Do Chronic Primary Insomniacs have Impaired Heat Loss when Attempting Sleep?

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Abstract

For good sleepers, distal skin temperatures (e.g. hands and feet) have been shown to increase when attempting sleep. This process is said to reflect the body’s action to lose heat from the core via the periphery. However, little is known 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 attempting sleep between 11 chronic primary insomniacs (mean age = 40.0±13.3) and 8 good sleepers (mean age = 38.6±13.2) in a 26-hr 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 good sleepers (p=.001), even though there was no significant differences in baseline finger temperature (p=.25). These significant increases occurred despite insomniacs reporting significantly greater sleep anticipatory anxiety (p<.0008). Interestingly, the core body temperature mesor of insomniacs (37.0±0.2°C) was significantly higher than good sleepers (36.8±0.2°C, p=.03). Whether insomniacs could have impaired heat loss that is masked by elevated heat production is discussed.

Keywords: finger temperature, core body temperature, insomnia, sleep.
INTRODUCTION

Temperatures of distal skin regions (i.e. hands and feet) have been shown to increase prior to 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 to 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 to 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 recognised 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 prior to 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).

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

Participants

Twelve insomniacs (9 females, 3 males, mean age = 40.50 yrs, SD = 12.77) and eight good sleepers (5 females, 3 males, mean age = 38.63 yrs, SD = 13.18) 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 two weeks 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’s Syndrome, back pain, etc.) and they 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 interview by a sleep therapist. As 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 co-morbid 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 females and three males (mean age = 40.0 yrs, SD = 13.2). The mean (SD) length of chronicity of their insomnia was 11.8 (10.7) yrs.

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] < 20 min; [wake after sleep onset, WASO] < 20 min; [total sleep time, TST] = 7.49±0.9 hr; [sleep efficiency, SE] > 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 two weeks prior to 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 one week and alcohol for three days prior to the experiment.

Design

The experiment used a two-way mixed model design. The experiment consisted of a 26-hr laboratory modified constant routine (CR) session. The CR was modified by the inclusion of multiple sleep latency tests (MSLTs) (12) conducted half-hourly.

Procedure

Prior to the 26-hr modified CR, participants from both groups completed the Sleep Anticipatory Anxiety Questionnaire (SAAQ) (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 SAAQ was used.

Subjects arrived at the Flinders University Sleep Research Laboratory at approximately 1800 h, where they were fitted with EEG (Cz, Oz) and EOG electrodes. A rectal thermistor (YSI 400 Series Indwelling Thermistor Probe, Yellow Springs Instruments Co. Inc.) was self-inserted, and a skin
thermistor (YSI 400 Series Probe, 409B, time constant = 1.1 sec; Yellow Springs Instrument Co Inc, Ohio, USA) taped to the palmar surface of their right index finger. Fingertip temperature ($T_{\text{fing}}$) was used as the measure of distal skin temperature, as it has been found to be a sensitive psychophysiological measure of both anxiety (22, 23) and distal skin temperature changes prior to sleep onset (32). Furthermore, $T_{\text{fing}}$ 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 $T_{\text{fing}}$ for the present paper. Subjects were instructed to keep their right hand outside of the bedcovers at all times. The 26-hr CR began with the first sleep latency test (SLT) occurring at 1930 h.

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 2-hourly, and water provided when required. The 26-hr 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 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 electrophysiological and raw temperature data were continuously displayed and recorded using LabVIEW 5 software (National Instruments Corporation, Austin, Texas, USA). Rectal temperature data were averaged into 30-min bins, and $T_{\text{fing}}$ data averaged into 30-sec bins. Finger temperature readings
commenced prior to starting each sleep latency test. 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 awoken after three consecutive 30-sec 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-hr CR finished with the last SLT occurring at 2130 h on the second evening.

