

Cold thermoregulatory responses following exertional fatigue

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1. ABSTRACT

Participants in prolonged, physically demanding cold-weather activities are at risk for a condition called “thermoregulatory fatigue”. During cold exposure, the increased gradient favoring body heat loss to the environment is opposed by physiological responses and clothing and behavioral strategies that conserve body heat stores to defend body temperature. The primary human physiological responses elicited by cold exposure are shivering and peripheral vasoconstriction. Shivering increases thermogenesis and replaces body heat losses, while peripheral vasoconstriction improves thermal insulation of the body and retards the rate of heat loss. A body of scientific literature supports the concept that prolonged and/or repeated cold exposure, fatigue induced by sustained physical exertion, or both together, can impair the shivering and vasoconstrictor responses to cold (“thermoregulatory fatigue”). The mechanisms accounting for this thermoregulatory impairment are not clear, but there is evidence to suggest that changes in central thermoregulatory control or peripheral sympathetic responsiveness to cold lead to thermoregulatory fatigue and increased susceptibility to hypothermia.

2. INTRODUCTION

Traditionally, hypothermia experienced by participants in winter sports and recreational activities was simply attributed to effects of sustained cold exposure combined with inadequate clothing. However, research has documented degraded thermoregulatory effector responses (shivering and vasoconstriction) following physical exertion and prolonged cold exposure, increasing the risk for hypothermia. We have termed this degraded effector response “thermoregulatory fatigue” (1). The purpose of this paper is to review those studies and other relevant findings reported that could contribute to a better understanding of physiological mechanisms affecting susceptibility to hypothermia during outdoor activities in cold weather. Thermoregulatory fatigue factors clearly identified include prior physical exercise, repeated cold exposure, and physical exhaustion, while evidence suggests that sleep loss and caloric deficit with and without hypoglycemia may be additional factors.

3. HISTORICAL PERSPECTIVE

3.1. Field Observations

Persons engaged in outdoor activities during cold weather are at risk for accidental hypothermia. Pugh first

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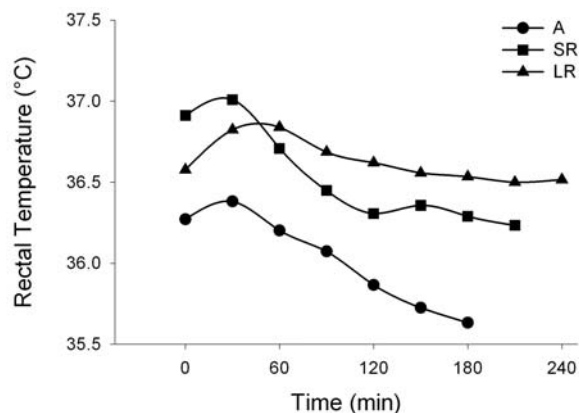


Figure 1. Rectal temperature vs. time during sedentary exposure to 10°C air following 61-days of exertional fatigue, sleep deprivation, and negative energy balance (A), after 48-h of rest and recovery (SR) and after 16 weeks of rest and recovery. From (6).

suggested that physical exertion might increase the risk of hypothermia (2). He analyzed reports of 23 separate occasions that had led to numerous cases of hypothermia and 25 deaths. On one occasion, 240 men began walking at 0600 in a drizzle and light wind, vying to be the fastest participants in the Four Inns Walking Competition, (3) a combination race/hike traversing 45 miles over the moors of Derbyshire, England (time to complete usually ranged from 9.5 to 22 hours). During this particular contest, the weather became progressively worse with heavy rain and high winds (25 knots). Only 22 competitors completed the race that year (compared to 66-85% typically observed) and three died from hypothermia. In his analysis of these incidences, Pugh identified physical exhaustion as a contributing factor for hypothermia, although he was unable to provide conclusive evidence. More recently exhaustion and hypothermia were again linked as causal factors for a large number of serious hypothermia casualties and four deaths during cold exposure following a grueling 60-day military training course (U.S. Army Ranger School) in which soldiers are underfed, sleep deprived, and physically exhausted. These cases suggested the possibility that physical exhaustion was linked to hypothermia, but again the scientific evidence for a cause and effect association between physical exertion and susceptibility to hypothermia was lacking.

