BRIEF REPORT

Fifty-Three Hours of Total Sleep Deprivation Has No Effect on Rewarming From Cold Air Exposure

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Objective.—Sleep deprivation and cold air exposure are both experienced in occupational and military settings but the combined effects of these 2 stressors is unknown. The purpose of this study was to determine the effects of 53 hours of total sleep deprivation on thermoregulation during the rewarming phase (25°C air) after acute cold air exposure (10°C air).

Methods.—Eight young men underwent 2 trials in which they either received 7 hours of sleep at night or were totally sleep deprived. On 3 consecutive mornings, the subjects underwent 2 hours of cold air exposure followed by 2 hours of rewarming. Rectal temperature, mean skin temperature, oxygen consumption, and thermal sensation were measured.

Results.—Rewarming from acute cold air exposure caused a decline in rectal temperature ($\sim 0.5^{\circ}\text{C}$) each day but this was not different between subjects who were totally sleep deprived and subjects who received 7 hours of sleep at night. During this same period, mean skin temperature increased (from $\sim 22^{\circ}\text{C}$ to 27°C), oxygen consumption decreased (from ~ 7 to $4 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$), and the participants felt warmer.

Conclusions.—Under the conditions of the present study, sleep-deprived persons are not at a greater risk for a decline in rectal temperature (ie, a hypothermic afterdrop) during rewarming from cold air.

Key words: body temperature regulation, humans, hypothermia, thermal sensation

Introduction

Sleep deprivation is often experienced during military operations, transmeridian travel, and space exploration. Even without exposure to hot or cold temperatures, body core temperature and heat dissipation mechanisms follow a circadian rhythm.^{1,2} Previous research has demonstrated inconsistent results, including increased,³ decreased,^{4,5} and unchanged⁶ core temperature after sleep deprivation as compared with normal sleep. These contradictory results are likely attributable to the duration of sleep deprivation as well as the time (morning or afternoon) and the measurement location (tympanic, oral, rectal, esophageal).

Cold ambient conditions also pose a significant threat to thermoregulation. Currently, little information exists regarding the combined stressor of sleep deprivation and cold air exposure. Previous research has shown that 33 hours of sleep deprivation does not impact rectal temperature during 3 hours of acute cold air exposure^{7,8} but a longer period of sleep deprivation or multiple cold exposures may have a greater effect. Also, the recovery (rewarming) from cold exposure was not assessed in these cited studies. After removal from a cold environment, the “afterdrop phenomenon” occurs through conductive (core to skin heat transfer) and convective (circulatory adjustment) pathways. Much of the work on this topic has been conducted using a water immersion model (ie, accidental hypothermia),^{9,10} and it is uncertain whether rewarming from cold air exposure would also result in an afterdrop.¹¹ Given that many outdoor recreational activ-

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ities (eg, mountain climbing, hiking, and fishing) may involve cold air exposure, we chose a cold air stressor for the current experiments. If sleep deprivation affects any of the compensatory responses to cold (ie, hypothalamic setpoint, skeletal muscle shivering, skin temperature, decision making),¹² it is possible that a person's ability to thermoregulate during the recovery (rewarming) period might be altered.

Rodent models suggest that excessive heat loss and elevated thermoregulatory setpoint are both prominent features of sleep deprivation.^{13,14} As these 2 concepts would shift core temperature in opposite directions, it is unclear how a person may respond to the added stressor of cold exposure. Accordingly, we sought to determine the effects of total sleep deprivation and repeated cold air exposure on thermoregulation in healthy men. The current investigation describes the following: 1) the effect of repeated cold exposures on the afterdrop phenomenon; 2) the effect of 27 hours of sleep deprivation on the afterdrop phenomenon; and 3) the effect of 51 hours of sleep deprivation on the afterdrop phenomenon. We hypothesized that cold exposure on multiple days (when normal sleep was allowed) would have no effect on rewarming from cold exposure. We also hypothesized that thermoregulation during rewarming would be impaired as sleep loss progressed (ie, greater decline in rectal temperature and lower metabolic rate).

Methods

SUBJECTS

This study protocol was approved in advance by the Kent State University Institutional Review Board. Each subject provided written informed consent before participating. Eight healthy Caucasian men (aged 23 ± 2 years) volunteered to participate for this study, which was part of a larger protocol in our laboratory.^{15,16} Experiments were conducted between the months of April and June 2008. Subjects were 179 ± 12 cm tall and had a body mass of 80 ± 10 kg. They also had peak aerobic fitness (cycle ergometer) of 44 ± 8 ml·kg⁻¹·min⁻¹ and body fatness (7-site skinfold) of $12\% \pm 6\%$. Women were not included owing to documented gender differences in thermoregulation.¹⁷ All participants were nonsmokers, not currently taking any medications that would affect

Figure 1. Experimental timeline for both sleep-deprived trial (left panel) and normal sleep trial (right panel). Acute cold exposure always occurred between 0600 hours and 0800 hours, followed by rewarming from 0800 to 1000 hours. Sedentary activities were permitted for the remainder of the trials.

thermoregulation or metabolism, and reported no sleep disturbances.

