Attenuated Thermoregulatory Response to Mild Thermal Challenge in Subjects With Sleep-Onset Insomnia

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Study Objectives: To determine if heat loss capacity of sleep onset insomniacs was different from that of healthy sleepers.

Design: Measure skin temperature responses following brief exposure to a warm peripheral thermal challenge (PTC).

Setting: Sleep research laboratory in South Australia.

Participants: Eight primary insomniacs with sleep onset insomnia according to DSM-IV-TR criteria (SOI; 5 male, 3 female; mean age±SEM=35.2±4.2 years) and ten healthy sleeping control subjects (HS; 7 male, 3 female; mean age=28.2±2.8 years).

Interventions: Two PTC conditions in counterbalanced order on non-consecutive days. During each condition, the subject’s non-dominant forearm and hand were immersed for 3 minutes in Warm (45°C) or Control water (i.e. same as the subject’s non-dominant index finger temperature just prior to immersion, range 30-35°C).

Measurements and Results: HS had a significantly higher maximum finger temperature response after immersion than SOI (P<0.05). Expressed relative to Control PTC temperatures, the Warm PTC caused a significant increase in mean finger temperature for HS of 4.1±0.8°C, compared with SOI of 0.9±0.4°C. A significant negative relationship was observed between maximum finger temperature response and self-reported sleep onset latencies (R=0.57, P<0.05). There were no main effects of sleep status (SOI vs. HS) or interactions by time, in skin temperatures measured at either the back of hands or feet.

Conclusions: SOI were observed to have significantly attenuated thermoregulatory responses to a mild positive thermal challenge, providing evidence that impaired heat loss capacity from the periphery is associated with sleep onset insomnia.

Keywords: Insomnia, sleep, thermoregulation, sleep initiation and maintenance disorders, body temperature regulation, skin temperature, arteriovenous anastomoses

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INTRODUCTION

INSOMNIA IS COMMON AND A SIGNIFICANT PUBLIC-HEALTH ISSUE, WITH ESTIMATES OF UP TO A THIRD OF ADULTS IN WESTERN POPULATIONS REPORTING chronic sleeping difficulties requiring treatment within any 12-month period.1-4 Insomnia is associated with significant adverse health, psychological, and social consequences. The typical features of chronic insomnia include excessive daytime sleepiness, yet longer sleep-onset latencies on the Multiple Sleep Latency Test, when compared with good sleepers; increased pain, stress, depression, and anxiety; decreased vigor; subjective overestimation of poor sleep; and increased core body temperature and other indicators of metabolic rate.5-7 Until recently, these latter symptoms were thought to demonstrate a tendency for chronic insomniacs to display physiologic hyperarousal,8 supporting the view that the secondary symptoms of insomnia are not due to poor sleep per se but may occur due to the hyperarousal in the central nervous system.9

Relationship Between Sleep and Circadian Body-Temperature Changes

In healthy adults, clear associations between changes in sleep propensity and body temperatures have been observed prior to sleep onset.9-11 Typically, an increase in peripheral temperature precedes a decrease in core body temperature and the onset of sleep, and it has been verified that the nocturnal decrease in core temperature is achieved by heat loss from peripheral skin rather than a reduction in metabolic heat production.12 Our group has previously reported that a significant increase in sleep propensity and a concomitant increase in heat loss and reduction in core body temperature also occurs immediately after ingestion in the afternoon of somnogenic agents, including melatonin and temazepam.13 Consistent with this finding, it has been shown that the degree of peripheral heat loss, proportional to cutaneous vasodilatation in the periphery, is also a significant predictor of melatonin-induced sleepiness when melatonin is administered in the evening.14 Together, these results support the suggestion that heat loss is a correlate of both natural and pharmacologically mediated sleep initiation in healthy adults.13-15-19

Our group has previously suggested that an impaired ability to lose heat from the periphery, preventing the normal nocturnal decline in core temperature, may underlie difficulty initiating sleep at night in people with sleep-onset insomnia (SOI).10,17,20 If this is the case, then an inability to increase peripheral temperature would be directly responsible for decreased sleep propensity, possibly mediated by sustained central nervous system hyperarousal. A seminal observation by Freedman and Sattler21 was that patients with SOI have significantly lower finger temperatures, from lights out through to the onset of stage 2 sleep, supporting a case for attenuated peripheral heat loss in the etiology of SOI.

