Do chronic primary insomniacs have impaired heat loss when attempting sleep?

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Gradisar, Michael, Leon Lack, Helen Wright, Jodie Harris, and Amber Brooks. Do chronic primary insomniacs have impaired heat loss when attempting sleep? Am J Physiol Regul Integr Comp Physiol 290: R1115–R1121, 2006. First published November 23, 2005; doi:10.1152/ajpregu.00266.2005.—For good sleepers, distal skin temperatures (e.g., hands and feet) have been shown to increase when sleep is attempted. This process is said to reflect the body’s action to lose heat from the core via the periphery. However, little is known regarding whether the same process occurs for insomniacs. It would be expected that insomniacs would have restricted heat loss due to anxiety when attempting sleep. The present study compared the finger skin temperature changes when sleep was attempted for 11 chronic primary insomniacs [mean age = 40.0 years (SD 13.3)] and 8 good sleepers [mean age = 38.6 years (SD 13.2)] in a 26-h constant routine protocol with the inclusion of multiple-sleep latency tests. Contrary to predictions, insomniacs demonstrated increases in finger skin temperature when attempting sleep that were significantly greater than those in good sleepers (P = 0.001), even though there was no significant differences in baseline finger temperature (P = 0.25). These significant increases occurred despite insomniacs reporting significantly greater sleep anticipatory anxiety (P < 0.0008). Interestingly, the core body temperature of insomniacs (37.0 ± 0.2°C) was significantly higher than good sleepers (36.8 ± 0.2°C; P = 0.03). Whether insomniacs could have impaired heat loss that is masked by elevated heat production is discussed.

TEMPERATURES OF DISTAL SKIN regions (i.e., hands and feet) have been shown to increase before sleep onset (11, 13, 24, 26–28, 31, 32, 46). These distal skin temperature increases have been interpreted as the body’s action to lose heat from the core via the extremities (29), with the difference between distal and proximal skin regions (i.e., torso) considered the best predictor of sleep propensity (27, 28). It has been further proposed that this peripheral heat loss may be primarily governed by a decrease in sympathetic nervous system (SNS) activity as individuals relax and attempt sleep (32). However, these processes have been studied mainly in good sleepers.

Unlike good sleepers who are generally relaxed when attempting sleep, individuals experiencing insomnia are often not relaxed, even to the point of being anxious when retiring for bed and attempting sleep (1, 8). This anxiety, or inability to relax, may interfere with the normal decrease of SNS activity or even produce an increased SNS activity. This could result in the attenuation of the normal distal skin temperature increase, even producing a temperature decrease, both of which should be associated with a lengthening of sleep latency.

Some evidence does exist that insomniacs have different distal skin temperature changes when attempting sleep. Compared with good sleepers, insomniacs have been found to have significantly lower finger temperatures from lights out through to stage 2 sleep onset (16). Although for the most part, insomniacs do show increases in toe skin temperature when attempting sleep, sometimes there is no observable change (11). When toe temperature increases are observed, they are more variable and can take twice as long to reach the same amount of temperature change compared with those in good sleepers (11). These variable and lengthy temperature changes occur in conjunction with longer sleep latencies (11, 16). However, once sleep is achieved, differences in finger temperature between insomniacs and good sleepers disappear (16).

Lights out has been recognized as an implicit cue to attempt sleep (30). Given that insomniacs are anxious when attempting sleep (8) and that anxiety-provoking stimuli result in decreased distal skin temperatures (22, 23), it is likely that insomniacs would show an attenuated distal skin temperature increase, no change at all, or even a decrease in response to lights out and the sleep attempt. It is therefore the aim of the present study to investigate the distal skin temperature changes between insomniacs and good sleepers before sleep onset. If the normal distal skin temperature increases are attenuated (or even decrease) for insomniacs, this may be related to their typical lengthened sleep latency, as has been shown with experimental manipulations of proximal skin temperatures in good sleepers (39).

Because distal skin temperature increases before sleep onset are not restricted to a normal individual’s typical bedtime (32), the present study used a 26-h modified constant routine (CR) method with half-hourly sleep-latency tests (SLTs) to test the difference in distal skin temperature changes before sleep onset at all circadian phases between good sleepers and chronic insomniacs.