DESIGN

This experiment employed a repeated measures, cross-over design. As shown in Figure 1, bouts of acute cold air exposure (ACE [10°C]) and rewarming (RW [25°C]) were experienced on 3 consecutive mornings under both sleep deprived (SDEP) and control/normal sleep (CON) conditions. The order of treatment (SDEP, CON) was counterbalanced and separated by at least 7 days. In total, each of the 8 participants underwent a total of 6 ACE and RW sessions, 3 during SDEP and 3 during CON.

EXPERIMENTAL PROTOCOL

On the first visit to the laboratory, the protocol was explained, a health history was obtained, and informed consent was obtained. Height, weight, body fat, and peak aerobic fitness were determined following standard procedures for our laboratory.¹⁸ Participants returned twice within the next 1 to 2 weeks to undergo an initial overnight sleep in the laboratory followed by 53 total hours of either SDEP or CON, in random order. Owing to the duration of the study and personnel required, subjects were always studied in pairs (ie, 2 subjects underwent the same protocols at the same time).

Participants were asked to refrain from alcohol, caffeine, and exercise for 24 hours before enrolling in the SDEP and CON trials. Subjects obtained approximately 7 hours of sleep in the laboratory per night during the CON trial and were totally sleep deprived during the SDEP trial (Figure 1). Cold air exposures (10°C) occurred from 0600 to 0800 hours each morning, followed by passive RW in thermoneutral air (25°C) from 0800 to 1000 hours. Air temperature in the room was changed by turning a dial on the outside of the chamber. Subjects remained seated from 0600 to 1000 hours. To ensure the subjects remained awake for the duration of the 53 hours during the SDEP trial, they were supervised in the Exercise and Environmental Physiology Laboratory at all times by an investigator who was not sleep deprived. The subjects were allowed to do sedentary activities (eg, watch movies, read, work on homework, play cards) when not undergoing the ACE and RW trials. During the SDEP trial, participants were allowed approximately 3 extra 10-minute walks during the night to alleviate boredom. During the CON trial, participants were asked each morning to document the hours slept the night before. To monitor physical activity, an Actigraph monitor (Model GT1M, Actigraph LLC, Pensacola, FL) was worn.

During both CON and SDEP trials, subjects were supplied a standardized 2500 kcal per day diet that consisted of approximately 55% carbohydrates, 30% fat, and 15% protein. Each subject consumed a minibagel at 0500 hours each morning, before ACE. Additional meals included breakfast at 1000 hours, lunch at 1400 hours, and dinner at 1900 hours. Snacks (juice and minibagel) were served at 1630 hours and 2115 hours.

INSTRUMENTATION

All trials occurred in a temperature-controlled environmental chamber (Western Environmental Chamber, Napa, CA). During the experiment (ie, 0500 to 1000 hours), subject wore only shorts, socks, and gloves. Rectal temperature (T_{re}) was measured by a thermistor (ER400-12, Respiratory Diagnostic Products, Irvine, CA) inserted 13 cm into the rectum. Mean skin temperature (T_{sk}) was measured by thermistors (Model 409B, Yellow Springs Instruments, Yellow Springs, OH) at the following sites: chest, tricep, thigh, calf, and forearm, as previously described.¹⁹ The skin thermistors were held in place by waterproof tape (Hytape, Brooklyn, NY). Core and skin temperature data were collected by a personal computer (iNet-100HC, Omega Engineering, Stamford, CT) at 1-minute intervals.

To assess metabolic rate, oxygen consumption (VO_2), and carbon dioxide production were measured by indirect open circuit spirometry with a TrueMax 2400 metabolic measurement system (Parvo Medics, Sandy, UT). This system included a mouthpiece with 2 one-way valves, expiratory gas air hose, and nose clip. The sampling rate was set at 30-second averages, and data were collected in 5-minute increments at intermittent time points (base, 10 to 15 minutes, 25 to 30 minutes, 55 to 60 minutes, 85 to 90 minutes, and 115 to 120 minutes of each stage). A 3-L syringe (Hans Rudolph Model 5530, Kansas City, MO) and known concentration calibration gases were used to calibrate the metabolic measurement system before each trial. Thermal sensation was measured using the DuBois thermal sensation scale,²⁰ where 0 is very cold, 4 is neutral, and 8 is very hot. Throughout the study, participants wore an Actigraph (Model GT1M, Actigraph, Pensacola, FL) to monitor intensity and amount of physical activity. Physical activity counts per minute were calculated for both the day and night hours, and are presented in the Table. Caloric expenditure per minute (an index of heat production) was calculated by the following equation, per manufacturer guidelines:

$$\begin{aligned} \text{Calories} \cdot \text{min}^{-1} \\ = 0.0000191 \times \text{counts} \cdot \text{min}^{-1} \times \text{body mass (kg)} \end{aligned}$$

