Thermoregulation during cold exposure: effects of prior exercise

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Castellani, John W., Andrew J. Young, James E. Kain, Amy Rouse, and Michael N. Sawka. Thermoregulation during cold exposure: effects of prior exercise. J. Appl. Physiol. 87(1): 247–252, 1999.—This study examined whether acute exercise would impair the body’s capability to maintain thermal balance during a subsequent cold exposure. Ten men rested for 2 h during a standardized cold-air test (4.6°C) after two treatments: 1) 60 min of cycle exercise (Ex) at 55% peak O\textsubscript{2} uptake and 2) passive heating (Heat). Ex was performed during a 35°C water immersion (WI), and Heat was conducted during a 38.2°C WI. The duration of Heat was individually adjusted (mean = 53 min) so that rectal temperature was similar at the end of WI in both Ex (38.2°C) and Heat (38.1°C). During the cold-air test after Ex, relative to Heat 1) rectal temperature was lower (P < 0.05) from minutes 40–120, 2) mean weighted heat flow was higher (P < 0.05), 3) insulation was lower (P < 0.05), and 4) metabolic heat production was not different. These results suggest that prior physical exercise may predispose a person to greater heat loss and to experience a larger decline in core temperature when subsequently exposed to cold air. The combination of exercise intensity and duration studied in these experiments did not fatigue the shivering response to cold exposure.

heat flow; hypothermia; shivering; thermal sensation; vasoconstriction

EXERCISE HAS BEEN CONJECTURED to increase an individual’s risk of hypothermia during cold exposure (3, 5, 27). However, experimental and clinical evidence for this are largely anecdotal. Over 30 years ago, Pugh (18, 19) concluded that exercise-induced fatigue was an etiologic factor predisposing hikers, climbers, and outdoor enthusiasts to hypothermia, but he provided no data demonstrating this belief with a physiological mechanism for this predisposition. Recently, Thompson and Hayward (25) suggested that exercise during cold-wet exposure may fatigue shivering thermogenesis, but their findings did not definitively support their speculation. Others (16, 28) have reported that exercise performed before subsequent cold-water immersion exacerbates the fall in core temperature (T\textsubscript{core}) but these results were inconclusive because preimmersion T\textsubscript{core} differed between the experiments (16), or a cross-sectional methodology was employed (28). Furthermore, because water has such a high thermal conductivity, peripheral heat loss during cold-water immersion may be too pronounced for exercise effects on thermal balance and thermoregulatory effector responses to be detected.

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. For example, our laboratory (29) has observed that a prolonged period of physical exertion coupled with sleep deprivation and negative energy balance resulted in a lowered threshold for shivering, despite normal plasma glucose concentrations. Those findings, however, did not allow isolation of the effects of previous exercise from sleep deprivation and negative energy balance. Second, cold exposure immediately after performing leg exercise might result in accentuated heat loss from “thermoregulatory lag.” Thermoregulatory responses are aimed at facilitating heat dissipation during exercise in temperate conditions (21), and subsequent cold exposure might mediate a “lag” in switching from heat loss to conservation. Evidence for this might include increased heat loss from areas of active cutaneous vasodilation such as the torso and arms. Third, exercise might mediate greater heat loss during subsequent cold exposure due to “heat redistribution” to active limbs. During exercise, active skeletal muscle increases perfusion, and perfusion can remain elevated for extended durations (24), facilitating regional heat loss over these active limbs during exercise (20). Evidence for heat redistribution might include greater regional heat loss over the active limbs (legs) during subsequent cold exposure.

This study examined whether exercise impairs the body’s capability to maintain thermal balance during subsequent cold exposure. It was hypothesized that a greater decrease in T\textsubscript{core} would occur during cold exposure after exercise compared with cold exposure preceded by resting. We hypothesized that exercise would mediate some combination of thermoregulatory fatigue, thermoregulatory lag, and/or heat redistribution, which would be manifested as a more rapid cooling rate during cold exposure. To distinguish between these potential mechanisms, and the “thermal” consequences of exercise (increased T\textsubscript{core}), control experiments were performed after passive heating to elevate the initial T\textsubscript{core} to the same levels achieved by exercise.