3.2. Concept of Shivering Fatigue

The first experimental observation suggesting that a thermoregulatory response, namely shivering, could exhibit an impairment possibly reflecting a fatigue of that response was reported by Thompson and Hayward (4). Those authors reported that during a five-hour walk at a constant pace in a controlled experimental environment that simulated hiking in cold, rainy conditions, they observed one subject who, having maintained stable metabolic rate and core temperature for the first three hours of exposure, exhibited a progressive decline in metabolic rate and core temperature over the final two hours, despite the fact that walking pace remained unchanged, throughout (4). The

authors concluded that the decline in metabolic rate exhibited by this subject reflected a decrease in shivering, since they reported that the subject maintained the same walking pace, and they concluded that this decrease in shivering was evidence for a fatigue or exhaustion of shivering responses. However, since this effect was only observable in one of the five subjects reported, and decreases in muscular activity besides shivering might account for a decline in overall metabolic rate without a change in walking pace, the author's conclusions remained speculative. More quantitative evidence for shivering fatigue has been reported by Bell *et al.* (5). They found that over a 2-h period during resting exposure in 10°C air, the central frequency of the EMG recording in the pectoralis major decreased with time, suggesting fatigue of this muscle group.

4. CENTRAL ATTENUATION OF THERMOREGULATION—SET POINT CHANGES

4.1. Multi-Stressor Studies

Based on the intriguing findings of Thompson and Hayward, our laboratory investigated how physiological responses to cold were affected by fatigue associated with more prolonged physical exertion, controlling for possible confounders. Responses to cold were measured in 8 men who had completed an arduous nine-week military training course, throughout which participants perform very strenuous physical activity and daily sleep is limited to about four hours (6). During this training, daily energy expenditures averaged 4,100 kcal (17.2 mJ) per day, while daily energy intakes averaged only about 3,300 kcal (13.8 mJ) per day, and there were many periods when energy expenditures were much higher and intakes lower. The subjects in this study completed a standardized experimental cold air exposure within two hours after finishing this regimen (no rest), again following a short (48 hours) recovery period for rest and refeeding, and again a third time following 16 weeks of recovery. Our experiments demonstrated that cold tolerance (ability to maintain a core temperature above 35.5°C) and the ability to maintain normal body temperature (Figure 1) during cold exposure was compromised during the trial performed without rest and remained compromised even after 48-hr of recovery.

Alterations in normal shivering responses to cold may have contributed to the impaired maintenance of thermal balance. Shivering responses were delayed (Figure 2) during the cold-exposure trial performed immediately after subjects had completed the exhaustive training course, compared to trials completed after rest and recovery (6). This shift to the left in the metabolic heat production as the mean body temperature declined is suggestive of a central attenuation in this thermoregulatory effector response.

Subsequently, we were able to use a controlled, laboratory-based simulation to expose subjects to the multi-stressor environment studied by Young *et al.* (6) and we replicated their observations that exertional fatigue, negative energy balance and sleep loss, in combination, impair cold-induced shivering (7). Subjects were exposed

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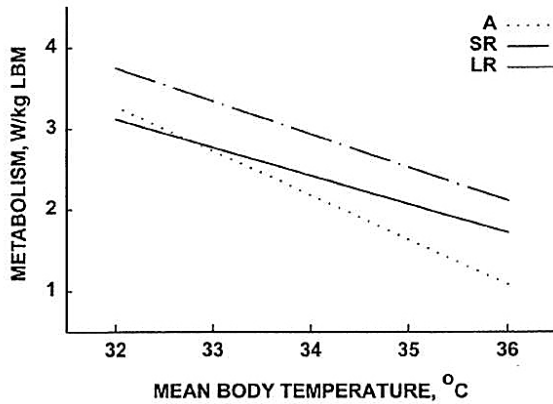


Figure 2. Metabolic heat production vs. mean body temperature during sedentary exposure to 10°C air following 61-days of exertional fatigue, sleep deprivation, and negative energy balance (A), after 48-h of rest and recovery (SR) and after 16 weeks of rest and recovery. A demonstrated a significant shift for the onset of shivering thermogenesis. Reprinted with permission from (6).