Table 1. Physical activity as measured by accelerometer counts per minute during normal sleep and sleep deprivation

Trial	Day 1	Night 1	Day 2	Night 2	Total
	0500–2200 h	2200–0500 h	0500–2200 h	2200–0500 h	
CON	277 ± 76	11 ± 6	182 ± 76	13 ± 12	154 ± 46
SDEP	290 ± 54	274 ± 47 ^a	236 ± 73	306 ± 137 ^a	270 ± 54

Data shown as mean ± SD. CON, normal sleep trial; SDEP, sleep-deprived trial.

^a $P < .001$.

STATISTICAL ANALYSIS

Separate repeated-measures analysis of variance (condition × day × time) were performed for each dependent variable using SPSS software (Version 17.0, SPSS Inc., Chicago, IL). Planned comparisons were performed on the baseline measurements for each day. In an effort to directly address each individual hypothesis, data were also analyzed as Tre change (Δ) from the start of RW (ie, 0800 hours, when the air temperature in the chamber was turned to 25°C). Significance was set a priori at $P \leq .05$, and all data are presented as mean ± SD.

Results

Physical activity was greater during SDEP, resulting in increased counts per minute (Table 1) and estimated caloric expenditure (SDEP total 1030 ± 182 kcal vs CON total 605 ± 225 kcal). Figure 2 presents the integrative response to ACE and RW under both SDEP and CON conditions.

For Tre, analysis of variance demonstrated that there was no significant three-way (condition × day × time) interaction ($F[12,72] = 0.658$, $P = .785$, $\eta^2 = .099$). However, there was a significant main effect for day ($F[2,12] = 4.698$, $P = .031$, $\eta^2 = .439$), a main effect for time ($F[6,36] = 19.074$, $P < .001$, $\eta^2 = .761$), and a significant condition by time interaction ($F[6,36] = 6.195$, $P < .001$, $\eta^2 = 0.508$). As noted in Figure 2A, Tre declined during the experiment each day, but there was no difference between conditions during RW. Baseline Tre (at 0600 hours) was significantly higher during SDEP on day 2 ($P = .014$) and day 3 ($P = .018$), but this had no impact on RW from ACE. No significant differences between SDEP and CON were found at any time point during RW. The Δ Tre during RW was not different on day 1 between SDEP ($\Delta = -0.41^\circ \pm 0.28^\circ\text{C}$) and CON ($\Delta = -0.34^\circ \pm 0.07^\circ\text{C}$). Similarly, on day 2, the Δ Tre during RW was not different between SDEP ($\Delta = -0.45^\circ \pm 0.13^\circ\text{C}$) and CON ($\Delta = -0.30^\circ \pm 0.14^\circ\text{C}$). The Δ Tre during RW was also not different on day 3 between SDEP ($\Delta = -0.43^\circ \pm 0.21^\circ\text{C}$) and CON ($\Delta = -0.35^\circ \pm 0.15^\circ\text{C}$). Regardless of day, SDEP tended to

cause a greater decline in Tre during RW (main effect for condition $F[1,6] = 4.072$, $P = .090$, $\eta^2 = 0.404$).

The Tsk increased during RW each day (Figure 2B), while metabolic rate decreased (Figure 2C). Further, participants reported higher thermal sensation, namely, they felt warmer (Figure 2D). For these variables, however, there were no between-days or between-conditions differences at any time point throughout the investigation.

Discussion

We hypothesized that thermoregulation during RW would be impaired as sleep loss progressed. However, the current investigation demonstrated that 27 to 29 hours and 51 to 53 hours of SDEP had no effect on RW from ACE. Furthermore, repeated bouts of cold air exposures (when normal sleep was allowed) had no effect on any of the measured variables during the RW period. Although contrary to our hypothesis, these findings suggest that thermoregulation during RW from cold air exposure is not impaired under 53 hours of SDEP. The time course of physiological and perceptual responses offers valuable information to persons, such as mountain climbers and military personnel, who experience the combination of cold temperature and sleep deprivation.