METHODS

Subjects. Ten healthy men volunteered to participate in this study as test subjects. Physical characteristics were age, 24.7 ± 1.7 (SE) yr; height, 176.8 ± 2.1 cm; mass, 78.1 ± 3.5 kg; body surface area, 1.93 ± 0.05 m\textsuperscript{2}; peak oxygen uptake

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247
Cumulative body heat debt was defined as the total negative heat storage integrated over time and expressed as a positive number. Body heat storage (S; W·m⁻²) was calculated: \( S = M - W - L - E - (R + C) \), where M is the metabolic rate, W is work rate (0 in this experiment), L is the respiratory heat losses by convection and evaporation, E is evaporative heat loss (set at 4.1 W·m⁻² in this experiment), K represents conductive heat loss (0 in this experiment), and R + C represents dry heat loss, measured by heat flow disks (8, 26).

Blood was drawn from an indwelling venous catheter (antecubital) in the left arm before the CAT began (minute 0) and at minutes 15, 30, 60, 90, and 120 during CAT. Catheter patency was maintained between blood draws by injecting heparinized saline into the catheter. Blood samples were analyzed to determine plasma glucose concentration by using an autoanalyzer (model 2300, Yellow Springs Instrument) to ensure that subjects maintained euglycemia. Plasma norepinephrine (NE) was determined by gas chromatography (31).

Statistical analyses. Data were analyzed by using a two-way repeated-measures analysis of variance. When significant F-ratios were calculated, paired comparisons were made post hoc by using Newman-Keuls tests. The slope and threshold of each individual's \( T_{re} \) vs. change in M (ΔM) relationship was determined by least-squares linear regression. Paired t-tests were used to determine whether differences in slope or intercept data existed between Ex and Heat for \( T_{re} \), vs. ΔM.

Data are reported as means ± SE. Significance was accepted at \( P < 0.05 \).

RESULTS

Water immersion. All subjects completed 60 min of cycling during Ex. The mean immersion time required during Heat to match the \( T_{re} \) rise observed during Ex was 53.4 ± 5.0 min. The mean \( T_{re} \) at the end of the immersion periods were 38.19 ± 0.14 and 38.08 ± 0.10°C, during Ex and Heat, respectively (\( P > 0.05 \)). The average \( V\dot{O}_2 \) during immersions were 1.97 ± 0.12 and 0.34 ± 0.02 l/min for Ex and Heat, respectively (\( P < 0.05 \)). For Ex, this \( V\dot{O}_2 \) corresponded to 55.4 ± 2.3% of the measured \( V\dot{O}_2\text{peak} \). Final heart rates during immersion were 149.3 ± 6.1 and 102.1 ± 3.1 beats/min for Ex and Heat, respectively (\( P < 0.05 \)). Weight loss from sweat was 1.07 ± 0.15 and 1.06 ± 0.18 kg during Ex and Heat, respectively (\( P > 0.05 \)).

\( T_{re} \) (CAT). During the transition from the immersion pool to the cold-air chamber, \( T_{re} \) fell during Heat. Therefore, \( T_{re} \) at minute 0 was slightly but significantly higher (0.14°C, \( P < 0.05 \)) in Ex vs. Heat (Fig. 1). By minute 10 of cold-air exposure, differences between trials were no longer apparent. However, by minute 40 of CAT, \( T_{re} \) had fallen lower (\( P < 0.05 \)) during Ex compared with Heat, and the difference between trials grew larger as exposure continued to minute 120. The cooling rate from minute 10 to the end of the exposure was faster (\( P < 0.05 \)) for Ex (−0.64 ± 0.07°C/h) than Heat (−0.57 ± 0.04°C/h).

\( T_{sk} \) (CAT). \( T_{sk} \) and the \( T_{re} - T_{sk} \) gradient are shown in Fig. 2. Cold-air exposure caused \( T_{sk} \) to decrease until a new steady-state value of −23°C was achieved. There was a concomitant increase in the \( T_{re} - T_{sk} \) gradient during CAT. The apparent tendency for higher \( T_{sk} \) and lower \( T_{re} - T_{sk} \) in Ex vs. Heat during the last 60 min of
the cold exposure did not achieve statistical significance.