MATERIALS AND METHODS

Participants. Twelve insomniacs (9 women, 3 men; mean age = 40.5 years, SD = 12.7) and eight good sleepers (5 women, 3 men; mean age = 38.6 yrs, SD = 13.2) participated in the study. Insomnia subjects were recruited through a newspaper advertisement. They were assessed via a Sleep History Questionnaire devised by the Flinders University Sleep Research Laboratory and 2 wk of sleep diaries as having difficulty initiating sleep [e.g., sleep onset latency (SOL) >30 min at least 4 nights per week] and subsequent negative daytime consequences (e.g., fatigue). Insomnia participants reported daytime sleepiness in the normal range (i.e., <10), as determined by the Epworth Sleepiness Scale (ESS) (21), and normal levels of depression (i.e., <10) (as determined by the DASS-21) (34). No physical conditions disrupted their sleep (e.g., headaches, diabetes, Raynaud syndrome, back pain), and participants were not on any medications known to affect sleep or temperature. Insomniacs did not meet criteria for delayed sleep phase syndrome, as assessed by the
Sleep History Questionnaire, two 7-day sleep/wake diaries, and phone interviews by a sleep therapist. Because insomnia participants were also participating in two other projects associated with the present experiment (9, 18), they were reimbursed A$50 for their part in the present experiment. After data collection was complete, it was discovered that one of the insomniacs had a comorbid severe obstructive sleep apnea condition, despite her ESS score being very low (i.e., ESS = 2). Therefore, her data were excluded from the analyses, leaving an insomnia sample of eight women and three men (mean age = 40.0 years, SD = 13.3). The mean (SD) length of chronicity of their insomnia was 11.8 (10.7) years.

Good sleepers [i.e., group-matched on age, sex, and body mass index (BMI)] were recruited from the Flinders University Employment Service. These control subjects were selected to be good sleepers in good health (i.e., SOL of <20 min; wake after sleep onset <20 min; total sleep time = 7.49 ± 0.9 h; sleep efficiency >85%), with no physical conditions disturbing their sleep. Other exclusion criteria for all subjects included smokers, excessive consumers of alcohol (>2 standard drinks per day) or caffeine (>3 cups per day), users of prescribed medication, clinical levels of depression (DASS-21), excessive daytime sleepiness (ESS), and extreme morning or evening types (as determined by the Time of Day Preference Scale) (20). Control subjects were paid A$300 for their participation. Females from both groups participated during the follicular phase of their menstrual cycle to avoid any possible effects of ovulation and the luteal phase on sleepiness and temperature measures (14, 15). The study was approved by the Social and Behavioural Ethics Committee of the Flinders University of South Australia. Informed consent was obtained from all subjects.

Participants from both groups were instructed to maintain their regular sleep/wake pattern for 2 wk before the experiment. All participants complied with these instructions, as indicated by wrist activity monitors (ActiTrac, IM Systems, Baltimore, MD) and sleep/wake diaries. Participants were instructed to avoid caffeine for 1 wk and alcohol for 3 days before the experiment.

Design. The experiment used a two-way mixed model design. The experiment consisted of a 26-h laboratory-modified CR session. The CR was modified by the inclusion of multiple sleep-latency tests (12) conducted every 0.5 h.

Procedure. Before the 26-h-modified CR, participants from both groups completed the Sleep Anticipatory Anxiety Questionnaire (8). Although the Anxiety subscale of the DASS-21 measures general levels of anxiety, it was considered not sensitive enough to measure specific levels of anxiety associated with attempting sleep. For this purpose, the Sleep Anticipatory Anxiety Questionnaire was used.

Subjects arrived at the Flinders University Sleep Research Laboratory at ~1800, where they were fitted with EEG (Cz, Oz) and EOG electrodes. A rectal thermistor (YSI 400 series in-dwelling thermistor probe; Yellow Springs Instruments, Yellow Springs, OH) was self-inserted, and a skin thermistor (YSI 400 series probe 409B, time constant = 1.1 s; Yellow Springs Instruments) taped to the palmar surface of their right index finger. Fingertip temperature (T_fing) was used as the measure of distal skin temperature because it has been found to be a sensitive psychophysiological measure of both anxiety (22, 23) and distal skin temperature changes before sleep onset (32). Furthermore, T_fing has very good correlations with skin temperature gradients (3), which have been previously used as indirect measures of distal vasodilation and heat loss (27, 28, 40). Thus heat loss will be inferred from T_fing for the present paper. Subjects were instructed to keep their right hand outside of the bedcovers at all times. The 26-h CR began with the first SLT occurring at 1930.

