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84 Hours of Exertional Fatigue, Negative Energy Balance, and Sleep Deprivation Impairs Shivering During Cold Air Exposure in Men

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Summary

Long-term (61-d) military operations that induce exertional fatigue, caloric deficiency, and sleep deprivation impair thermoregulatory responses to cold. However, there is no information regarding thermoregulation following short-term (3.5-d) sustained military operations (SUSOPS). This study examined thermoregulatory control during cold exposure following this multi-factorial stress. Thermoregulation of six men (22.8 ± 1.4 yrs) was assessed during a standardized cold air test (SCT) both before (CONTROL) and following an 84-h SUSOPS (sleep = $2\text{-h}\cdot\text{d}^{-1}$, energy intake = $\sim 1800\text{ kcal}\cdot\text{d}^{-1}$, energy expenditure = $\sim 5000\text{ kcal}\cdot\text{d}^{-1}$). SCT consisted of a ramp from 25°C to 10°C during the initial 30-min, with the ambient temperature then remaining at 10°C for an additional 150-min. SUSOPS decreased ($P < 0.05$) body weight, % body fat, and fat free mass by 3.9 kg, 1.6 %, and 1.8 kg, respectively. Metabolic heat production was lower ($P < 0.05$) during CAT following SUSOPS. Examination of the mean body temperature-metabolic heat production relationship indicated that the threshold for shivering was lower ($P < 0.05$) following SUSOPS (34.61°C) than CONTROL (35.73°C). There were no differences between trials in either peripheral heat flow ($\text{W}\cdot\text{m}^{-2}$) or mean weighted skin temperature ($^{\circ}\text{C}$). Partitioned calorimetry revealed that body heat content tended to decrease ($P = 0.09$) more during the CAT following SUSOPS (-1298 kJ) vs. CONTROL (-1164 kJ). These results indicate that 84-h of SUSOPS impairs the shivering response to cold exposure but does not increase the risk of hypothermia after 3-h of cold air exposure, but may potentially increase risk during longer exposures.

Introduction

Future military conflicts are expected to involve small rapidly mobile units who will work at a high intensity for sustained periods of time with sufficient, but limited, supplies to support work for several days. It is expected that the soldiers will work long hours with minimal sleep and will likely eat insufficient calories to balance caloric expenditure. Under such conditions, physiological alterations could occur which would compromise soldier performance and thermoregulation (1, 2, 14).

The individual effects of SUSOPS stressors including exertional fatigue, sleep deprivation, and energy deficits on thermoregulation in the cold have been studied, although not thoroughly. However, there are no studies examining how a short term (2-4 days) multifactorial SUSOPS affects thermoregulation in a cold environment. Only one study has reported the interaction of multiple factors on thermoregulation in the cold, but responses were examined after a long-term military course. In that study, our laboratory (14) studied 15 US Army Rangers following completion of their 61-day training interval. We found that immediately following Ranger School, shivering thermogenesis and peripheral heat retention were blunted, thus body temperature could not be maintained. Although this study gives insight into potential mechanisms that may be compromised during SUSOPS, the length of Ranger training leads to physiological changes (7.4 kg weight loss, substantial insulation loss) not likely to occur during a 72-80 hour sustained operation.

Studies examining sleep deprivation have generally observed no effect on acute cold responses, but the methodologies and study protocols preclude any definitive conclusions. For example, Fiorica et al. (4) observed no effect following 82-h of sleep deprivation, but in their control group, rectal temperature progressively increased over 4 days, despite testing at the same time of day and accounted for the differences in resting T_{re} before cold exposure. Kolka et al. (7) measured thermoregulatory responses during exercise in cold air which resulted in greater heat storage and elevated core temperatures. Thus, that study did not examine physiological adjustments needed to prevent a fall in core temperature. Finally, Savourey and Bittel (13) utilized only a 27-h period of sleep deprivation, which was likely inadequate to cause an effect. In summary, the studies to date suggest that sleep deprivation does not impair thermoregulatory responses to acute cold exposure. The studies however do suggest that T_{re} is regulated at a lower set point after a prolonged period (> 50-h) of sleep deprivation. Likewise, following a sustained operations field study, Bahr et al. (11) reported that core temperature was depressed 0.55°C. This finding is important as a lower starting core temperature may increase the risk of hypothermia (3).