**Statistical Analyses**

Since $T_{fing}$ 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 sympathetic nervous system 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 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 as 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 ($T_c$) minimum was visually identified from their raw rectal temperature data using a 24-hr cosine plus 12-hr harmonic best-fit curve (insomnia average $R^2 = 0.75$; good average $R^2 = 0.88$) with the software program Kaleidagraph (Synergy Software, Reading, PA). This method also provided data for the amplitude and mesor for $T_c$. The mean (SD) time of $T_c$ minimum was calculated (insomniacs: 0500 h (1.6 hr); good sleepers: 0530 h (1.3 hr)). After all of the individual subjects’ $T_c$ minima were aligned to the group minimum time, individual baseline $T_{fing}$, $T_{fing}$ increase, and sleep latency (SOL) data were re-aligned based on their adjusted individual $T_c$. Due to this re-alignment, 26.5 hr of data were calculated and used for analysis.
To test for significant variation of baseline $T_{fing}$, $T_{fing}$ increase, SOL, and $T_c$ over time, between insomniacs and good sleepers, as well as any interaction effects, a series of non-linear mixed model regressions were performed controlling for covariates (i.e. gender). SOL showed a significant, positive skew, and baseline $T_{fing}$ a significant negative skew. SOL was log-transformed, and baseline $T_{fing}$ underwent a reflect and logarithmic transformation (44). Transformations of these variables resulted in normal distributions. For pre-laboratory measures, independent 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 standard deviation) and inferential statistics for age, BMI, and data from the sleep/wake diary, wrist activity monitor, and sleep questionnaires. Insomniacs and good sleepers did not significantly differ in age or body mass index. Insomniacs reported taking longer to fall asleep, a greater amount of time awake after sleep onset, less amount of total sleep, and less efficient sleep than good sleepers. These same differences were verified with the wrist actigraphy data. Furthermore, insomniacs rated themselves as being significantly more anxious while attempting sleep. There were also trends approaching significance for the insomniacs having higher levels of general anxiety and stress. No significant differences were found between the two groups for depression or daytime sleepiness.
Sleep Onset Latencies, Rate of Finger Temperature Increase, and Core Body Temperature

Despite the subjective and objective evidence of significant differences in sleep onset latency recorded in the home environment, these differences were not evident in the laboratory. No significant effects were found for ‘group’ (i.e. insomniac v. good sleeper), $F(1, 98.94)^1 = 0.55, p = .46$, nor the interaction (i.e. ‘group*time’), $F(53, 477.01) = 0.90, p = .68$. A significant effect for time was found, $F(53, 477.01) = 1.70, p = .002$, indicating a circadian rhythm for SOL (see Figure 1a). It was surprising that there was no significant difference in the SOLs between the two groups, especially in the early phase of the 26-hr constant routine (i.e. up to 2200 h) as this period would correspond to the time when the two groups differed in the home environment. Further analyses at each time point for the first several trials (e.g. 1930 h to 2200 h) showed that at no point were insomniacs’ SOLs significantly longer than good sleepers (all $t$-values <1.33, $p > .05$).

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, $F(1, 81.21) = 1.37, p = .25$ (Figure 1b). There was a

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1 The non-linear mixed model regression includes random variance components derived from the data, resulting in varying denominator degrees of freedom between analyses.
Figure 1. Mean (SEM) curves of a) SOL, b) baseline finger temperature ($T_{fing}$), c) finger temperature increase, and d) core body temperature ($T_c$) for insomniacs and good sleepers.

*Note.* Good sleepers’ SOL, baseline $T_{fing}$, $T_{fing}$ increase, and $T_c$ rhythms (light grey) are duplicated against the insomnia curves.
trend for a significant interaction effect, \( F(53, 465.86) = 1.36, p = .055 \), which may be explained by the good sleepers having lower mean finger temperatures in two periods over the 26-hr CR (i.e. 1930 h to 2130 h on the first day, and 1600 h to 1800 h on the second day; see Figure 1b). A significant main time effect was also found, \( F(53, 465.86) = 1.53, p = .01 \), indicating a circadian rhythm of baseline \( T_{\text{fing}} \).

Analysis of the 5-min \( T_{\text{fing}} \) increase data surprisingly showed a significant main group effect, \( F(1, 216.05) = 10.55, p = .001 \), however in the opposite direction to that predicted, with the mean (SD) \( T_{\text{fing}} \) increase 5 min after lights out greater for insomniacs (1.5±1.2°C) than good sleepers (1.2±1.2°C) (Figure 1c). No significant interaction effect, \( F(53, 464.68) = 0.60, p = .99 \), or main effect for time, \( F(53, 464.68) = 0.59, p = .99 \), were found. The mean \( T_{\text{fing}} \) changes from baseline to 5-min after lights out are shown in Figure 2.

