the 12-h rhythm is advanced by about 3 h relative to the 24-h rhythm compared to the phase relationship as shown in Fig. 2, a peak of the sleep-alert rhythm will coincide with the trough of the 24-h temperature rhythm. This would create opposing sleep tendencies and result in less than maximum sleepiness. This phase relationship may exist in persons suffering from sleep maintenance insomnia who show longer nocturnal awakenings and more nocturnal awake time than normal sleepers (41). The coincidence of the 24-h trough with a 12-h peak would result from a temperature rhythm in which the trough is attenuated or flattened out. This tendency was apparent in Monroe's poor sleepers (42) and more recently in the ambulatory temperature study by Lack and co-workers, whose insomniacs were mainly of the sleep maintenance type (43). Although many self-reported sleep maintenance insomniacs may be discovered to have sleep apnea, nocturnal myoclonus, or behavioral or psychological disorders (44), another factor which should now be considered is the phase relationship between the 24-h and 12-h components of the body temperature rhythm.

A non-intuitive finding by Seidel and Dement (45) and Stepanski and colleagues (46) that also may be explicable from the reasoning above is the discovery that insomniacs appear no more sleepy during the day than good sleepers despite the fact that insomniacs generally get less total sleep time and are subjectively more tired than normal. When sleepiness was objectively measured with the multiple sleep latency test (MSLT) in middle-aged and older insomniacs, most likely the sleep maintenance type, somewhat less sleepiness (not significantly) was found during the day than in normal controls (45,46). Stepanski and co-workers also found a significant negative correlation only in the insomniac group between the number of brief arousals from sleep and the sleepiness index from the MSLT. In other words, those subjects who showed more arousals during sleep took longer to fall asleep during the day. These results would, in fact, be predicted if sleep maintenance insomnia occurs when the peak of the 12-h sleep/alert rhythm coincides with the trough of the 24-h temperature rhythm during the nocturnal sleep period. In that phase relationship it would also be the case that the earlier of the two 12-h peaks would coincide with the peak of the 24-h rhythm. Thus, both the sleep-alert rhythm and temperature rhythm peaks would contribute to increased

arousal over much of the daytime and result in decreased sleepiness according to the MSLT.

Implications for Treatment

This study has established that the endogenous circadian temperature rhythms of sleep-onset insomniacs are delayed compared to those of normal sleepers with respect to clock time, to LOT, and to sleep onset time. It is possible that this rhythm delay is an important etiological factor in the sleep onset disorder. To confirm the causal link suggested by these findings the circadian temperature rhythm in the insomniacs could be manipulated by resetting the phase position to an earlier time. If their rhythms were advanced or reset 2 to 4 h earlier and SOLs were subsequently normalized, it would establish the circadian rhythm delay as a significant cause of an important and common form of insomnia.

If phase advance of the circadian rhythm normalized SOL, it would be recommended as a potential nonpharmacological therapy for sleep-onset insomnia. Delayed sleep phase syndrome (DSPS) is most probably caused by a delay of the circadian system that is more extreme than found in the insomniacs of the present study. DSPS also results in sleep-onset insomnia if sleep is attempted earlier than 0300 to 0400 h. Weitzman and colleagues (47) devised and demonstrated the efficacy of a treatment program for DSPS termed chronotherapy. Because phase advance seemed impossible with DSPS patients, chronotherapy consisted of sleep delays over several successive days until the target bedtime (e.g., 2300 h) was achieved and then maintained. During and after the chronotherapy, subjects experienced rapid sleep onsets at bedtime and sufficient total sleep time. We believe that the sleep-onset insomniacs of the present study are essentially less extreme cases of DSPS than described in the Association of Sleep Disorder Centers (ASDC) nosology (44) and as treated by Weitzman and associates with chronotherapy (47). There is some evidence that the more mild form of DSPS is more prevalent than DSPS and is quite common in some subgroups, such as adolescents and university students (48–52). If the sleep-onset insomnia we have studied is basically a mild form of DSPS, chronotherapy could be considered as a possible treatment, at least for the insomniacs with more extreme rhythm delays.

However, for many sleep-onset insomniacs the 7 to 10 days required to carry out chronotherapy may be too impractical to be considered. Other procedures have also shown some ability to shift the phase of the body temperature rhythm in humans. Exposure to bright light in the evening produces a phase delay of the circadian system (53–55) and exposure to bright light in the morning has the capacity to advance the circadian rhythm phases of winter depressives and possibly sleep-onset insomniacs (53–56). Early morning aerobic exercise, independent of bright light, may also have phase advancing potential (57). These procedures need their phase advancing capacity and their possible benefit to sleep-onset insomniacs investigated further.

Possible Causes of Phase Delay

If, as suggested by the present findings, a delayed circadian system is a major cause of sleep-onset insomnia, it would be important to investigate the origins of this delay. The circadian rhythm delay may arise from a behavioral factor, such as a late life style with frequent late bedtimes and waketimes, particularly on weekends. This would allow the circadian rhythm of body temperature, which in most people has an endogenous period length of about 25 h, to delay to a later clock time. The evening WMZ may then

coincide with bedtimes on weeknights, resulting in frequent sleep-onset difficulty. For these persons, consistent early waketimes may be sufficient to phase advance their temperature rhythm and decrease the sleep-onset difficulty.

Biological factors contributing to the delayed rhythm may involve a longer endogenous period length or a weaker advance portion of the phase response curve, or both. Because most people have an endogenous period length longer than 24 h, there is a tendency to delay with respect to the 24-h day. This tendency is used to explain the greater speed of re-entrainment following westward (delay) time zone changes and the cessation of summer daylight savings time (1 h delay) compared with eastward time zone shifts or phase advances (58). Those persons who have longer than average endogenous temperature rhythms would be predicted to have a greater tendency to delayed circadian rhythms. Some persons with particularly long (e.g., 25.5 to 26.0 h) endogenous period lengths may find the 24-h daily period close to the limits of their entrainment capability. In other words, for a person with an endogenous period length of 26 h, a phase advance of 2 h every 24 h would be required to avoid further delays with respect to clock time. Because this degree of phase shift would be extremely difficult to maintain continuously, these persons would be prone to phase delay with respect to the 24-h clock. Thus, the prediction that sleep-onset insomniacs would tend to have longer than normal endogenous circadian period lengths should be tested.

Sleep-onset insomniacs may also have abnormal phase response curves (PRC) in which the phase advance portion is weaker than normal. This was suggested as a cause of DSPS by Weitzman and colleagues (47). It may result from a reduced sensitivity to one or more of the normal environmental entraining stimuli or a reduced phase advancing effect from correctly timed exposure to these entraining stimuli. To summarize, the phase delay of sleep-onset insomniacs may arise from the behavioral factor of late and irregular sleep patterns, a longer endogenous period length, or a deficiency of the phase advance portion of the PRC. Of course, people may possess any combination of these independent factors. A person showing all three factors would have great difficulty avoiding a free running delaying circadian system in the normal environment.

Nosology of Sleep-Onset Insomnia

One final consideration stemming from the findings and predictions of this study relate to the nosology of sleep disorders. At present, delayed and advanced sleep phase syndrome are placed in a category of disorders of the sleep-wake schedule (44). If it eventuates that sleep-onset insomnia and early morning insomnia arise mainly from delayed and advanced circadian systems, respectively, this should be recognized in the diagnostic classification of sleep and arousal disorders, perhaps as a separate category within the main section on DIMS (disorders of initiating and maintaining sleep), as suggested by Askenasy (59). The clinical significance of circadian phase delays and advances would then be more directly related to insomnia.

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