MacDonald and associates have studied the effect of short term fasting (36-48 hours) on thermoregulatory responses during cold exposure, both at rest and during exercise. Generally, they found that T_{re} is not maintained as well after fasting in men (8) and women (9), either at rest or during exercise. At rest, the mechanism appears to be greater peripheral heat loss as blood flow is significantly greater following fasting both in thermoneutral and cool environments. In the study with women, metabolic heat production was also blunted during cooling. While this response was not observed in the men, the men's data suggest that heat production, as a function of core temperature, may also be attenuated.

Our laboratory has recently (1, 2) been examining the concept of "thermoregulatory fatigue", defined as a blunting of the shivering and/or vasoconstrictor response to cold exposure, relative to control conditions. For example, Castellani et al. (2) showed that one hour of leg exercise before cold exposure causes core temperature to fall to a greater degree than under control conditions. The mechanism is greater peripheral heat loss as shivering thermogenesis was not affected. Castellani et al. (1) also have shown that 3 days of exhaustive exercise followed by a 6-h cold exposure causes T_{re} to fall significantly. Again, the mechanism appears to be thermoregulatory fatigue of the vasoconstrictor response as mean skin temperatures were higher and metabolic heat production was unaffected, compared to control trials. This latter study only examined exertional fatigue as a potential stressor since the subjects slept 6-7 hours per night and consumed ~2500 kilocalories per day.

The purpose of this study was to examine thermoregulatory effector responses (shivering, vasoconstriction) following 84 hours of sustained operations. It was hypothesized that thermoregulatory fatigue of both shivering and vasoconstriction would occur following 84 hours of sustained operations.

Methods

Subjects. Six healthy soldiers volunteered to participate in this study as test subjects. Physical characteristics were age, 22.8 ± 1.4 (SE) yr; height, 186 ± 8 cm; mass, 84.3 ± 3.5 kg; and percent body fat, 18.1 ± 2.2 %.

Preliminary Testing. Height, body mass, and % body fat (dual energy x-ray absorbitometry; Model DPX-L, Lunar Corp., Madison, WI) were obtained before the experiment. Mass and % fat were also obtained following the 84-h sustained operation.

Experimental Design. Subjects completed two experimental cold exposure trials, between 1300-1630 hours, on separate days, spaced by one week. Each trial consisted of a standardized cold air test (SCT) preceded by one of two manipulations: A) Control or B) following SUSOPS. During the Control week, subjects were not sleep deprived or in a state of negative energy balance. However, they did perform or were subjected to a variety of physical and cognitive tests before undergoing the SCT. During the SUSOPS week, subjects performed the same physical and cognitive tests before the SCT, but overlaid on them was a limited amount of sleep and food (see below). Subjects consumed the cracker and spread from an Army Meal-Ready-to-Eat (MRE) ~105-min prior to the SCT. The subjects were dressed in only shorts, socks and woolen glove liners for the SCT. Baseline values for temperature, metabolic heat production, plasma norepinephrine, and thermal sensation were collected during a 20-minute period with conditions maintained at 25°C and 50% RH. Following this, T_{amb} was reduced by $0.5^{\circ}\text{C}\cdot\text{min}^{-1}$ over a thirty-minute period, after which T_{amb} was maintained constant at 10°C and 50% RH for an additional 150 minutes. Oxygen uptake, carbon dioxide output, and minute ventilation were measured by open-circuit spirometry at min 30, 60, 90, 120 and 150. T_{re} and mean skin temperature (T_{sk}) were obtained every minute. While exposed to the cold, the subjects were not allowed to employ behavioral thermoregulation (no unnecessary physical activity or “huddling”).