During lights-on wakeful periods, subjects were instructed to remain in bed in a near-supine position with head and shoulders slightly raised. Activity was restricted to quiet activities. Small snacks were given every 2 h, and water was provided when required. The 26-h CR was conducted in constant environmental conditions (i.e., ambient temperature 20 ± 0.4°C, light intensity <50 lux) free of time cues. Subjects wore light bedclothes under light bed covers.

At the start of each SLT, subjects were instructed to imagine that they were at home lying in their own bed, ready to attempt sleep. They were then instructed to assume a comfortable sleeping position, close their eyes, remain still, and allow themselves to drift off to sleep. The lights were turned off, and the door was closed.

EEG and EOG were amplified at the bedside with a physiological acquisition amplifier (Flinders University Psychology Electronics Workshop). The amplified EEG and EOG signals were digitized at a sampling rate of 200 Hz and then inputted to a Power Macintosh computer in the adjacent experimental room. Rectal and skin temperature were digitized at a sampling rate of 200 Hz. Amplified electro-physiological and raw temperature data were continuously displayed and recorded using LabVIEW 5 software (National Instruments, Austin, TX). Rectal temperature data were averaged into 30-min bins, and T_fing data were averaged into 30-s bins. Finger temperature readings commenced before each SLT was started. LabVIEW 5 was used to detect the precisely quantified 50% decrease in alpha wave power as an indication of stage 1 sleep onset (33, 40).

Sleep latency trials commenced at lights out. Trials ceased, and subjects were awakened after three consecutive 30-s epochs of stage 1 sleep. If sleep onset did not occur in the 25-min opportunity, SOL was scored as 25 min. This occurred for 13% of all trials for the insomnia group, and 8% of all trials for the good sleepers. The 26-h CR finished with the last SLT occurring at 2130 on the second evening.

Statistical analyses. Because T_fing invariably increases from lights out to sleep onset (32), differences in sleep latency could contribute to differences in T_fing at sleep onset. To derive a measure of finger temperature change that was not confounded by sleep latency and yet reflected the degree of SNS withdrawal, the initial 5-min increase in T_fing was used. Calculations of the T_fing increase were made for each subject for each trial by using the following formula: T_fing (6 min) − T_fing (1 min), where the T_fing at 1 min typically occurred in conjunction with lights out. A 5-min T_fing increase was used because the vast majority of all trials contained the greatest rate of increase in this timeframe. In addition, this included 99% of all trials before sleep onset.

Each subject’s core body temperature (Tc) minimum was visually identified from their raw rectal temperature data using a 24-h cosine plus 12-h harmonic best-fit curve (insomnia average R2 = 0.75; good average R2 = 0.88) with the software program Kaleidagraph (Synergy Software, Reading, PA). This method also provided data for the amplitude and mesor for Tc. The mean (SD) time of Tc minimum was calculated [insomniacs: 0500 (1.6 h); good sleepers: 0530 (1.3 h)]. After all of the individual subjects’ Tc minimums were aligned to the group minimum time, individual baseline T_fing, T_fing increase, and SOL data were realigned on the basis of their adjusted individual Tc. From this realignment, 26.5 h of data were calculated and used for analysis.

To test for significant variation of baseline T_fing, T_fing increase, SOL, and Tc over time, between insomniacs and good sleepers, as well as any interaction effects, a series of nonlinear mixed model regressions were performed controlling for covariates (i.e., gender). SOL showed a significant, positive skew, and baseline T_fing showed a significant negative skew. SOL was log-transformed, and baseline T_fing was reflected and then log-transformed (44). Transformations of these variables resulted in normal distributions. For prelaboratory measures, independent Student’s t-tests were performed to determine any significant differences between insomniacs and good sleepers.