The current study examined whether the heat-loss capacity of otherwise healthy subjects with sleep onset insomnia is significantly different from that of matched, healthy-sleeping control subjects. This was achieved using a mild peripheral thermal challenge (PTC), which we have previously used to distinguish be-
between an efficient and an attenuated capacity to lose heat from the periphery in young and older adult women, respectively.\textsuperscript{17} We hypothesized that the normal response of healthy sleepers to immersion of 1 arm in warm water—which is an increase in skin temperature on the contralateral limb to compensate for the systemic heat gain—would be significantly attenuated in subjects with SOI and, therefore, indicative of an attenuated thermoregulatory heat-loss capacity.

**METHODS**

**Subjects**

Eighteen individuals (6 women, 12 men) aged between 20 and 52 years (mean ± SEM = 32.1 ± 2.7 years) participated in the current study. Potential subjects were recruited through advertisements in newspapers and on community noticeboards seeking both insomniacs and healthy sleepers. Respondents were screened for medical and psychiatric illnesses and sleep disorders by completing a General Health Questionnaire, Beck Depression Inventory-II, Beck Anxiety Inventory, and 14-day sleep-wake diaries. According to their self-reported nocturnal sleep-onset latencies (SOL) over a 14-day period, subjects with SOI all had a mean SOL greater than 30 minutes and healthy sleepers all had a mean SOL less than 30 minutes. In addition to SOL, all subjects with SOI were required to have chronic difficulty initiating sleep, persisting for at least 6 months prior to recruitment and occurring 3 or more nights per week. During recruitment, we attempted to match healthy-sleeping controls as closely as possible to subjects with SOI for age, sex, and body mass index (BMI). Note that all subjects (by chance) were right-handed, so we did not need to account for any effects of handedness in this study.

Volunteers were selected as subjects if they were nonsmokers and consumed only moderate or lower amounts of caffeine (0-250 mg/day) and alcohol (0-6 drinks/week). All subjects reported subclinical scores for depression (Beck Depression Inventory-II < 14) and anxiety (Beck Anxiety Inventory < 8) and were within a healthy weight range (mean BMI for men = 27.1 ± 1.0 kg/m\(^2\) and women = 21.5 ± 1.4 kg/m\(^2\)). Other than chronic difficulty initiating in the SOI group, all subjects reported no current health problems and were unmedicated except for females taking oral contraceptives. None of the healthy-sleeping group reported a history of sleep problems, and no subjects had taken part in shift work or transmeridian travel in the prior 3 months. All subjects gave informed consent and were reimbursed $50 for their involvement. No subjects withdrew from the study after commencing the experimental protocol. Institutional ethical approvals for the protocol were granted by the relevant committees at The Queen Elizabeth Hospital, University of South Australia, and Adelaide University.

**Equipment**

Temperature recordings were made continuously throughout each session using a digital temperature system (Strawberry Tree, Sunnyvale, CA). The system consists of a personal computer with Strawberry Tree Data Acquisition Boards, Terminal Panels, and Workbench for Windows software for real-time recording and display of multiple temperature channels. Hand and foot skin temperatures were measured using Steri-Probe® skin-surface thermistors provided by Cincinnati Sub-Zero Products Inc. (CSZ, Cincinnati, OH). Rectal core temperatures were not recorded because we have shown that the PTC (see Experimental Protocol below) does not affect core temperatures in either healthy young adults or elderly subjects with attenuated thermoregulatory responses.\textsuperscript{17}

Additional hand-temperature recordings were made using a Meditherm Med2000 Digital Infrared Thermal Imaging (DITI) camera (MMS, Queensland, Australia). The DITI camera takes false-color, 244 × 193-pixel, images at user-selectable intervals, in which each pixel is a spot measure of temperature. The color of each pixel is directly proportional to the thermal energy emitted at that site. Images were recorded on to a computer hard disk and subsequently analyzed using WinTES thermal evaluation software (Compix, Lake Oswego, OR). DITI images were displayed on a computer screen and analyzed by selecting a region of interest on the back of the hand defined by anatomic landmarks. In this study, the region of interest was the largest possible rectangle within an area defined by the knuckles, the side of the hand and the narrowest part of the wrist. The software calculates a mean temperature over the region of interest that was then entered into a database. Three trained and experienced operators, blinded to the status of subjects (e.g., insomniac or healthy control), conducted all the DITI image analyses. This method for obtaining temperature measures from DITI images has been previously published and validated against thermistor-based thermometry of skin temperature in our laboratory.\textsuperscript{22}

**Experimental Protocol**

Subjects reported to the sleep laboratory on 2 separate occasions at least 3 days apart, wearing short-sleeved shirts or T-shirts and jeans or pants (no shorts). From 10:30 AM on each occasion, subjects were seated in a comfortable lounge chair in an individual room and then remained seated and still until 1:00 PM, the same day. Before 10:45 AM, subjects were fitted with thermistors, while seated, to record the skin temperature at the palmar tip of the right index finger and the instep of the right foot. At all times, room temperature was maintained at 21°C ± 1°C, at the lower end of the thermoneutral range, to facilitate heat loss, and ambient room light was also kept below 50 lux.