Sustained Operations. The experiment consisted of 84 h (from 0600-h on Day 1 to 1800-h, Day 4) of sustained physical activity with limited time allotted for sleep and only ~1,800 kcal of food per day. Forty-nine hours of this time period was spent doing military-relevant field exercises. The timetable of experimental tests during the Control and SUSOPS weeks is presented in Figure 1 below.

Sleep was restricted by scheduling only limited blocks of sleep and keeping soldiers busy performing mental and physical tasks for the majority of each 24 h day. Two hours per day were scheduled for sleep and sleep patterns were monitored via actigraph activity monitors.

Subjects consumed one US Army Meal-Ready-to-Eat per day during the SUSOPS week, supplemented with a bagel, juice, and a piece of fruit on the morning of Days 1, 3, and 4 and a candy bar on Day 2. Estimated caloric intake was ~1800 kilocalories per day with an estimated energy expenditure of 5000 kilocalories per day.

Physical performance tests before each SCT were the same during the Control and SUSOPS weeks. In brief, these tests consisted of an obstacle course (2 runs, ~35-sec duration), two power tests (bench throw, squat jump; ~ 30-sec duration), and a repetitive box lift for 10-min. These performance tasks occurred between 0930-1115 hours with rest scheduled between tasks.

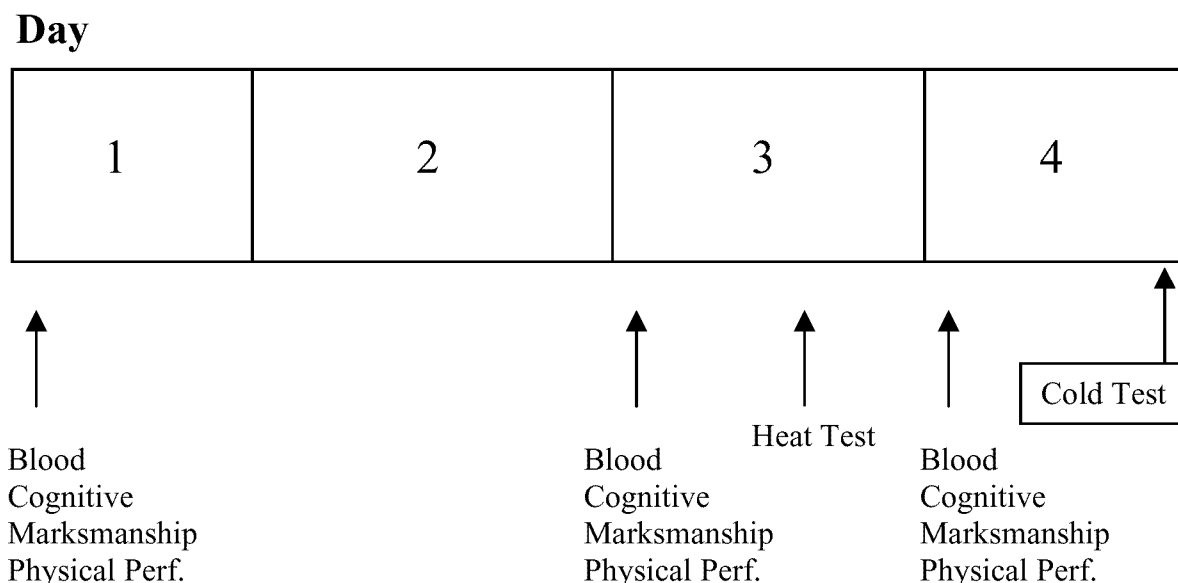


Figure 1. Experimental tests performed during Control and SUSOPS weeks.