RESULTS

Characteristics of insomniacs and good sleepers. Table 1 presents the descriptive (mean and SD) and inferential statistics
Table 1. Descriptive and inferential statistics of age, BMI, and data from sleep/wake diary, wrist activity monitor, and questionnaires for insomniacs and good sleepers

<table>
<thead>
<tr>
<th></th>
<th>Insomniacs</th>
<th>Good Sleepers</th>
<th>t Value (P)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, years</td>
<td>40.0 (13.3)</td>
<td>38.6 (13.2)</td>
<td>0.22 (0.83)</td>
</tr>
<tr>
<td>BMI</td>
<td>24.3 (3.0)</td>
<td>23.2 (2.5)</td>
<td>0.91 (0.37)</td>
</tr>
<tr>
<td>*swd SOL, min</td>
<td>73.6 (61.4)</td>
<td>12.0 (6.6)</td>
<td>2.80 (0.01)</td>
</tr>
<tr>
<td>*swd WASO, min</td>
<td>83.8 (82.4)</td>
<td>16.3 (10.2)</td>
<td>2.29 (0.04)</td>
</tr>
<tr>
<td>*swd TST, h</td>
<td>5.6 (1.7)</td>
<td>7.5 (0.9)</td>
<td>2.94 (0.01)</td>
</tr>
<tr>
<td>*swd SE, %</td>
<td>67.4 (20.6)</td>
<td>93.2 (3.7)</td>
<td>3.46 (0.003)</td>
</tr>
<tr>
<td>*act SOL, min</td>
<td>36.7 (17.8)</td>
<td>14.2 (4.2)</td>
<td>3.48 (0.003)</td>
</tr>
<tr>
<td>*act WASO, min</td>
<td>76.5 (45.0)</td>
<td>11.6 (4.9)</td>
<td>4.04 (0.001)</td>
</tr>
<tr>
<td>*act TST, h</td>
<td>6.0 (1.3)</td>
<td>7.3 (0.7)</td>
<td>2.64 (0.02)</td>
</tr>
<tr>
<td>*act SE, %</td>
<td>64.8 (20.2)</td>
<td>94.4 (1.8)</td>
<td>4.12 (0.0009)</td>
</tr>
<tr>
<td>Depression</td>
<td>7.6 (8.6)</td>
<td>3.3 (4.0)</td>
<td>1.32 (0.21)</td>
</tr>
<tr>
<td>Anxiety</td>
<td>5.6 (3.1)</td>
<td>2.5 (3.2)</td>
<td>2.10 (0.05)</td>
</tr>
<tr>
<td>Stress</td>
<td>16.0 (10.7)</td>
<td>6.5 (7.8)</td>
<td>2.10 (0.05)</td>
</tr>
<tr>
<td>*SAAQ</td>
<td>14.6 (3.9)</td>
<td>6.0 (5.0)</td>
<td>4.13 (0.0008)</td>
</tr>
<tr>
<td>ESS</td>
<td>4.1 (2.0)</td>
<td>6.9 (3.6)</td>
<td>2.07 (0.06)</td>
</tr>
</tbody>
</table>

Values are means (with SD in parentheses). Actigraphy SOL was estimated from lights out to the first epoch of 5 min of no activity; Actigraphy WASO estimated by the number of 30-s epochs of movement. Act, wrist activity monitor; BMI, body mass index; ESS, Epworth Sleepiness Scale; SAAQ, Sleep Anticipatory Anxiety Questionnaire; SE, sleep efficiency; SOL, sleep onset latency; swd, sleep/wake diary; TST, total sleep time; WASO, wake after sleep onset. *Significance at P < 0.05. Differences tested with independent Student’s t-tests.

Contrary to the prediction that insomniacs would have lower T_fing than good sleepers, analysis of the baseline T_fing showed no significant main group effect, F(1,81.21) = 1.37, P = 0.25 (Fig. 1B). There was a trend for a significant interaction effect, F(53,465.86) = 1.36, P = 0.055, which may be explained by the good sleepers having lower mean finger temperatures in two periods over the 26-h CR (i.e., 1930 to 2130 on the first day and 1600 to 1800 on the second day; see Fig. 1B). A significant main time effect was also found, F(53,465.86) = 1.53, P = 0.01, indicating a circadian rhythm of baseline T_fing.

Surprisingly, analysis of the 5-min T_fing increased data showed a significant main group effect, F(1,216.05) = 10.55, P = 0.001, although in the opposite direction to that predicted, with the mean (SD) T_fing increase 5 min after lights out greater for insomniacs (1.5 ± 1.2°C) than for good sleepers (1.2 ± 1.2°C) (Fig. 1C). No significant interaction effect, F(53,464.68) = 0.60, P = 0.99, or main effect for time, F(53,464.68) = 0.59, P = 0.99, were found. The mean T_fing changes from baseline to 5 min after lights out are shown in Fig. 2.