Experience in our laboratory with similar protocols\textsuperscript{17} has shown that a 30-minute period of quiet sitting, such as that allowed in this study from 10:30-11:00 AM, is more than sufficient to allow all subjects’ skin and core temperatures to equilibrate to thermoneutral ambient conditions. From 11:00 AM in each session, skin-temperature measurements were recorded at 60-second intervals until the end of the session at 1:00 PM. In addition to thermistors, skin temperatures were also recorded every 60 seconds using the Meditherm DITI camera, sited 30 inches directly above the subject’s right hand.

Subjects were assigned to a different PTC condition on each of the nonconsecutive visits by randomizing the condition for the first visit and counterbalancing so they completed the remaining condition on the second visit. The 2 conditions were:

1. **Warm PTC**—At 1200 noon, the subject’s left forearm and hand (up to the elbow) were immersed for 3 minutes in water at 45°C.
2. **Control PTC**—At 1200 noon, the subject’s left forearm and hand were immersed for 3 minutes in water that was the same temperature (± 1°C) as recorded at their left index finger 3°C. **SLEEP, Vol. 29, No. 9, 2006**
minutes prior to commencement of the PTC (i.e., 11:57 AM). The range of Control water temperatures across all subjects was subsequently found to be between 30°C and 35°C.

In both cases, an assistant began preparing the water for Warm or Control PTC at 11:57 AM by mixing approximately 15 liters of hot and cold water in a plastic container and verifying the temperature using an alcohol-filled thermometer marked with 1°C increments (Fowler Vacola, Australia). The container was then brought to the subject for immersion by 12:00 noon.

At all times during each 2-hour seated period, subjects watched television and were instructed to keep as still as possible, especially their hands and feet, so that unnecessary movement would not influence temperature measures. To minimize confounding by external factors before each session, subjects were instructed to wake up at their habitual time reported in sleep diaries and engage in regular activities on the morning that the study was conducted. Subjects were also asked to abstain from any caffeine, alcohol, or any other drugs for the 24 hours prior to attendance at each session. All subjects reported compliance with these instructions, but they were not otherwise verified.

Data Analyses

To decrease interindividual variability in temperature data, skin temperatures for each subject were first expressed relative to the temperature recorded at 12:00 noon (Time = 0), and then a Baseline temperature for each subject in each condition was established by averaging across the data from -60 to -1 minutes. Statistical analyses were then performed on relative data (Control condition subtracted from the Warm condition) for all subjects at Baseline and at the following time points relative to the time of immersion: 0, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 15, 30, 45, and 60 minutes. Each relative skin-temperature measure thus indicates the overall effect of the PTC; that is, the temperature change recorded on the right (dominant) side due solely to a 3-minute immersion in warm water of the left (nondominant) hand and forearm. Using SuperANOVA software (Abacus Concepts, Berkeley, CA), a series of repeated-measures analysis of variance tests were conducted on the temperature data to test for main effects of sleep status (i.e., Group), Time, and any Interactions. Planned contrasts on the within-subjects factor (Time) were then conducted for each group in which a significant main effect or interaction had been observed to compare means at subsequent time points with that at the start of immersion (i.e., Time=0). Significance was set at \( \alpha = .05 \) for all tests. Data are presented hereafter as mean ± SEM.

RESULTS

Independent samples t-tests were conducted to determine whether there were significant differences in variables between healthy-sleeping and SOI groups. The variables analyzed were age, BMI, anxiety score, depression score, and self-reported SOL from screening questionnaires. As summarized in Table 1, only SOL showed any significant difference between groups, with the SOI group taking an average of 42.3 minutes longer to fall asleep each night than the healthy-sleeping control group. Observation of a significant difference in group SOL here verified that this criterion was able to select between groups.