Measurements and calculations. Rectal temperature (T_{re} ; $n = 5$) was measured using a thermistor inserted 10 cm past the anal sphincter. Esophageal temperature (T_{es} ; $n = 1$) was measured using a thermistor inserted into the esophagus to a length of $\frac{1}{4}$ body height. Skin temperature (T_{sk}) was measured using thermistor disk sensors (Concept Engineering, Old Saybrook, CT) attached on the skin surface (right side of body) at five sites (calf, medial thigh, tricep, forearm (ventral), and subscapular). Mean weighted skin temperature (T_{sk}) was calculated as: $T_{sk} = 0.28T_{subscapular} + 0.14T_{forearm} + 0.08T_{triceps} + 0.22T_{calf} + 0.28T_{thigh}$. Mean body temperature (T_b) during cold exposure was calculated as follows: $T_b = 0.67 \cdot T_{re(es)} + 0.33 \cdot T_{sk}$. Percent oxygen (Model S-3A, Applied Electrochemistry) carbon dioxide (model LB-2, Beckman) and volume (Tissot spirometer, Collins) were measured from a 90-sec collection of the subjects' air expired into a 150L Douglas Bag. Metabolic heat production ($W \cdot m^{-2}$) was estimated from VO_2 and respiratory exchange ratio (R) using the following equation: $M = (0.23[R] + 0.77) \cdot (5.873)(VO_2) \cdot (60/A_D)$ where A_D is body surface area (m^2). Body heat storage (S , $W \cdot m^{-2}$) was calculated as follows (7): $\pm S = M - W - L - E - K - (R+C)$, where M is the metabolic rate, W is work rate (0 in this experiment), L is the respiratory heat losses by convection and evaporation ($0.08 \cdot M$), E is evaporative heat loss (presumed to be negligible in this experiment and set at 0), K represents conductive heat loss (0 in this experiment) and $R+C$ ($0.83 \cdot [T_{re} - T_{sk}]$) represents dry heat loss.

Whole blood samples were drawn before cold exposure (min 0) and at minutes 30 and 175 of cold air exposure from an indwelling venous catheter (18 gauge) placed in a superficial forearm vein. Aliquots were centrifuged at $4^\circ C$ to separate the plasma. Plasma samples were frozen at $-40^\circ C$ before analysis. Plasma norepinephrine (NE) concentration was determined from mass spectroscopy-gas chromatography.

Statistical Analyses. Data were analyzed using a two-factor (experimental trial X time) repeated measures ANOVA. When significant F ratios were calculated, paired comparisons were made post-hoc using a Newman-Keuls test. The slope and intercept of each individual's T_b vs. ΔM relationship, as well as body composition variables were analyzed using repeated t-tests. Unless otherwise specified, the level of significance for differences reported is $P < 0.05$. Values are mean $\pm$ SE.

Results

Body Composition. Body composition changes are presented in Figure 2. Body mass, % body fat, and fat free mass significantly declined ($P < 0.05$) after SUSOPS.