Interestingly, when T_c was analyzed, a significant main effect for group was found, F(1,122.36) = 8.92, P = 0.007. Independent t-tests on the T_c mesor data demonstrated that insomniacs had significantly higher values (37.0 ± 0.5°C) than good sleepers (36.8 ± 0.2°C), t(17) = 2.41, P = 0.03 (see Table 2). No significant interaction effect was found, F(53,659.72) = 1.31, P = 0.07, although this approached significance. As expected, there was a significant main effect for time, F(53,659.72) = 5.66, P < 0.0001, demonstrating significant circadian variation (see Fig. 1D). No significant differences were found in the amplitude of T_c between the insomniacs (0.2 ± 0.1) and good sleepers (0.2 ± 0.1), t(17) = 0.28, P = 0.78. No other significant differences between the circadian rhythms of insomniacs and good sleepers were found (see Table 2).

Inspection of Fig. 1 clearly shows an offset of T_c between the two groups at all time points. However, baseline T_fing appears similar. This suggests that, during quiet wakefulness, there may be a difference in the core finger temperature gradient between insomniacs and good sleepers. Plots of core temperature against finger temperature for both groups also suggest this to be the case (Fig. 3), with the slope of the insomniacs T_c-T_fing gradient being more than twice the magnitude of the good sleepers (−0.12 vs. −0.05, respectively).

However, when a nonlinear mixed model regression was performed on the core finger temperature gradient data (i.e., T_c − baseline T_fing), no significant effect for group was found, F(1,83.45) = 0.72, P = 0.40, although there was a trend for a significant interaction effect, F(53,450.10) = 1.35, P = 0.056. A significant main effect for time was also found, F(53,450.10) = 1.94, P < 0.0001, indicating circadian rhythmicity.

With regard to the circadian rhythms of SOL and body temperatures, inspection of Fig. 1 demonstrates that SOL decreases in conjunction with a decline in T_c [insomniacs: r(54) = 0.53, P < 0.0001; good sleepers: r(54) = 0.70, P < 0.0001], and an increase in baseline T_fing [insomniacs: r(54) = −0.37, P = 0.004; good sleepers: r(54) = −0.58, P < 0.0001]. Core temperature and T_fing are also inversely correlated [insomniacs: r(54) = −0.69, P < 0.0001; good sleepers: r(54) = −0.58, P < 0.0001].
DISCUSSION

Using a similar CR protocol to an earlier study (32), we found that both insomniacs and good sleepers demonstrated consistent increases in finger temperature when attempting sleep. In fact, the magnitude and range of $T_{\text{fing}}$ increases for good sleepers in the present study are comparable to a previous study also using good sleepers (32). However, contrary to predictions, insomniacs had a greater increase of $T_{\text{fing}}$ than good sleepers. Therefore, the prediction that insomniacs would have lower and attenuated acute $T_{\text{fing}}$ changes before sleep...
no change at all (11). However, increases of Tfing in insomniacs do not support previous findings that an insomniac’s toe skin temperature changes during sleep latency trials for insomniacs and good sleepers. It therefore appears that these insomniacs had a Tfing increase greater than good sleepers. This does not support previous findings that an insomniac’s toe skin temperature changes during sleep latency trials for insomniacs and good sleepers.

With respect to circadian rhythms, insomniacs had a significantly higher Tc mesor than the good sleepers. No significant differences were found in SOL, the mean baseline Tfing, or the core-finger temperature gradient.

**Acute finger temperature changes during SLTs between insomniacs and good sleepers.** Across the 26-h CR, insomniacs demonstrated consistent Tfing increases during SLTs. This does not support previous findings that an insomniac’s toe skin temperature change when attempting sleep sometimes shows no change at all (11). However, increases of Tfing in insomniacs have been previously reported (16). Like most distal skin areas (including the feet), the palmar surface of the finger skin contains many small blood vessels known as arteriovenous anastomoses (AVAs) (42), which are integral in heat loss (17, 29). These AVAs are primarily controlled and innervated by sympathetic constrictor neurons (37). Thus a decrease in SNS activity would result in a reduced firing of these neurons, causing vasodilation of AVAs, and increased heat loss, as indicated by increasing finger temperature.