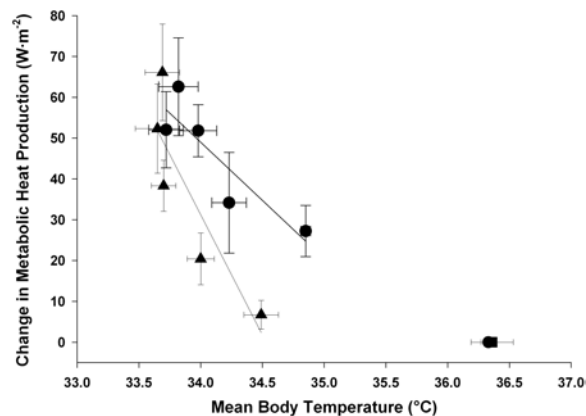


Figure 3. Metabolic heat production vs. mean body temperature during sedentary exposure to 10°C air following 84-hours of exertional fatigue, sleep deprivation, and negative energy balance (SUSOPS) vs. rested conditions (Control). SUSOPS demonstrated a significant shift for the onset of shivering thermogenesis. Reprinted with permission from (7).

to 84-h of negative energy balance (energy intake of 1,653 kcal·d⁻¹ vs. an estimated mean total daily energy expenditure of 4500 kcal·d⁻¹), sleep restriction (6-h total throughout the 84-h period), and high levels of physical activity. The principal finding in this study was that core temperature fell to a greater extent during cold-air exposure following 84-h of exertional fatigue, negative energy balance and sleep loss. Figure 3 shows the mean body temperature-metabolic heat production response before and after the 84-h multi-stress period. As in the Ranger study, there was a delayed onset of shivering following the 84-h multi-stressor environment, compared to rested conditions. However, in this new experiment we avoided the confounding effects of large changes in body fat and tissue

insulation observed following 61-d of US Army Ranger training (6). It is well known that higher body fat percentages (causing higher tissue insulation) maintain overall thermal balance during cold exposure (8,9).

Three primary stressors were present during the 61-day and 84-h studies that may impact shivering thermogenesis. They are sleep deprivation, negative energy balance, and exertional fatigue caused by previous exercise. However, we have demonstrated that prior exercise does not blunt shivering thermogenesis during subsequent cold exposure (1,10).

Sleep deprivation studies (11) (12) (13) have generally observed no effect on core temperature responses to cold exposure, but the methodologies (e.g., using exercise to keep people awake vs. sedentary exposure) and study protocols preclude definitive conclusions. The studies cited above had the subjects engage in no physical activity. Interestingly, in contrast to our finding of a delayed shivering response, Savourey and Bittel (13) found that sleep deprivation increased the sensitivity of the shivering response, i.e., shivering began earlier. Landis *et al.* (14) found that one night of sleep deprivation lowered forearm blood flow responses to an initial fall of skin temperature from 35°C to 32°C (suggestive of enhanced vasoconstriction), but when skin was heated to 38°C and then cooled to 32°C, esophageal temperature declined more rapidly after sleep deprivation, although forearm blood flow was not altered and could not explain the change in core temperature. Sleep deprivation may also play a role by changing the set-point temperature at which physiological responses are regulated. Following both a multi-stressor scenario (15) and sleep deprivation alone (12), core temperature was lowered ~0.5°C at rest, but neither of these studies provides conclusive evidence that sleep loss reduces set-point. Thus sleep deprivation effects on thermoregulatory responses to cold are unclear.