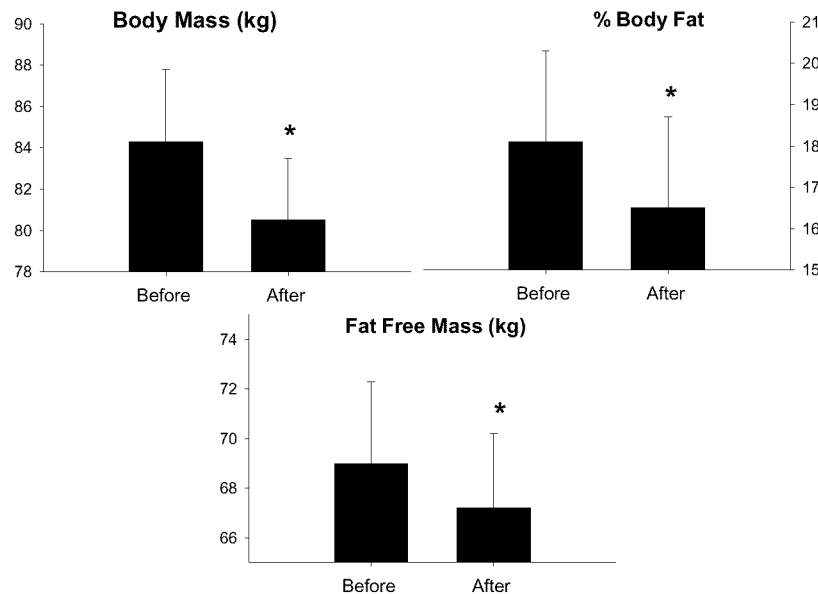


Figure 2. Body composition changes before and after SUSOPS.

Thermoregulatory Responses. Metabolic heat production was significantly lower ($P < 0.05$) at min 30, 60, and 90 during SUSOPS compared to the Control trial (Figure 3).

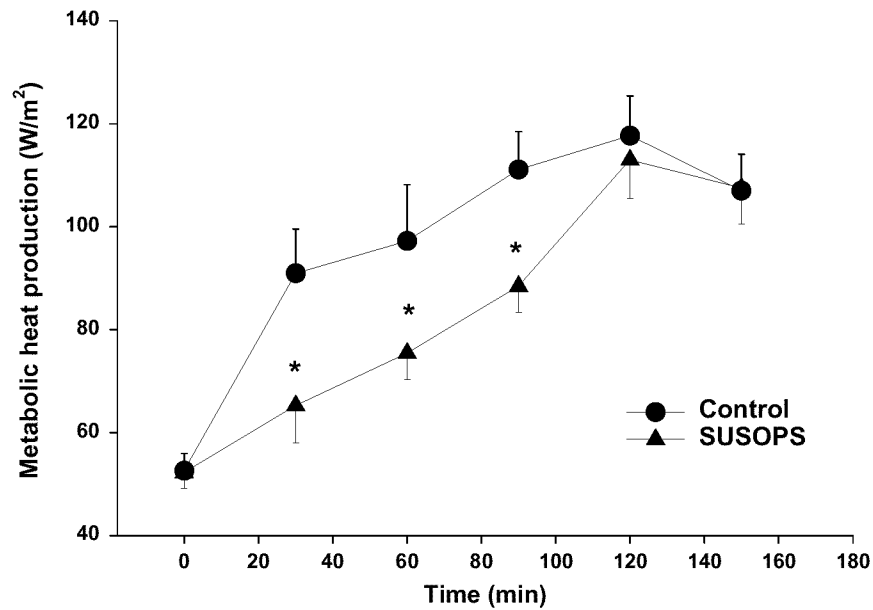


Figure 3. Metabolic heat production vs. time during Control and SUSOPS trials.

The slopes of the T_b - ΔM relationship (Figure 4) between the Control ($-28.3 \pm 4.9 \text{ W}\cdot\text{m}^{-2}\cdot^\circ\text{C}^{-1}$) and SUSOPS ($-58.0 \pm 14.1 \text{ W}\cdot\text{m}^{-2}\cdot^\circ\text{C}^{-1}$) trials approached significance ($P = 0.06$). However, there was a significant ($P < 0.05$) difference in the threshold for the onset of shivering between Control ($35.73 \pm 0.10^\circ\text{C}$) and SUSOPS ($34.61 \pm 0.19^\circ\text{C}$).