-Finger Temperature-

Repeated-measures analysis of variance of finger-temperature data revealed a significant main effect of sleep status (F value = 5.80, p < .05), with significantly higher mean relative finger temperature across the whole experimental session in healthy sleepers (1.51°C ± 0.18°C) compared with that of subjects with SOI (0.02°C ± 0.09°C). In addition, there was a main effect for Time (F = 2.74, p < .001) averaged across both healthy sleepers and subjects with SOI. Planned contrasts showed that the effect of warm immersion across both groups was to significantly elevate mean relative finger skin temperature above that at Time 0, from 5 to 30 minutes after immersion, inclusive (all F values > 4.14, p < .05).

Most importantly, there was a significant interaction between Sleep Status and Time (F = 1.81, p < .05). As shown in Figure 1, the healthy-sleeping group had higher finger temperature after immersion than did the subjects with SOI. Contrasts revealed that mean relative finger temperatures for healthy sleepers were significantly higher than at Time 0 from 5 to 30 minutes after immersion, inclusive (5 min F = 8.45, p < .05; 6 min F = 9.51, p < .05; 7 min F = 12.27, p < .01; 8 min F = 12.46, p < .01; 9 min F = 11.23, p < .01; 10 min F = 9.02, p < .05; 15 min F = 7.47, p < .05; 30 min F = 6.00, p < .05). Mean relative finger temperatures for subjects with SOI did not differ significantly from Time 0 at any time during the hour subsequent to immersion (all F values < 1.20, p > .50).

The maximum increase in right-finger temperature in the 60 minutes after commencement of the PTC was calculated and is represented by Figure 2. An unpaired t-test revealed that the mean maximum finger-temperature response was significantly different between groups (t = 3.20, p < .01). The maximum finger-temperature response for healthy sleepers was 4.09°C ± 0.81°C, compared with only 0.91°C ± 0.44°C for subjects with SOI.

The time at which maximum finger temperature response occurred was also compared between groups using an unpaired t-test, but no significant difference was observed (t = 0.50, p = .62). The mean time after start of the PTC (i.e., Time = 0) at which the maximum finger temperature occurred was 30.2 ± 7.5 minutes for healthy sleepers, and 24.3 ± 9.3 minutes for SOI.

A series of simple linear regressions were then performed, to measure the strength of any relationships between age, body mass

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Table 1—Baseline Characteristics of Healthy-Sleeping Subjects and Subjects with Sleep-Onset Insomnia

<table>
<thead>
<tr>
<th>Healthy sleepers (n = 10)</th>
<th>Subjects with SOI (n = 8)</th>
<th>t</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>28.2 ± 2.8</td>
<td>35.2 ± 4.2</td>
<td>1.43</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>24.4 ± 1.5</td>
<td>26.3 ± 1.4</td>
<td>0.94</td>
</tr>
<tr>
<td>BAI</td>
<td>6.4 ± 1.1</td>
<td>6.4 ± 2.6</td>
<td>0.01</td>
</tr>
<tr>
<td>BDI-II</td>
<td>11.5 ± 2.2</td>
<td>9.4 ± 3.2</td>
<td>0.56</td>
</tr>
<tr>
<td>SOL, min</td>
<td>16.1 ± 1.3</td>
<td>58.4 ± 7.5</td>
<td>6.22</td>
</tr>
</tbody>
</table>

Data are were taken from subject's 14-day sleep-wake diaries and other screening questionnaires and are presented as mean ± SEM. SOI refers to sleep-onset insomnia. BMI, body mass index, BAI, Beck Anxiety Inventory; BDI, Beck Depression Inventory; SOL, self-reported sleep-onset latency.
index, self-reported sleep onset latency and maximum finger temperature response. No significant correlations were observed between age and body mass index ($R = 0.36/R^2 = 0.13$, $p = .15$), age and SOL ($R = 0.23/R^2 = 0.05$, $p = .36$), age and maximum finger temperature response ($R = -0.23/R^2 = 0.05$, $p = .37$), BMI and SOL ($R = 0.15/R^2 = 0.02$, $p = .56$), or BMI and maximum finger temperature response ($R = -0.19/R^2 = 0.04$, $p = .44$). There was however a significant negative relationship between maximum finger temperature response and SOL (see Figure 3). The correlation was moderately strong between these factors ($R = -0.57/R^2 = 0.32$, $p < .05$), with a correlation equation of $y = 4.67-0.06x$. Correlation coefficients for each group separately were not significant (healthy sleepers: $R = -0.24/R^2 = 0.06$, $p = .51$ and SOL: $R = -0.37/R^2 = 0.14$, $p = .37$).