![Figure 2](image)

Figure 2. Mean (SEM) finger temperature increase during sleep latency trials for insomniacs and good sleepers.
Interestingly when analysing $T_c$, a significant main effect for group was found, $F(1, 22.36) = 8.92$, $p=.007$. Independent t-tests on the $T_c$ mesor data demonstrated that insomniacs had significantly higher values ($37.0\pm0.2\, ^\circ C$) than good sleepers ($36.8\pm0.2\, ^\circ C$), $t(17) = 2.41$, $p=.03$ (see Table 2). No significant interaction effect was found, $F(53, 659.72) = 1.31$, $p=.07$, though this was approaching significance. As expected, there was a significant main effect for time, $F(53, 659.72) = 5.66$, $p<.0001$, demonstrating significant circadian variation (see Figure 1). No significant differences were found in the amplitude of $T_c$ between the insomniacs ($0.2\pm0.1$) and good sleepers ($0.21\pm0.1$), $t(17) = 0.28$, $p=.78$. No other significant differences between the circadian rhythms of insomniacs and good sleepers were found (see Table 2).

Inspection of Figure 1 clearly shows an offset of $T_c$ between the two groups at all time-points. However, baseline $T_{fing}$ appears similar. This suggests 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 (Figure 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 non-linear 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, $F(1, 83.45) = 0.72$, $p=.40$, though there was a trend for a significant interaction effect, $F(53, 450.10) = 1.35$, $p=.056$. A significant main effect for time was also found, $F(53, 450.10) = 1.94$, $p<.0001$, indicating circadian rhythmicity.

With regard to the circadian rhythms of SOL and body temperatures, inspection of Figure 1 demonstrates that SOL decreases in conjunction with a decline in $T_c$ (insomniacs: $r(54) = .53$, $p<.0001$; good sleepers: $r(54) = .70$, $p<.0001$), and an increase in baseline $T_{fing}$ (insomniacs: $r(54) = -.37$, $p=.004$; good sleepers: $r(54) = -.58$, $p<.0001$). Core temperature and $T_{fing}$ are also inversely correlated (insomniacs: $r(54) = -.69$, $p<.0001$; good sleepers: $r(54) = -.58$, $p<.0001$).
Figure 3. Plots of core temperature versus baseline finger temperature. Data are from the group mean data across the 26-hr CR.
DISCUSSION

Using a similar constant routine protocol to an earlier study (32), both insomniacs and good sleepers demonstrated consistent increases in finger temperature when attempting sleep. In fact, the magnitude and range of $T_{fing}$ increases for good sleepers in the present study are comparable to the previous study also using good sleepers (32). However, contrary to predictions, insomniacs had a greater increase of $T_{fing}$ than good sleepers. Therefore, the prediction that insomniacs would have lower, and attenuated acute $T_{fing}$ changes prior to sleep onset was not supported. This would suggest that the sleep anticipatory anxiety reported by insomniacs in their typical home environment had no depressive effect on acute $T_{fing}$ increases in the laboratory.

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

Acute Finger Temperature Changes during Sleep Latency Tests between Insomniacs and Good Sleepers.

Across the 26-hr constant routine, insomniacs demonstrated consistent $T_{fing}$ increases during SLTs. This does not support previous findings that insomniacs’ toe skin temperature change when attempting sleep sometimes shows no change at all (11). However, increases of $T_{fing}$ 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 arterio-venous 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.

Not only did insomniacs demonstrate $T_{fing}$ increases during SLTs, but these $T_{fing}$ changes were greater than good sleepers. It therefore appears that these insomniacs had a $T_{fing}$ 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 prior to the 26-hr 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 generalise 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 $T_{fing}$, as well as SOLs, is unclear. Future studies are needed not only to record sleep anticipatory anxiety in the laboratory, but also finger temperature changes during sleep attempts in the home environment that show extended latencies to sleep onset.

Core Body Temperature

The one striking difference from the averaged core body temperature ($T_c$) curves was that the insomniacs had a higher $T_c$ mesor. This concurs with previous research (1, 36). Interestingly, the mean $T_c$ of sleep-onset insomniacs has been reported as equivalent to that of good sleepers (38), although their $T_c$ 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 $T_c$ than good sleepers (35), suggesting the higher the $T_c$, the more interruptions of sleep during the night.