Negative energy balance also impacts shivering thermogenesis. Macdonald and colleagues found a reduced gain (sensitivity) in the metabolic rate-core temperature relationship after 2 days of fasting in men (16). Similarly, a decline in the metabolic rate response to cold after fasting was observed in women following 48-h of food deprivation (17). These findings contrast somewhat with our 84-h multiple stressor results which demonstrated a decrease in the shivering onset, i.e., a temperature threshold change rather than a change in the sensitivity of the response. The different shivering responses to the underfeeding stress between these fasting studies and the 61-day and 84-h multi-stressor studies may be due to the type of underfeeding. In the multiple stressor studies, subjects were underfed relative to their energy expenditure for > 2 days, whereas in the fasting studies, subjects were sedentary and consumed no food at all. One possibility is that the diminished metabolic heat response is due to an elevated basal norepinephrine level that has been observed following either 48-h of fasting (17) and which we observed following 61-days of Ranger training may lead to a down-regulation of beta-adrenergic receptors (18), that is, a decrease in the density of beta-adrenergic receptors as well as a decreased affinity of the receptors for NE.

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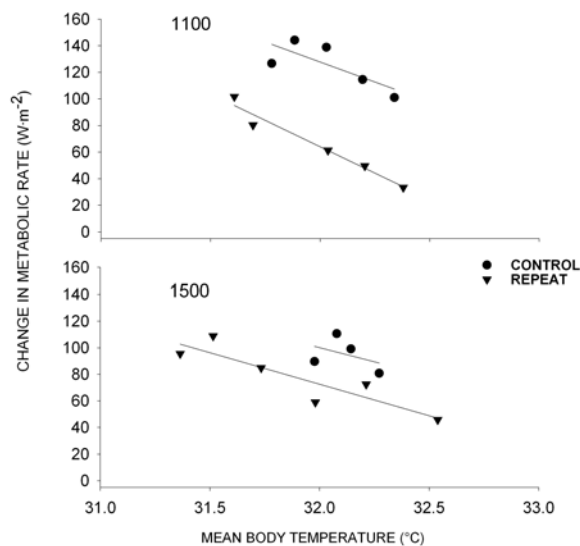


Figure 4. Metabolic heat production vs. mean body temperature during sedentary exposure to 20°C water during immersion at different times of day 1100 and 1500. During Control (circle), only a single immersion per day was completed, while during Repeat (triangle), three immersions were completed serially in a single day. Repeat demonstrated a significant shift to the left for the onset of shivering thermogenesis so that any given mean body temperature, shivering thermogenesis was lower. Reprinted with permission from (19).

4.2. Serial Cold Water Immersion

The multiple stressor studies provided insight into possible mechanisms of shivering fatigue, but did not answer the question directly, i.e., does a muscle that is shivering for long durations fatigue over time? To determine whether shivering responses to cold exhibited signs of fatigue, metabolic heat production was measured during 2-hour cold-water immersions (20°C) repeated three times, serially in a single day (2-hour rewarming intervening), and compared metabolic heat production measured during a single immersion, completed at the same time of day (19). Cold-water immersion produces more rapid core and peripheral cooling and induces higher shivering rates than cold air, potentially causing fatigue. As shown in Figure 4, metabolic heat production was lower during the serial immersions (REPEAT), than when only a single immersion was completed at that same time of day (CONTROL), suggesting that the shivering response did indeed exhibit a fatigue during prolonged or repeated activation (19). The blunted thermogenic response in REPEAT appears to be due to a delay in the shivering onset, i.e. the intercept for the mean body temperature-change in metabolic heat production relationship shifted such that the increase in metabolic heat production during the 1100 and 1500 REPEAT exposures was not observed until the subjects achieved a lower mean body temperature. These data, like the shift in shivering onset observed in the multiple stressor studies suggests a centrally-mediated change in the recruitment of muscle for shivering thermogenesis, leading to a greater susceptibility to hypothermia.