**Hand Temperature**

Analyses of digital infrared thermal images of the back of the right hand showed no significant main effects for sleep status ($F = 1.33$, $p = .27$). Mean relative hand temperatures were $0.40 \pm 0.08^\circ C$ (healthy sleepers) and $0.16 \pm 0.05^\circ C$ (subjects with SOI). There was however, a main effect of Time ($F = 2.65$, $p < .005$), with planned contrasts showing that mean relative hand temperature (across both subject groups) was elevated above that of Baseline between 7 and 60 minutes after immersion, inclusive (all F-values $>4.16$, $p < .05$). However, there was no significant interaction between sleep status and time ($F = 0.74$, $p = .74$), indicating that there were no differences between the healthy-sleeping and SOI groups in hand temperature after immersion (see Figure 4).

**Foot Temperature**

Skin-temperature measures at the instep of the right foot showed no significant main effects for Sleep Status ($F = 0.37$, $p = .55$). The mean relative foot temperature of the healthy-sleeping group ($0.27 \pm 0.07^\circ C$) was thus not different than that of the SOI group ($0.16 \pm 0.04^\circ C$). No main effect was found for Time ($F = 1.17$, $p = .30$), nor was there any interaction between Sleep Status and Time ($F = 0.36$, $p = .99$). Inspection of Figure 5 shows the time-series curves for foot temperature in both healthy-sleeping and SOI groups.

**DISCUSSION**

The current study investigated whether chronic difficulty initiating sleep, in otherwise-healthy subjects with SOI, might be associated with a diminished heat-loss capacity from the periphery. Heat-loss capacity was investigated using a mild PTC—specifically, immersing the left hand and forearm in warm water for 3 minutes and then measuring the change in temperature at the contralateral limb. Heat-loss capacity was taken as the finger skin temperature responses in the 60 minutes following immersion in
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Figure 4—Time-series data for skin temperature of the dorsum surface of the right hand, expressed relative to the start of immersion of the left hand and forearm (Time = 0), and calculated by subtracting data for the Control condition from the Warm condition. The 2 curves are mean relative skin temperatures for healthy sleepers (filled circles) and subjects with sleep-onset insomnia (unfilled squares), expressed as group means ± SEM.

Figure 5—Time-series data for skin temperature of the instep of the right foot, expressed relative to the start of immersion of the left hand and forearm (Time = 0), and calculated by subtracting data for the Control condition from the Warm condition. The 2 curves are mean relative skin temperatures for healthy sleepers (filled circles) and subjects with sleep-onset insomnia (unfilled squares), expressed as group means ± SEM.

Warm (45°C) water, as compared with a control immersion (30°C-35°C). When compared with the healthy-sleeping group, there was a significant attenuation of the finger temperature response in subjects with SOI over the 60 minutes following the PTC. Whereas the maximum rise in finger skin temperature following the warm PTC was approximately 4.1°C in healthy sleepers, the response of finger temperature in subjects with SOI was only 0.9°C under the same conditions. Thus, this study provides direct evidence supporting the hypothesis that heat-loss capacity, in response to a mild positive thermal load, is attenuated in subjects with chronic SOI.

We were also anticipating significant increases in skin temperature measured at the back of the hand contralateral to the immersed limb, albeit attenuated to that expected in finger skin temperature. This is because the palmar surface of the hand contains a denser concentration of arteriovenous anastomoses, which are the blood vessels primarily involved in regulating heat loss from distal skin sites to the environment. Therefore, the skin at the back of the hand was not expected to be as actively involved in the homeostatic response to a positive thermal load in the PTC, and we observed no significant differences between the groups, although hand skin temperatures did tend to be higher in the healthy-sleeping group.

Foot skin temperature was measured to determine whether both hands and feet on the contralateral side would contribute to the homeostatic heat loss following application of a positive thermal load. However, as expected from our previous test of the PTC, only skin sites on the contralateral limb, specifically the palmar surface of the fingers, displayed significant temperature increases. We assume, but have not previously tested the hypothesis, that immersion of 1 foot in warm water would cause a change in temperature at the other foot but not the hand on the contralateral side. In any case, studies in the future should aim to investigate more thoroughly the thermoregulatory inefficiency that we have observed here in subjects with SOI. For example, it is not yet known whether the current group differences in distal heat-loss capacity are also present at other times of day, especially around the time of habitual sleep onset. Also, the mechanism by which the reduced heat-loss capacity is manifest deserves greater scrutiny, whether it occurs by impaired autonomic control of skin blood flow, a reduced efficiency of blood flow through arteriovenous anastomoses and other blood vessels, or other means. Once the underlying mechanism has been identified, we will be able to identify and test new treatments for SOI that directly affect thermoregulatory processes and thereby manipulate sleep propensity.