Insomniacs demonstrate not only Tfing increases during SLTs; in addition, these Tfing changes were greater than good sleepers. It therefore appears that these insomniacs had a Tfing response to the sleep attempt that was not impaired by sleep anticipatory anxiety. However, it should be noted that subjects from both groups reported their sleep anticipatory anxiety before the 26-h CR, and the questionnaire asks about feelings during their typical attempt to fall asleep in their home environment. The degree of sleep anticipatory anxiety in the home environment may not generalize completely, or indeed at all, to the laboratory environment. The fact that the greater sleep latencies of the insomniacs in the home environment are not perpetuated in the laboratory is consistent with the possibility that the sleep anticipatory anxiety was reduced or eliminated in the laboratory. However, sleep anxiety was not recorded continuously during the CR. Therefore, the extent to which sleep anticipatory anxiety during SLTs may have affected Tfing, as well as SOLs, is unclear. Future studies are needed to record sleep anticipatory anxiety in the laboratory, as well as finger temperature changes during sleep attempts in the home environment that show extended latencies to sleep onset.

**Tc.** The one striking difference from the averaged Tc curves was that the insomniacs had a higher Tc mesor. This concurs with previous research (1, 36). Interestingly, the mean Tc of sleep-onset insomniacs has been reported as equivalent to that of good sleepers (38), although their Tc rhythm was delayed. That study, however, used insomniacs with only sleep-onset difficulties (38). Studies with insomnia samples reporting greater time awake during the night show higher endogenous Tc than that shown in good sleepers (35), suggesting that the higher the Tc the more interruptions of sleep during the night.

Tc is regulated by the processes of heat loss (i.e., high distal skin temperature) and heat production (e.g., increased metabolic rate) (4, 5, 25). Tc becomes higher when heat production exceeds heat loss (4, 5, 25). A higher Tc would therefore be a product of increased heat production and/or decreased heat loss. Elevated Tc due to higher heat production? Although it has been suggested elsewhere that insomniacs have impaired heat loss (45), it appears not to be true for the insomniacs in the present experiment. Therefore, it seems likely that their insomniacs is related to excessive heat production. Because the higher Tc of insomniacs was observed in restful constant conditions, higher heat production is not due to greater activity but is more likely due to higher basal metabolic rate (6).

The mean Tc curve for insomniacs was offset ∼0.2–0.3°C above the good sleepers’ Tc curve. This occurred for both day and night phases. Insomniacs have been shown to have an elevated metabolic rate across the day and night (6, 7), which would result in greater heat production (4). As such, Tc would be elevated if heat production exceeded heat loss. Because the heat loss of insomniacs (as measured by the Tc-Tfing gradient) was equivalent to the good sleepers, it could be the greater heat production of insomniacs that contributed to the elevated Tc. It may be that the set point of Tc is effectively elevated for insomniacs, in which case the elevated Tc may be at an appropriate level. At this set-point level, there would be no signal to the thermoregulatory system to produce greater heat loss to downregulate Tc.

When attempting sleep, insomniacs clearly demonstrated a greater increase in Tfing. This could be explained if in fact it were the case that the Tc set point is not elevated for insomniacs but is due to other factors such as sleep anxiety or higher basal metabolic rate.

### Table 2. Descriptive statistics of temperature and sleepiness circadian rhythms for insomniacs and good sleepers

<table>
<thead>
<tr>
<th></th>
<th>Insomniacs</th>
<th>Good Sleepers</th>
<th>t Value (P)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tc, mesor, °C</td>
<td>37.0 (0.2)</td>
<td>36.8 (0.2)</td>
<td>2.41 (0.03)</td>
</tr>
<tr>
<td>Tc amplitude, °C</td>
<td>0.2 (0.1)</td>
<td>0.2 (0.1)</td>
<td>0.28 (0.78)</td>
</tr>
<tr>
<td>Tfing, °C</td>
<td>33.1 (1.1)</td>
<td>32.2 (2.3)</td>
<td>1.07 (0.30)</td>
</tr>
<tr>
<td>Tfing amplitude, °C</td>
<td>1.2 (0.8)</td>
<td>2.0 (1.7)</td>
<td>1.45 (0.16)</td>
</tr>
<tr>
<td>SOL, °C</td>
<td>8.2 (5.6)</td>
<td>7.5 (3.6)</td>
<td>0.40 (0.70)</td>
</tr>
<tr>
<td>Tc, minimum time</td>
<td>0500 (1.6)</td>
<td>0550 (1.3)</td>
<td>0.75 (0.46)</td>
</tr>
<tr>
<td>Peak Tc, time</td>
<td>0200 (1.9)</td>
<td>0230 (2.4)</td>
<td>0.37 (0.72)</td>
</tr>
<tr>
<td>Peak SOL, time</td>
<td>2100 (1.2)</td>
<td>2100 (1.3)</td>
<td>0.31 (0.76)</td>
</tr>
</tbody>
</table>