Core body temperature 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). Core body temperature becomes higher when heat production exceeds heat loss (4, 5, 25). A higher $T_c$ could therefore be a product of increased heat production and/or decreased heat loss.
Elevated Core Body Temperature 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 insomnia is related to excessive heat production. Since the higher $T_c$ 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 $T_c$ curve for insomniacs was offset approximately 0.2-0.3°C above the good sleeper’s $T_c$ 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, $T_c$ would be elevated if heat production exceeded heat loss. As the heat loss of insomniacs (as measured by the $T_c$-$T_{fing}$ gradient) was equivalent to the good sleepers, it could be the greater heat production of insomniacs that contributed to the elevated core body temperature. It may be that the set-point of $T_c$ is effectively elevated for insomniacs in which case the elevated $T_c$ 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 in order to down-regulate $T_c$.

When attempting sleep, insomniacs clearly demonstrated a greater increase in $T_{fing}$. This could be explained if in fact it were the case that the $T_c$ set-point is not elevated for insomniacs. In response to the sleep attempt, the thermoregulatory system may see this as an ‘opportunity’ to lose heat in order to down-regulate $T_c$ 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 if insomniacs not only have impaired peripheral heat loss (45), but also what mechanisms
are involved. However, a further question is raised 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 due to either that the insomniacs had prior participation in another experiment in the same laboratory (9), or ‘first night effects’.

It appears then that familiarity with an environment that does not contain stimuli that usually evokes a conditioned sympathetic nervous system response, could produce decreased sleep latencies, as well as $T_{\text{fin}}$ increases free from anxiety-related vasoconstriction. Sleep latency and $T_{\text{fin}}$ 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 research may want to
consider studying insomniacs in their own home environment in order to obtain a more typical understanding of psychophysiological processes in this group.

**Summary**

Contrary to expectations, insomniacs showed greater $T_{\text{fing}}$ 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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REFERENCES


17. Hales JRS. Skin arteriovenous anastomoses, their control and role in thermoregulation. In: 
Cardiovascular Shunts, Alfred Benzon Symposium 2, edited by Johansen K, and Burggren WW. 


22. Johnsen EL, and Lutgendorf SK. Contributions of imagery ability to stress and relaxation. Ann 


24. Kleitman R, Ramsaroop A, and Engelmann T. Variations in skin temperatures of the feet and 

25. Kräuchi K. How is the circadian rhythm of core body temperature regulated? Clin Auton Res 12: 
147-149, 2002.

changes begin after lights off and not after onset of sleep stage 2. Sleep (Suppl.) 24: A165, 2001.

27. Kräuchi K, Cajochen C, Werth E, and Wirz-Justice A. Warm feet promote the rapid onset of 


29. Kräuchi K, and Wirz-Justice A. Circadian rhythm of heat production, heart rate, and skin and core 
temperature under masking conditions in men. Am J Physiol Regulatory Integrative Comp Physiol 267: 


31. Kubo H, Yanase, T, and Akagi H. Sleep stage and skin temperature regulation during night-sleep in 

32. Lack L, and Gradisar M. Acute finger temperature changes preceding sleep onsets over a 45-h 


Figure 1. Mean (SEM) curves of a) SOL, b) baseline finger temperature ($T_{\text{finger}}$), c) finger temperature increase, and d) core body temperature ($T_c$) for insomniacs and good sleepers.

*Note.* Good sleepers’ SOL, baseline $T_{\text{finger}}$, $T_{\text{finger}}$ increase, and $T_c$ rhythms (light grey) are duplicated against the insomnia curves.

Figure 2. Mean (SEM) finger temperature increase during sleep latency trials for insomniacs and good sleepers.