There were some limitations in the current study, which may have influenced the results. Firstly, there was a lack of blinding of subjects to their condition order after their first visit, as everyone had been informed that they would participate in both conditions. This was unlikely to be a significant issue, however, due to the randomized and counterbalanced order of the conditions across all subjects. Secondly, despite an attempt to match subjects with SOI with healthy-sleeping controls for age and BMI, these were both slightly but not significantly higher in the SOI group. Insomniacs were on average 7 years older and had a BMI that was 1.9 kg/m² higher than those measures in the control group. However, age was also not likely to be a significant factor in the response to a warm thermal challenge, as our previous study reported no significant differences in contralateral hand temperature following a warm thermal challenge between 6 younger and 10 older women with a mean age difference of 42.2 years. The average maximum temperature responses at the contralateral hand in this study were 0.68°C ± 0.21°C and 0.51°C ± 0.15°C for younger and older healthy-sleeping women, respectively. Furthermore, a recent review of aging and temperature regulation found that, despite evidence of reduced skin blood flow, reduced cardiac outputs, and smaller redistributions of blood flow from the splanchnic and renal circulations in response to heat stress, there was no systematic inability to thermoregulate with advanced age. It is also possible that there are significant effects of BMI or sex on hand-temperature response to a warm thermal challenge, but these are yet to be investigated.

One possible limitation in our study that may explain group differences in finger temperature response to a thermal challenge was that compliance with prestudy instructions was not checked objectively. It remains a possibility, therefore, that intake of caffeine and/or alcohol against instructions may have influenced our results. Finally, it may be possible that if the ambient temperature were higher, about 25°C, peripheral skin-temperature responses
after the PTC would have been higher. The ambient temperature of 21°C, plus the fact that the subjects were bare foot and wearing short sleeves, may have influenced the results because the subjects were colder. If so, subjects may have retained body heat rather than lost it after the warm immersion. Nevertheless, this would act to reduce the difference between the groups and, thus, may have underestimated the effect.

In trying to understand the cause of sleep problems, many previous studies have found that insomniacs do display symptoms consistent with elevated measures of autonomic arousal during the night. The seminal study of Monroe for example, found that poor sleepers, when compared with healthy sleepers, had increased measures of arousal, including rectal temperature, vasoconstriction, skin conductance, and body movement. This study continues to be cited widely as evidence that insomniacs are hyperaroused during the night. However, Varkevisser and colleagues’ recent negative findings of systematic differences in measures of arousal between insomniacs and controls, which novelly employed a 24-hour constant routine protocol, suggests that previous findings of hyperarousal may apply only to sleep per se.

In the present study, without direct or indirect measures of autonomic activity, it is not feasible to infer from changes in skin temperature alone whether the observed finger-temperature difference between groups relates to, say, increased sympathetic or decreased parasympathetic control of skin blood flow. Nevertheless, we hope that future studies will achieve a more-complete understanding of insomnia, especially the relationship between heat loss from distal skin sites and the regulation of sleep propensity, by taking these factors and limitations discussed above into consideration.

In summary, immersion of 1 hand or forearm for 3 minutes in warm water (i.e., PTC), compared with a control immersion, caused a significant increase in finger skin temperature on the contralateral limb in healthy sleepers. However, the finger skin-temperature response to the PTC in subjects with SOI did not change significantly and was effectively the same following both warm and control immersions. In addition, a significant negative relationship was observed between maximum finger-temperature response following the PTC and self-reported SOL across all subjects. This study used a mild positive thermal challenge between finger-temperature response and SOL may be shown to hold over a larger range of skin temperatures. In any case, this study provides direct evidence in support of the hypothesis that attenuated heat-loss capacity is associated with SOI. The decreased thermal response in insomniacs, if present at night when attempting to fall asleep, may interrupt the normal nocturnal cascade of heat changes, including loss of heat from the periphery and decline in core temperature previously observed to occur in association with normal sleep onset. These results may provide a foundation for the development of better treatments that address the physiologic etiology of poor sleep.

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