Values are means (with SD in parentheses). Tc, core body temperature; Tfing, fingertip temperature. Note that times for Tc, minimum and peak Tfing are for the 2nd day of the constant routine (CR); peak SOL is for the 1st day of the CR. Times are rounded to the nearest 0.5 h. All values are taken from the 24-h cosine plus 12-h harmonic-fitted curves of Tc, Tfing, and SOL. *Significance at P < 0.05.
In response to the sleep attempt, the thermoregulatory system may see this as an “opportunity” to lose heat to downregulate $T_c$ to a more appropriate level. Thus more research is needed to understand the thermoregulatory processes of insomniacs. For instance, simultaneously measuring the heat production and heat loss of insomniacs and good sleepers who have an equivalent $T_c$ mesor could help to unmask the effects of sleep anticipatory anxiety on body temperatures and sleep. Some caution should be made though of using fingertip skin temperature alone as an index of heat loss, as it may not be as accurate a measure as skin-temperature gradients. Nonetheless, such research is needed to determine more conclusively whether insomniacs not only have impaired peripheral heat loss (45) but, if so, what mechanisms are involved. However, a further question is raised as to whether such experiments should be performed in the laboratory or the home environment.

Sleep latency. The main selection criterion for the two groups in the present experiment was that the insomniacs typically took longer to fall asleep in their home environment. However, this feature was not demonstrated in the laboratory. The SOL circadian rhythm, as well as the time of peak SOL, was very similar between the two groups, with no significant group differences found. Moreover, analysis of the first several trials of the experiment also showed no significant differences.

One explanation for the lack of significant differences in mean SOL could be due to a “first night effect” for good sleepers (2, 43), and a “reverse night effect” for the insomniacs (2, 19). For good sleepers, it has been found that their SOLs in the laboratory can be longer than in their home environment. Free from the stimuli that induce sleepiness at home, good sleepers could have taken longer to initiate sleep in the laboratory. Conversely in the laboratory, insomniacs were free from stimuli in their own bedrooms that induce arousal and hence may have fallen asleep quicker than at home. Furthermore, it has been reported that prior adaptation to a laboratory environment can also reduce the length of subsequent sleep latencies (10). Thus the lack of difference in SOLs between insomniacs and good sleepers in the present experiment may be because the insomniacs had prior participation in another experiment in the same laboratory (9) or because of the first night effect.

It appears then that familiarity with an environment that does not contain stimuli that usually evoke a conditioned SNS response could produce decreased sleep latencies, as well as $T_{fing}$ increases free from anxiety-related vasoconstriction. Sleep latency and $T_{fing}$ changes may therefore be more adaptable in individuals experiencing insomnia. In contrast, $T_c$ cannot be as easily amenable to such changes, providing evidence for the chronicity of the psychophysiological hyperarousal of insomnia in subjects in the present study. Therefore, it should be emphasized that future researchers may want to consider studying insomniacs in their own home environment to obtain a more typical understanding of psychophysiological processes in this group.

In summary, contrary to expectations, insomniacs showed greater $T_{fing}$ increases than good sleepers during sleep latency trials, despite that the insomnia group reported greater sleep anticipatory anxiety. The only circadian difference between the two groups was that insomniacs had a higher $T_c$ mesor, indicating greater heat production than good sleepers. The greater heat production of insomniacs and the loss of differences in sleep latency made observations of any heat loss impairment difficult. More research is needed to further understand the relationship between acute distal skin temperature changes and sleep initiation in insomniacs in the conditions in which their insomnia is evident.

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