Figure 3. Plots of core temperature versus baseline finger temperature. Data are from the group mean data across the 26-hr CR.
Table 1.
Descriptive [mean (SD)] and Inferential Statistics of Age, BMI and Data from Sleep/Wake Diary (swd), Wrist Activity Monitor (act), 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 (yrs)</td>
<td>40.0 (13.3)</td>
<td>38.6 (13.2)</td>
<td>0.22, (.83)</td>
</tr>
<tr>
<td>BMI</td>
<td>24.4 (3.0)</td>
<td>23.2 (2.5)</td>
<td>0.91, (.37)</td>
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<td>*swd SOL (mins)</td>
<td>73.6 (61.4)</td>
<td>12.0 (6.6)</td>
<td>2.80, (.01)</td>
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<tr>
<td>*swd WASO (min)</td>
<td>83.8 (82.4)</td>
<td>16.3 (10.2)</td>
<td>2.29, (.04)</td>
</tr>
<tr>
<td>*swd TST (hr)</td>
<td>5.6 (1.7)</td>
<td>7.5 (0.9)</td>
<td>2.94, (.01)</td>
</tr>
<tr>
<td>*swd SE (%)</td>
<td>67.4 (20.6)</td>
<td>93.2 (3.7)</td>
<td>3.46, (.003)</td>
</tr>
<tr>
<td>*act SOL (min)</td>
<td>36.7 (17.8)</td>
<td>14.2 (4.2)</td>
<td>3.48, (.003)</td>
</tr>
<tr>
<td>*act WASO (min)</td>
<td>76.5 (45.0)</td>
<td>11.6 (4.9)</td>
<td>4.04, (.001)</td>
</tr>
<tr>
<td>*act TST (hr)</td>
<td>6.0 (1.3)</td>
<td>7.3 (0.7)</td>
<td>2.64, (.02)</td>
</tr>
<tr>
<td>*act SE (%)</td>
<td>64.8 (20.2)</td>
<td>94.4 (1.8)</td>
<td>4.12, (.0009)</td>
</tr>
<tr>
<td>Depression</td>
<td>7.6 (8.6)</td>
<td>3.3 (4.0)</td>
<td>1.32, (.21)</td>
</tr>
<tr>
<td>Anxiety</td>
<td>5.6 (3.1)</td>
<td>2.5 (3.2)</td>
<td>2.10, (.05)</td>
</tr>
<tr>
<td>Stress</td>
<td>16.0 (10.7)</td>
<td>6.5 (7.8)</td>
<td>2.10, (.05)</td>
</tr>
<tr>
<td>*SAAQ</td>
<td>14.6 (3.9)</td>
<td>6.0 (5.0)</td>
<td>4.13, (.0008)</td>
</tr>
<tr>
<td>ESS</td>
<td>4.1 (2.0)</td>
<td>6.9 (3.6)</td>
<td>2.07, (.06)</td>
</tr>
</tbody>
</table>

* indicates significance at \( p < .05 \). Differences tested with independent t-tests. 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-sec epochs of movement.
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>$T_c$ mesor (°C)</td>
<td>37.0 (0.2)</td>
<td>36.8 (0.2)</td>
<td>2.41, (.03)</td>
</tr>
<tr>
<td>$T_c$ amplitude (°C)</td>
<td>0.2 (0.1)</td>
<td>0.2 (0.1)</td>
<td>0.28, (.78)</td>
</tr>
<tr>
<td>$T_{fing}$ mesor (°C)</td>
<td>33.1 (1.1)</td>
<td>32.2 (2.3)</td>
<td>1.07, (.30)</td>
</tr>
<tr>
<td>$T_{fing}$ amplitude (°C)</td>
<td>1.2 (0.8)</td>
<td>2.0 (1.7)</td>
<td>1.45, (.16)</td>
</tr>
<tr>
<td>SOL mesor</td>
<td>8.2 (5.6)</td>
<td>7.3 (3.6)</td>
<td>0.40, (.70)</td>
</tr>
<tr>
<td>$T_c$ minimum time</td>
<td>0500 h (1.6)</td>
<td>0530 h (1.3)</td>
<td>0.75, (.46)</td>
</tr>
<tr>
<td>Peak $T_{fing}$ time</td>
<td>0200 h (1.9)</td>
<td>0230 h (2.4)</td>
<td>0.37, (.72)</td>
</tr>
<tr>
<td>Peak SOL time</td>
<td>2100 h (1.2)</td>
<td>2100 h (1.3)</td>
<td>0.31, (.76)</td>
</tr>
</tbody>
</table>

*indicates significance at $p<.05$

Note. time for $T_c$ minimum, and Peak FT are for the 2nd day of the CR; peak SOL is for the 1st day of the CR. Times rounded to the nearest half-hour. All values are taken from the 24-hr cosine plus 12-hr harmonic-fitted curves of $T_c$, $T_{fing}$, and SOL.