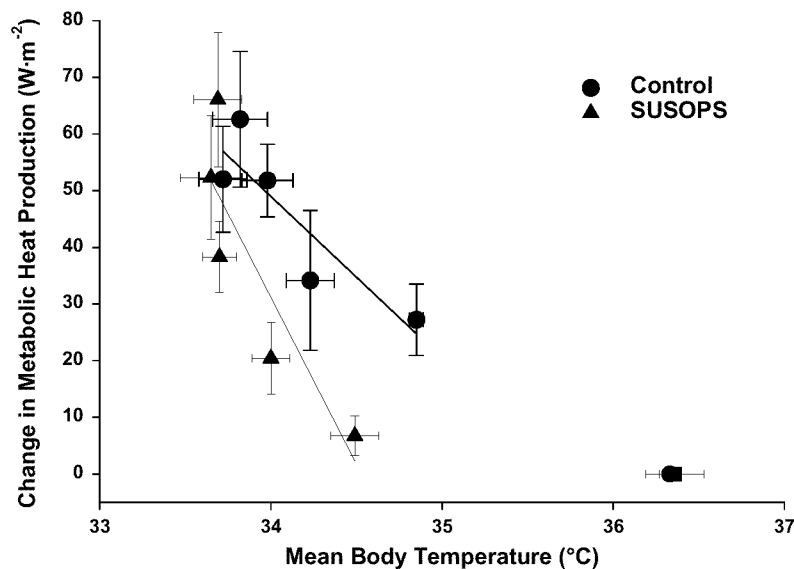


Figure 4. T_b - ΔM relationship during Control and SUSOPS trials ($n = 4$).

Mean skin temperatures were not significantly different between the two trials (at min 180, $26.75 \pm 0.31^{\circ}\text{C}$ and $26.44 \pm 0.39^{\circ}\text{C}$ for Control and SUSOPS, respectively). There also were no differences ($P = 0.09$) in the change in body heat content (Figure 5) and change in rectal temperature (Figure 6) between Control and SUSOPS trials.

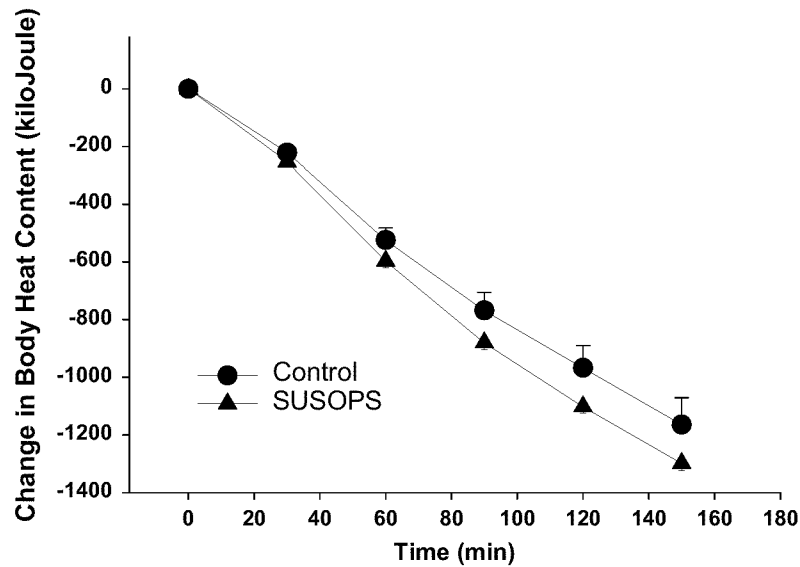


Figure 5. Change in body heat content (kJ) vs. time between Control and SUSOPS trials.