5. PERIPHERAL ATTENUATION – EFFECTS ON HEAT LOSS

5.1. Acute Exercise

Exercise could increase the risk of hypothermia during subsequent cold exposure for several reasons. First, exercise might mediate “thermoregulatory fatigue” which would blunt shivering responses and reduce vasoconstriction during subsequent cold exposure. Second, cold exposure immediately after performing exercise might result in accentuated heat loss from an inability to immediately switch from heat dissipation responses caused by exercise in temperate conditions (20) to heat conserving responses needed during cold exposure. Third, exercise might mediate greater heat loss during subsequent cold exposure due to “heat redistribution” to active limbs that develops during exercise. During exercise, perfusion of active skeletal muscle increases and perfusion can remain elevated for extended durations (21) facilitating regional heat loss over these active limbs during exercise (22).

Castellani *et al.* (1) tested these three hypotheses by exposing 10 men to cold air (5°C) following cycle ergometer exercise that increased core temperature by 1°C and comparing responses to those observed when cold exposure was preceded by passively heating subjects until they achieved the same pre-immersion rectal temperature. The experimental data appear to confirm the suggestion that exertional fatigue is a primary factor that can impair vasoconstrictor responses to cold. Skin surface heat flow was greater (Figure 5), skin temperatures tended to be higher, and rectal temperatures fell more and faster following exercise compared to passive exposure. Tikuisis *et al.* (23) also demonstrated that 5-h of acute exercise increased peripheral heat loss during subsequent cold-wet exposure. In contrast, Scott *et al.* (24) observed that passive heat exposure results in faster core cooling rates vs. exercise, most likely due to a greater skin to environmental temperature gradient than what we observed. Since the experimental conditions employed allowed sleep deprivation and energy substrate availability to be ruled out as significant influences, those findings were interpreted as evidence for a impairment of cold-induced vasoconstrictor response induced by exertional fatigue and elevated sympathetic nervous system activity (1). Cold-induced vasoconstriction is sympathetically mediated, and the norepinephrine (NE) response to cold, considered reflective of sympathetic nervous activation (25), was the same whether cold exposure was preceded by exercise or passive heating. On the other hand, sensitivity of peripheral arterioles to NE released in response to cold might be diminished following exercise (26).

There was no effect of exercise on shivering thermogenesis which suggests that this response to cold is not easily fatiguable. Scott *et al.* also observed a similar response (24). We observed no difference in the mean body temperature vs. change in metabolic heat production relationship between trials suggesting that the differences in core temperature between trials were not due to a change in central control of shivering thermogenesis. Perhaps

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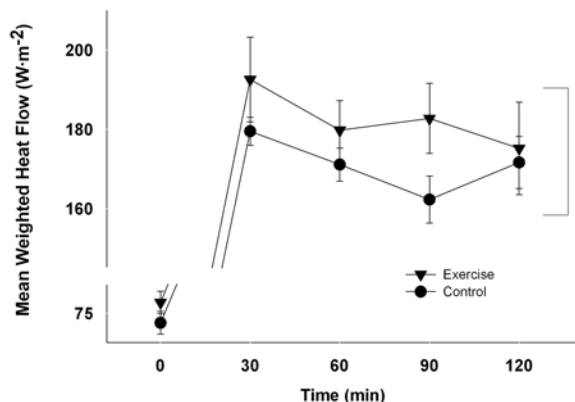


Figure 5. Skin surface heat flow (mean±SE) of men resting in cold air following exercise (Exercise) or passive heating (Control) to a similar pre-immersion rectal temperature. Asterisk indicates that heat flow was greater ($P < 0.05$) in Exercise vs. Control Reprinted with permission from (1).