The afterdrop phenomenon is most pronounced after removal from cold water^{9,10} or snow burial.²¹ However, cold air exposure is also commonly experienced in a wilderness setting and results in a reduced core temperature (albeit smaller than with cold water immersion).^{11,22} Both conductive mechanisms (core to skin gradient) and convective mechanisms (circulatory) have been implicated in the afterdrop phenomenon. We had hypothesized that the drop in core temperature would be greater with SDEP, but that was not the case. The Tre declined in all subjects during RW, but the magnitude (range 0.1° to 0.8°C) is unlikely to be of clinical significance. As expected, the decline in Tre was coincident with an increase in Tsk, a decrease in VO_2 , and an increase in thermal sensation. These findings confirm

Figure 2. Physiological and perceptual responses to a 53-hour period of sleep deprivation (SDEP) and normal sleep (CON). (A) Rectal temperature (T_{re}); (B) mean skin temperature (T_{sk}); (C) oxygen consumption (VO_2); (D) thermal sensation. Mean $\pm$ SD. *Significant difference between SDEP and CON. Acute cold exposure (ACE [in 10°C]) always occurred between 0600 hours and 0800 hours, followed by rewarming (RW [in 25°C]) from 0800 to 1000 hours.

previous literature and demonstrate that our chosen cold air protocol was indeed a significant stressor to the body. Several investigations have evaluated the effects of SDEP or cold air exposure on thermoregulation but few have examined the effects of combined stressors. No

studies to date have evaluated RW from ACE (ie, after-drop) under these conditions. Repeat bouts of cold exposure on separate days may occur in sleep-deprived military personnel. Acute cold air exposure initiates peripheral vasoconstriction (decreased T_{sk}), increased met-

abolic rate (increased VO_2), and the sensation of cold (thermal sensation decreases), which tend to increase Tre temporarily but then a progressive decline in Tre is experienced.¹² It is known that the amount of clothing as well as the duration and intensity of cold exposure modify these compensatory responses, although that was not examined in the current investigation.

In this study, baseline Tre was significantly higher at 0600 hours on both day 2 and day 3 of SDEP and may be due to the increased physical activity during the nights. A previous report from our laboratory^{7,8} revealed that 33 hours of SDEP had no effect on any of the measured variables (Tre, Tsk, VO_2) during cold exposure. However, that study contained a mixed-gender sample that underwent cold exposure in the afternoon (ie, when physiological core temperature is generally higher). That study also did not measure RW or multiple exposures to cold, so direct comparison to the current study is challenging.

Prior work in this discipline has shown that maintenance of Tre during cold exposure is hindered by exercise-induced fatigue in combination with sleep loss, weight loss, and multifactorial military stress.^{23,24} Although RW after cold exposure was not directly evaluated in these cited studies, the proposed mechanism of “hiker’s hypothermia” bears some resemblance to the circulatory (convective) theory of afterdrop. Specifically, increases in peripheral blood flow by exercise or RW would facilitate heat loss whereas underfeeding or decreased substrate availability would limit shivering-induced heat gain. Furthermore, both exertion-related fatigue and SDEP can impact higher brain function that ultimately could reduce behavioral thermoregulation. Future studies might be able to address this more directly.

Repeat exposures to a laboratory cold air stress may result in a cold acclimation, but this concept is highly debated and the mechanisms are not entirely clear.²⁵ The 3 consecutive morning cold air exposures during the CON trial were necessary for comparison with the SDEP trial. Although not the primary aim of this study, these 3 cold air exposures demonstrate that RW from ACE on days 2 and 3 (when normal sleep is allowed) is not significantly different from RW on day 1. This finding may be practically relevant for workers who undergo cold exposure and RW on consecutive days.

STUDY LIMITATIONS

The current investigation is limited by at least 4 factors. First, exposure to 10°C air is not as stressful as exposure to cold water, and it is possible that SDEP would cause a greater decline in Tre during cold water immersion. Second, no measurements of skin blood flow were ob-

tained as they are not available in our laboratory. Although Tsk was essentially identical between SDEP and CON during the ACE and RW protocol, Tsk is not as sensitive as laser-Doppler flowmetry for detecting changes in skin blood flow, which has been shown to be altered by sleep deprivation and circadian status.^{26,27} Third, rectal temperature is not an ideal method to measure the afterdrop phenomenon as it does not necessarily correspond to blood temperature under transient conditions.^{9,10} Nevertheless, the ER400-12 sensors used in our laboratory have previously tracked expected exercise-induced increases in Tre with an approximately 5-minute lag time.¹⁸ Fourth, sympathetic and cardiovascular physiology is affected by sleep deprivation,²⁸ but these measurements were not assessed during this protocol. The effect of combined SDEP and thermal stress on cardiovascular function could be an avenue for future research.

Conclusions

Military and occupational situations may involve both sleep deprivation and cold air exposure. The current investigation suggests that sleep-deprived persons are not at a greater risk for a decline in rectal temperature (ie, a hypothermic afterdrop) during rewarming from cold air.

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