Heat flow (CAT). HF was higher \( (P < 0.05) \) during CAT in Ex vs. Heat (Fig. 3). Also, Iti during CAT was lower \( (P < 0.05) \) in Ex compared with Heat (Fig. 3). Individual site heat flow and Iti are presented in Fig. 4. Calf heat flow and Iti demonstrated a significantly \( (P < 0.05) \) greater heat flow and lower Iti between Ex and Heat. Hand heat flow also tended \( (P = 0.06) \) to be higher in Ex.

\( \dot{M} \) and heat debt (CAT). \( \dot{M} \) did not differ between Ex and Heat at any time throughout CAT. The final \( \dot{M} \) at minute 115 was 146.6 \( \pm \) 6.5 and 136.1 \( \pm \) 3.6 W·m\(^{-2} \) for Ex and Heat, respectively. The relationships (slope and intercept) between \( T_b \) and the corresponding increment in \( \dot{M} \) over pre-CAT values \( (\Delta \dot{M}, \text{a measure of shivering thermogenesis}) \) did not differ between trials. Slopes were \( 33.8 \pm 3.0 \) and \( 32.7 \pm 3.4 \) W·m\(^{-2} \)·°C\(^{-1} \) for Ex and Heat, respectively. Intercepts were \( 34.5 \pm 0.2 \) and \( 34.3 \pm 0.1°C \) for Ex and Heat, respectively. Cumulative heat debt was not different between Ex (547.5 \( \pm \) 47.0 W·m\(^{-2} \)) and Heat (532.9 \( \pm \) 28.5 W·m\(^{-2} \)) after 120 min of exposure.

Plasma glucose and NE (CAT). Plasma glucose concentrations were not affected by CAT in either trial, and there were no differences between trials. Glucose
values averaged between 4 and 6 mmol/l throughout CAT. Plasma NE concentrations increased from 2.5 to 10–15 nmol/l during cold-air exposure, with no differences between Ex and Heat.

Heart rate (CAT). Heart rate tended (P < 0.06) to be higher from minutes 30–75 (~10 beats/min) in Ex compared with Heat.

**DISCUSSION**

This study determined whether exercise predisposes people to experience a greater decline in Tcore during subsequent cold exposure. An expected response to exercise, even in temperate climates, is an increased Tcore (21). Therefore, to isolate effects of body heat content and temperature changes from other exercise effects (thermoregulatory fatigue, thermoregulatory lag, heat redistribution), control experiments were needed in which initial pre-cold exposure Tcore values had been passively elevated to the same value as those measured postexercise. If such controls were not employed, the Tcore-Tsk gradient would be greater during cold exposure after exercise and, subsequently, heat loss would be facilitated. In addition, the absolute Tcore could not be compared between trials beginning with different initial values. However, it is experimentally difficult to match both Tcore and Tsk increases during exercise in air to increases induced by passive heating in air, especially if the durations of the interventions are also desired to be similar. Matching of Tcore and Tsk changes during rest (passive heating) and exercise sessions of similar duration are better accomplished by, using water immersion. The exercise intensity (55% VO2peak) was selected to represent moderately strenuous, fatiguing activities.

The primary finding from this study was that, when individuals exercised before cold exposure, they cooled faster than when rest preceded cold exposure. However, the data are not consistent with our hypothesis that exercise would lead to thermoregulatory fatigue of the shivering response to cold. We had based that hypothesis on findings from our laboratory (2) and those reported by others (18, 19, 25) suggesting that shivering can become fatigued. In this study, the shivering response to cold was the same regardless of whether exercise preceded the cold exposure. In contrast, HF measurements were higher and, concomitantly, tissue insulation less during cold exposure after exercise. Tsk during cold-air exposure also tended to be higher (0.2–0.5°C) after exercise. Collectively, these observations indicate that, after exercise, greater peripheral heat loss from the skin (thermoregulatory lag and/or heat redistribution) was responsible for the greater cooling rates during cold exposure.

Several factors might explain why peripheral heat loss during cold exposure was greater when preceded by exercise than passive heating. One possibility is that postexercise hyperemia in the leg muscles persists during cold exposure, increasing convective heat transfer from the body's core to the periphery overlying active muscle relative to cold exposure preceded by rest (heat redistribution). The higher heat flow and lower insulation in the calf during cold exposure after exercise, compared with passive heating, are consistent with this explanation. Another possibility is that the prior exercise blunted the drive for vasoconstriction normally elicited in response to cold (thermoregulatory lag). However, cold-induced vasoconstriction is sympathetically mediated, and the NE response to cold, considered reflective of sympathetic nervous activation (7), 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 after exercise (12).