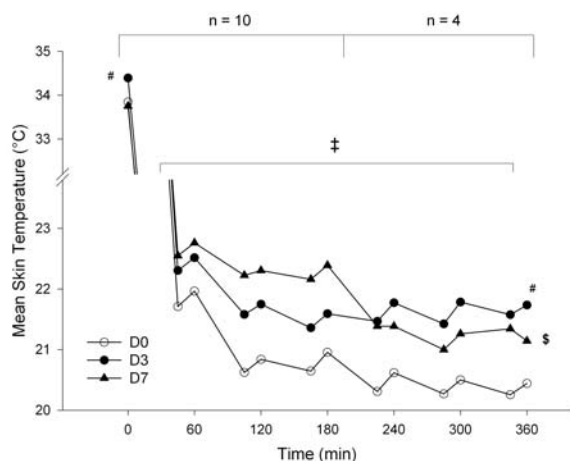


Figure 6. Mean skin temperature vs. time during cold exposure before (D0), after 3 days (D3) and after 7 days (D7) of physical exertion. Data from min 0 to min 180 are from 10 subjects and data from min 190 to min 360 are from 4 subjects. ‡, D3 and D7 significantly ($P < 0.05$) different than D0; #, D3 significantly ($P < 0.05$) different than D0 and D7; \$, D7 significantly.

exercise intensity and duration were not sufficient to fatigue the shivering mechanism, which is a relatively low intensity activity (27), at least compared to exercise. In Pugh's case report of the Four Inn's Walk (3), the participants were exercising up to 20-h in cold-wet conditions. Likewise, the subject in Thompson and Hayward's study (4) who developed shivering fatigue was exercising for 4-h in severe cold-wet conditions. Another possibility is that shivering impairments observed in these earlier studies may not reflect fatigue, but rather hypoglycemia, which is known to impair shivering (28,29). Plasma glucose levels were not measured in those previous studies (3,4). In our acute exercise study, plasma glucose concentrations remained normal throughout cold exposure.

5.2. Chronic Exercise

Because exercise-induced hyperemia may have persisted during cold exposure following acute exercise and accounted for the greater heat loss rather than impaired vasoconstriction, a follow-on study was conducted (10). In this experiment volunteers walked for up to 6 hours at $1.34 \text{ m}\cdot\text{s}^{-1}$ in 5°C air with a $5.4 \text{ m}\cdot\text{s}^{-1}$ wind while completely wet. Subjects wore Army clothing with an initial insulation value of 1.3 clo (though it became lower due to wetness). This experimental design utilized exercise at a fixed intensity so any effects of exercise-induced hyperemia should have been similar for all trials (exercise fatigue or no fatigue). A group of ten men performed this "wet-walk" on days 0, after 3 days (D3) of fatiguing exercise, and following 7 days (D7) of exercise. The daily exercise routine (4 hours) consisted of: 4.8 km runs at their personal best; weightlifting - one set of repetitions to exhaustion on four different resistance exercises (row, chest press, lat pull-down, biceps curl), each at 70% of the one repetition maximum; four consecutive 20 min sets of stair-stepping, rowing, treadmill walking, upright cycling, and semi-recumbent cycling, all at $\sim 65\% \text{ VO}_{2\text{peak}}$; and one 30-sec anaerobic test (Wingate test) on a cycle ergometer. Hiking was performed on D3 and D7 and consisted of a 9.7 km hike over varied terrain at $\sim 6.4 \text{ km}\cdot\text{hr}^{-1}$, carrying a 9.1 kg backpack. The exercise routine was designed to fatigue the subjects but no objective measure of fatigue was collected. Three control subjects performed the "wet-walk" on these days also, but did not perform the exhaustive exercise regimen. Also, experimental controls were again in place to obviate sleep deprivation and energy substrate levels as influential factors.

A similar impairment of the vasoconstrictor response to cold, as observed in the acute exercise studies, was observed following 3 and 7 days (Figure 6) of prolonged physical exertion for 4 hours each day compared to cold exposures completed when subjects were physically rested (10). Shivering thermogenesis was not affected by chronic exercise. Since the subjects were exercising at a fixed intensity in the cold ($1.34 \text{ m}\cdot\text{s}^{-1}$), the effects of exercise-induced hyperemia ("heat redistribution") should have been similar for all trials and thus that mechanism could be ruled out. Our observations indicate that fatigue induced by exhaustive exercise may indeed blunt the vasoconstrictor response during cold exposure. Furthermore, the development of cold habituation could also be ruled out as a potential confounder in this study because the design included a control group who completed the repeated cold exposure experiments without participating in the intervening exercise regimen, and demonstrated no between trial differences in thermoregulatory and body temperature responses. Thus, it seems reasonable to conclude that some other mechanism related to exertional fatigue acts to impair vasoconstrictor response to cold.