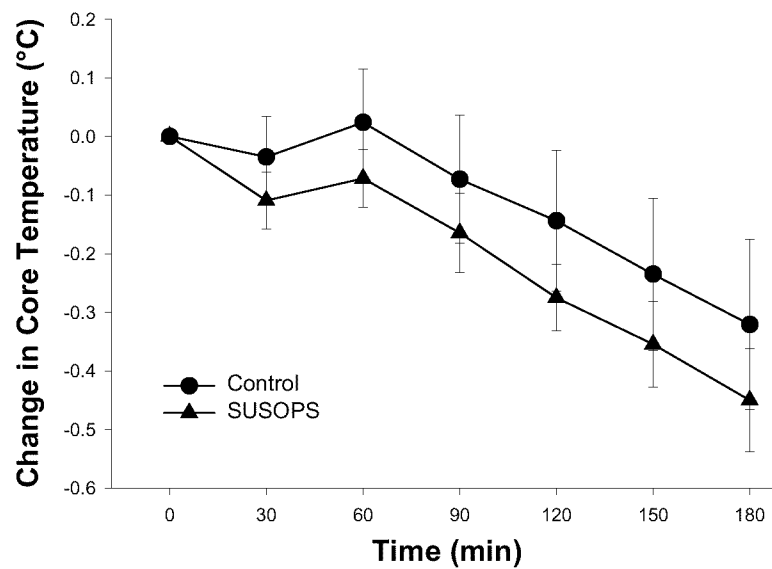


Figure 6. Change in core temperature ($^{\circ}\text{C}$) vs. time between Control and SUSOPS trials. Initial core temperatures were 37.24 ± 0.11 and 37.26 ± 0.11 , for Control and SUSOPS, respectively.

Plasma glucose and catecholamines. Plasma concentrations for glucose and norepinephrine are presented below in Tables 1 and 2. There were no significant differences within or between trials for glucose. Plasma norepinephrine was significantly higher (main effect) during SUSOPS compared to Control.

Table 1. Plasma glucose concentrations (mmol·L⁻¹)

Time	Control	SUSOPS
Pre	5.00 ± 0.17 (n = 6)	5.00 ± 0.62 (n = 5)
30-min	4.47 ± 0.37 (n = 5)	4.92 ± 0.18 (n = 4)
175-min	4.57 ± 0.18 (n = 4)	4.67 ± 0.12 (n = 4)

Table 2. Plasma norepinephrine concentrations (pg·mL⁻¹)

Time	Control	SUSOPS‡
Pre	820.5 ± 57.8 (n = 6)	1144.6 ± 179.8 (n = 6)
30-min	1868.5 ± 274.1* (n = 4)	3392.7 ± 808.4* (n = 4)
175-min	3275.0 ± 521.8* (n = 4)	4953.5 ± 344.8* (n = 4)

*, denotes significant within trial difference from Pre; ‡, significant main effect for trial, SUSOPS > Control

Discussion

Military sustained operations (SUSOPS) present a unique physiological challenge to the warfighter. Multiple stresses are imposed on the individual including sleep deprivation, negative energy balance, and heavy physical work. All three of these stressors may, independently, affect thermoregulation in the cold. The combination of these stressors has been shown, during long-term training (14) to impair both the shivering and vasoconstrictor response to cold exposure. However, this is the first study to examine the effects of a 3-4 day SUSOPS that is more common and operationally realistic, without the comparably large changes in subcutaneous fat and percent body fat observed during 61-d US Army Ranger training (14).

The principal finding in this study was the lower metabolic heat production and the lower mean body temperature for the onset of shivering following an 84-h SUSOPS, compared to rested conditions. Two possible independent mechanisms (or their interactions) for this effect are conceivable: sleep deprivation and negative energy balance. Exertional fatigue is ruled out as we have previously demonstrated this has no effect on shivering thermogenesis during subsequent cold exposure.

The role of sleep deprivation on thermoregulatory responses to cold have typically demonstrated no effect on core temperature changes and this study was no exception. However, several interesting findings from previous studies have been reported and are worth noting. For example, Fiorica et al. (4) observed that resting rectal temperatures were significantly lower following 53 and 82 hours of sleep deprivation in a sleep deprived group compared to a set of control subjects. In addition, Kolka et al. (6) also observed lower resting esophageal temperatures after 50 hours of sleep deprivation. However, we did not observe any changes in core temperature at rest just before cold exposure. Our data agree with those reported by Savourey and Bittel (13), in subjects who were sleep deprived for 27 hours. One difference between our data and Fiorica et al. (4) is that group employed a separate non-sleep deprived control group, who over the course of the 82-h experiment, experienced a significant rise in resting rectal temperature, whereas the sleep deprived group had no change in resting core temperature, thus accounting for the significant difference