Our results contrast with those reported by Kenny et al. (13), who found that the threshold for vasoconstriction was elevated after exercise. They suggested that exercise would result in the retention of heat during subsequent recovery in a cold environment (13). However, our subjects exercised for 1 h in water, whereas those studied by Kenny et al. only completed a short exercise bout (15 min), and thus our subjects may have been more fatigued. In addition, Kenny et al. did not control for differences in initial Tcore before cold exposure, but we matched initial Tcore values before cold exposure between our trials. Finally, our volunteers were subjected to a whole-body cold-air exposure at a

![Fig. 4. Individual heat flow (A) and insulation (B) for 8 sites measured. Calf heat flow was higher (P < 0.05) and insulation lower (P < 0.05) during Ex. Values are means ± SE. *P < 0.05.](image)
constant temperature compared with the water-perfused suit that Kenny et al. used. Thus methodological differences probably account for discrepant observations in our study and that of Kenny et al.

Although we observed a lower $T_{\text{core}}$ when cold exposure followed exercise as well as significantly higher peripheral heat flows and a tendency for higher $M$, compared with cold exposure after passive heating, we found no statistical difference in cumulative heat debt, measured by partitional calorimetry. There was a tendency for an increased $M$ during cold exposure after exercise compared with exposures after passive heating, which probably offset the increased heat flow. Therefore, the greater fall in $T_{\text{core}}$ during cold exposure after exercise may reflect a redistribution of body heat content (14, 15) from the core to the periphery because of a higher peripheral blood flow during, and for some time after, exercise (11).

The absence of an exercise effect on shivering thermogenesis suggests that this response to cold is not easily fatiguable. We observed no difference in the $T_f$ vs. $\Delta M$ relationship between trials, suggesting that the differences in $T_f$ between trials were not due to a change in central control of shivering thermogenesis. Perhaps exercise intensity and duration were not sufficient to fatigue the shivering mechanism, which is a relatively low-intensity activity (30), at least compared with exercise. In Pugh’s (18) case report of the Four Inns Walk, the participants were exercising up to 20 h in cold-wet conditions. Similarly, the subject in Thompson and Hayward’s study (25) 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 (9, 17). Plasma glucose levels were not measured in those previous studies (18, 25). In our study, plasma glucose concentrations remained normal throughout cold exposure.

A possible limitation to extrapolating our results to nonimmersed exercise relates to the potential effects of immersion, especially immersion-associated alterations in hormonal responses to exercise compared with exercise in air. However, during cycle exercise at $\sim 60\% V_{\text{O}_2 \text{peak}}$, performed in water up to the neck, there was no difference in catecholamine responses compared with cycling in air at the same intensity (4). Another study (22) also demonstrated that plasma osmolality, which is known to affect central temperature regulation (23), was not different during exercise at $60\% V_{\text{O}_2 \text{peak}}$ in water and air. In fact, hormonal responses to exercise have not been found to differ between air and immersion (22), except that plasma renin activity was lower and plasma atrial natriuretic peptide was higher during water exercise vs. air exercise at $60\% V_{\text{O}_2 \text{peak}}$. However, there is no known influence of renin and atrial natriuretic peptide on hypothalamic neurons regulating thermoregulatory responses to cold. Studies comparing thermoregulatory responses to cold subsequent to exercise in air are warranted to confirm our findings. However, it seems reasonable to conclude that our data indicate that, after exercise, the ability to maintain thermal balance in the cold may be compromised.

This study was the first to examine the possibility that acute exercise performed before whole-body cold-air exposure impairs the ability to maintain thermal balance, as others have speculated. Our findings demonstrated that exercise before cold-air exposure may lead to a greater fall in $T_{\text{core}}$ due to reduced insulation and increased heat loss and a redistribution of heat from the core to the periphery. The data also suggest that an exercise-related factor (heat redistribution) led to the greater fall in $T_{\text{core}}$ and not the rise in $T_{\text{core}}$ that accompanies exercise. These findings may also have potential implications for people who exercise hard and are then exposed to cold stress, or people who exercise hard outdoors in the cold and then stop, but do not return indoors immediately.

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