The blunting of the vasoconstrictor response to cold subsequent to severe physical exertion may be related to concomitant elevations in basal circulating norepinephrine levels that we observed in the D3 and D7 trials. Opstad (18) observed higher circulating NE levels in

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soldiers following multiple days of exhaustive exercise coupled with sleep deprivation, and Young *et al.* (6) reported similar effects in soldiers following Ranger school. In this chronic exercise study, we observed that basal NE levels were elevated in our subjects after three and seven consecutive days of exercise. Despite the elevation of basal NE concentrations, cold exposure elicited similar sympathetic activation during all three cold exposures, as evidenced by the increment in NE concentrations over pre-exposure levels observed by the end of each of the cold exposures, the magnitude of which did not differ among trials. Stimulation of adrenergic receptors is thought the primary mechanism which mediates cold-induced vasoconstriction (30). Since the increment in NE, relative to pre-exposure levels, was similar during all three cold exposure trials, a blunted sympathetic nervous stimulus does not appear to account for the less pronounced vasoconstrictor response. However, a diminished sensitivity of the adrenergic receptors remains as a viable mechanism to explain the blunting of cold-induced vasoconstriction observed. Chronically elevated NE levels have been shown to decrease adrenergic receptor sensitivity in animal models (31), and similar effects have been suggested to develop in humans in whom circulating NE levels remain chronically elevated (18).

6. MECHANISMS OF THERMOREGULATORY FATIGUE

6.1. Energy substrates

The specific fatigue-related physiological mechanisms by which thermoregulatory responses to cold become impaired following prolonged physical exertion remain unidentified. One possibility is that exercise-induced depletion of energy substrates impairs thermoregulation. Ainslie *et al.* (32) observed that men completing a 21-km, self-pace walk in mountainous terrain sustained lower core temperatures when energy intake was severely restricted (616 kcal, 2.6 mJ) compared to trials in which energy intake was significantly higher (3019 kcal, 12.6 mJ). However, a direct effect of dietary energy restriction to impair shivering and metabolic heat production during cold exposure seems unlikely. Research (33,34,35,36) has generally shown that exercise-associated depletion of muscle glycogen is an unlikely explanation for the impaired shivering responses that we have reported. Further, while severe depletion of circulating energy substrate (i.e., blood glucose) has been shown to impair shivering (28,29), this too has been ruled out as a factor in the experimental observations of shivering fatigue that we have reported (19,7,6). Ainslie *et al.* (32) speculated that lower blood glucose during the restricted energy intake trials of their hill-walking study, might have caused an increase in peripheral blood flow, facilitating heat loss, compared to trials when energy intake was high, but presented no data to support that speculation. However, in our studies that found an increase in heat flow and skin temperature, blood glucose levels did not decline and glucose levels appear unlikely to be mediating this response.