between the groups. Kolka et al. (6) measured only esophageal temperatures and this may partially account for the difference as well. One possibility is that the subjects in this SUSOPS study were physically active (physical performance tests) before cold exposure, whereas in the previous studies, the subjects were sedentary. This may have masked the effect of sleep deprivation on resting core temperature. Whether sleep deprivation, independently, is responsible for the delayed onset of shivering is debatable. In contrast to our findings, Savourey and Bittel (13) found that sleep deprivation increased the sensitivity of the shivering response, such that shivering began earlier. However, that study used a subjective measure of shivering to determine onset as opposed to an objective measure such as changes in oxygen uptake. Further evidence for a delayed shivering onset is provided by Young et al. (14). Subjects in that study were sleep-deprived prior to beginning their initial experimental cold exposure. It is difficult to attribute the changes in shivering thermogenesis solely to sleep deprivation in that study and in the present SUSOPS investigation, due to the multi-factorial stressors present. Examining the effects of 2-3 day sleep deprivation on thermoregulatory responses to the cold are warranted to independently evaluate this factor.

Underfeeding and 48-h fasting have also been suggested to impair thermoregulatory responses to cold (8, 9, 14). In one case (14), it was difficult to discern whether underfeeding (relative to caloric intake) per se was responsible for the blunted shivering and vasoconstrictor responses because large changes in body composition (10% fall in % body fat) also occurred. We observed significant falls in indices of body composition as well, but not severe in magnitude (~1.5% fall in % fat) to account for changes in effector responses. In the other case, the subjects consumed no food at all for 2 days. One obvious difference between studies is that the subjects in SUSOPS were not fasting, as they consumed approximately 1800 kilocalories per day. They also consumed a small meal within 2-h of cold exposure, in order to maintain plasma glucose concentrations during the 3-h cold exposure since low glucose values are known to blunt thermoregulatory effector responses (5, 12). Changes in the core temperature-metabolic rate relationship have also been observed after 48-h fasting. Unlike the changes seen in the present study (a decrease in the shivering onset, i.e., a temperature threshold change), MacDonald and colleagues (8) found the gain or sensitivity of the metabolic response to a given fall in core temperature was blunted after 2 days of fasting in men and a blunted metabolic heat response in women (9). One hypothesis for the diminished thermogenic response is an elevated basal norepinephrine concentration. We did find elevated plasma norepinephrine values at baseline following 4 days of SUSOPS. This has also been observed following either 48-h fasting (9) or a combination of sleep loss, underfeeding, and exertional fatigue (14), potentially causing a down-regulation of beta-adrenergic receptors (10).

Interestingly, SUSOPS caused no changes in the vasoconstrictor response to cold exposure, even though previous work from our laboratory (1, 2) suggests that exertional fatigue blunts the skin temperature response during cold exposure and that the vasoconstrictor response to cold is attenuated following 48-h of fasting (8, 9) and 27 hours of sleep deprivation (13). Why there were no changes following SUSOPS is not known.

In conclusion, SUSOPS decreased the mean body temperature threshold for the onset of shivering thermogenesis. However, peripheral heat loss was not affected by 4 days of exertional fatigue, sleep deprivation, and negative energy balance. Core body temperature and body heat content were also not different following SUSOPS. Thus, even though shivering was blunted, core temperature was not compromised by SUSOPS after 3-h of cold exposure, thus there is no greater risk for hypothermia following an 84-h sustained operation.

Disclaimer

The views, opinions and/or findings in this report are those of the authors and should not be construed as official Department of the Army position, policy, or decision unless so designated by other official designation. Human subjects participated in these studies after giving their free and informed voluntary consent. Investigators adhered to AR 70-25 and USMRDC Regulation 70-25 on Use of Volunteers in Research.

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