6.2. Sympathetic nervous system desensitization

There are some data to suggest that a blunting of sympathetic nervous activation may be a more viable mechanism mediating impaired thermoregulatory responses to cold following sustained physical exertion and environmental stress. Young *et al.* (6) reported that, besides showing signs of impaired thermoregulatory responses to cold, volunteers who had just completed a prolonged period of exhaustive physical exertion with inadequate rest also exhibited elevated basal levels of circulating norepinephrine, but smaller increments in circulating norepinephrine during cold exposure compared to when they were exposed to cold in rested conditions. Sympathetic nervous system release of norepinephrine is the primary mediator of cold-induced vasoconstriction, by stimulating alpha-adrenoreceptors in the cutaneous vasculature (30,37). Therefore, a reasonable working hypothesis to explain “thermoregulatory fatigue” and the impairment of thermoregulatory responses to cold with sustained overexertion is that with chronic sympathetic nervous activation, the capacity for further activation in response to an added cold stimulus may be limited. Perhaps adrenergic receptors involved in modulating cold-induced vasoconstriction become “down-regulated” (reduced receptor density and affinity for ligand) by chronic sympathetic activation, associated with chronic exertion. Mechanistically, this hypothesis is similar to that observed with aging, although many other factors affect age-related changes. Age-related decreases in the body temperature threshold for onset of sympathetic nervous response to cold, the magnitude of associated norepinephrine release, and the magnitude of the resulting vasoconstrictor response to cold have all been reported, and are thought to account for impairments in temperature regulation known to develop with aging (37,38). Numerous laboratories have documented an attenuated vasoconstrictor response to cold in aged humans (39,40,41); however it was not until recently that the underlying mechanisms have been elucidated (42). Using the same pharmacological agents as Stephens (43,44), Thompson and Kenney (42) demonstrated that the attenuated vasoconstrictor response in aged humans is due to a reduced NE contribution and a complete absence of co-transmitter function. In a follow-up study (45), they demonstrated that there is also decreased receptor sensitivity to exogenous NE during local cooling in aged human skin. Whether similar alterations in the mechanisms of vasoconstriction exist during thermoregulatory fatigue as suggested in aged human skin is unknown and merits further study.

6.3. Vasodilator Effects

Another possibility for the mechanism of thermoregulatory fatigue is a persistent vasodilator signal after exercise causing higher skin blood flow during subsequent cold exposure. Vasodilation is partly nitric oxide-dependent, as inhibition of nitric oxide synthase, the enzyme that produces nitric oxide, reduces cutaneous vasodilation ~50% (46). Exercise sympatholysis is an attenuation of the vascular response to an augmented sympathetic outflow (47) and may be responsible for reducing reflex activation of cold-induced vasoconstriction

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following exercise. Putative mediators of exercise sympatholysis include elevated muscle temperatures, acidosis, and exercise-induced release of vasodilators, such as adenosine, prostaglandins, and nitric oxide (47,48). For example, mild acidosis is a likely outcome following strenuous exercise and has been shown to cause selective attenuation of alpha-2 adrenergic receptors. Nitric oxide has been suggested to play an integral role in contributing to exercise sympatholysis (47). Future studies that selectively inhibit vasodilatory substances using techniques like microdialysis will enable further understanding of the mechanism underlying thermoregulatory fatigue.

7. SUMMARY

In conclusion, research suggests that the ability to increase insulation by reducing peripheral blood flow in response to cold exposure becomes impaired following exercise. It remains unclear whether this effect was due to a fatigue of the vasoconstrictor response to cold associated with a peripheral sympathetic nervous system mechanism. The shivering response to cold appears to be resistant to the effects of several hours or even several days of exhaustive exercise, but when extremely high levels of exertion are sustained for 3 days to many weeks in conjunction with negative energy balance and sleep loss, shivering does become impaired via a shift in the shivering onset. Furthermore, multiple bouts of cold immersion also impair the shivering response via a centrally-mediated shift in the shivering onset. These findings have practical importance for the development of several countermeasures to reduce the risk of hypothermia. Attenuated shivering and vasoconstrictor responses can be incorporated into thermoregulatory models that predict core temperature so that guidance can be developed for fatigued individuals in cold-weather. Furthermore, future research is warranted that determines the mechanistic underpinnings causing peripheral vasoconstrictor responses to be blunted so that countermeasures (e.g., cream) can be developed that reduce heat loss following fatiguing exercise.

8. DISCLAIMER

The views, opinions and/or findings contained in this report are those of the authors and should not be construed as an official Department of the Army position, policy, or decision unless designated by other official documentation. Approved for public release; distribution in unlimited.

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