Moderate exercise increases postexercise thresholds for vasoconstriction and shivering

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1Faculty of Health Sciences, School of Human Kinetics, University of Ottawa, Ottawa, Ontario K1N 6N5; and 2Laboratory for Exercise and Environmental Medicine, Health, Leisure and Human Performance Research Institute, University of Manitoba, Winnipeg, Manitoba, Canada R3T 2N2

Kenny, G. P., A. A. Chen, B. A. Nurbakhsh, P. M. Denis, C. E. Proulx, and G. G. Giesbrecht. Moderate exercise increases the postexercise thresholds for vasoconstriction and shivering. J. Appl. Physiol. 85(4): 1357–1361, 1998.—The purpose of this study was to evaluate the effect of exercise on the subsequent postexercise thresholds for vasoconstriction and shivering. On two separate days, with six subjects (3 women), a whole body water-perfused suit slowly decreased mean skin temperature (~7.0°C/h) until thresholds for vasoconstriction and shivering were clearly established. Subjects were then rewarmed by increasing water temperature until both esophageal and mean skin temperatures returned to near-baseline values. Subjects either performed 15 min of cycle ergometry (65% maximal O2 consumption) followed by 30 min of recovery (Exercise) or remained seated with no exercise for 45 min (Control). Subjects were then cooled again. We mathematically compensated for changes in skin temperatures by using the established linear cutaneous contribution of skin to the control of vasosconstriction and shivering (20%). The calculated core temperature threshold (at a designated skin temperature of 30.0°C) for vasoconstriction increased significantly from 36.64 ± 0.20 to 36.89 ± 0.22°C postexercise (P < 0.01). Similarly, the shivering threshold increased from 35.73 ± 0.13 to 36.13 ± 0.12°C postexercise (P < 0.01). In contrast, sequential measurements, without exercise, demonstrate a time-dependent decrease in both the vasoconstriction (0.10°C) and shivering (0.12°C) thresholds. These data indicate that exercise has a prolonged effect by increasing the postexercise thresholds for both cold thermoregulatory responses.

WE HAVE PREVIOUSLY DEMONSTRATED THAT AFTER 15 MIN OF MODERATE EXERCISE, SKIN BLOOD FLOW AND MEAN SKIN TEMPERATURES (Tsk,avg), AT ALL SITES EXCEPT OVER THE EXERCISED MUSCLE (I.E., THIGH AND CALF) RETURN TO BASELINE VALUES WITHIN 15–20 MIN AFTER EXERCISE, DESPITE THE SUSTAINED INCREASE IN ESOPHAGEAL TEMPERATURE (Tes) (27). THESE RESULTS ARE CONSISTENT WITH A POSTEXERCISE INCREASE IN THE THRESHOLD FOR ACTIVE VASODILATION DURING RECOVERY. THIS RESIDUAL EFFECT IS NOT A RESULT OF THE EXERCISE-INDUCED ELEVATION OF WHOLE BODY HEAT CONTENT ALONE, BECAUSE Tes WAS SHOWN TO RETURN TO BASELINE REST AFTER WARM-WATER IMMERSION (12). THE POSTEXERCISE INCREASE IN CORE TEMPERATURE IS LIKELY DUE TO RESIDUAL EXERCISE-RELATED FACTORS [I.E., ENDOCRINE CHANGES (5), ENDODERGIC METABOLIC BYPRODUCTS (2, 5), OR BAROREFLEX ACTIVITY (7, 9) LIKELY EXERT THE RESIDUAL THERMOREGULATORY EFFECTS].

WE HAVE ALSO STUDIED THE EFFECTS OF INTENSE EXERCISE ON SWEATING. ALTHOUGH THE SWEATING THRESHOLD DECREASED DURING EXERCISE, IT ACTUALLY INCREASED TO 0.3°C ABOVE BASELINE VALUES AFTER EXERCISE (11). ALTHOUGH OUR COMBINED DATA INDICATE THAT EXERCISE-RELATED FACTORS CAUSE A POSTEXERCISE INCREASE IN THRESHOLDS FOR BOTH WARM THERMOREGULATORY RESPONSES, THE EFFECTS ON COLD THERMOREGULATORY RESPONSES (I.E., VASOCONSTRICTION AND SHIVERING) ARE UNKNOWN. THIS INFORMATION MAY HAVE IMPORTANT IMPLICATIONS FOR EXERCISE RECOVERY UNDER DIFFERENT ENVIRONMENTAL CONDITIONS.

IF THE POSTEXERCISE EFFECT WAS A GENERAL THERMOREGULATORY INHIBITION (AS WITH MOST GENERAL ANESTHETICS), COLD RESPONSE THRESHOLDS WOULD DECREASE, THUS WIDENING THE INTERTHRESHOLD RANGE [DEFINED AS THE CORE TEMPERATURE RANGE BETWEEN THE SWEATING AND VASOCONSTRICTION THRESHOLDS (22)]. ON THE OTHER HAND, THESE THRESHOLDS WOULD INCREASE IF EXERCISE CAUSED A GENERAL RISE IN REGULATED CORE TEMPERATURE, AS SEEN IN OUR PREVIOUS WORK (13, 27). THE PRESENT STUDY, THEREFORE, EVALUATES THE HYPOTHESIS THAT MODERATE EXERCISE CAUSES A RESIDUAL INCREASE IN THE SUBSEQUENT POSTEXERCISE THRESHOLDS FOR BOTH VASOCONSTRICTION AND SHIVERING.

METHODS

Subjects. With approval from our Faculty Human Ethics Committee, six healthy subjects (3 men, 3 women) with no history of cardiovascular or respiratory disease, participated after providing written, informed consent. Subjects were physically active, although none engaged in daily or intensive training programs. Subjects were 23 ± 4 (SD) yr old, 1.7 ± 0.1 m tall, and weighed 74 ± 3 kg. Female subjects were eumenorrheic with regular, ~28-day-long menstrual cycles. To control for hormonal effects, female subjects were studied within 9 days after start of menstruation (follicular phase).

Instrumentation. Core temperature was measured by using an esophageal thermocouple inserted through a nostril to the level of the heart. Skin temperature was monitored at 11 sites by heat-flow sensors (Concept Engineering, Old Saybrook, CT), and the area-weighted mean (i.e., Tsk,avg) was calculated by assigning the following regional percentages: head 6%, upper arm 9%, forearm 6%, finger 2%, chest 19%, upper back 9.5%, lower back 9.5%, anterior thigh 10%, posterior thigh 10%, anterior calf 9.5%, and posterior calf 9.5%.

Fingertip blood flow was assessed with a laser Doppler flow probe placed on the middle digit (blood perfusion monitor,

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Tal maximal $\dot{V}O_2$ test on a cycle ergometer on the first day. The Packard, model PC-312, 9000). Oxygen consumption ($V\dot{O}_2$) was determined by an open-circuit method from measurements of expired minute volume and inspired and mixed expired gas concentrations sampled from a 10-liter fluted mixing box.

Temperatures were collected and digitized (Hewlett-Packard data-acquisition module, model 3497A) at 5-s intervals, displayed graphically on the computer screen, and recorded in spreadsheet format on a hard disk (Hewlett-Packard, model PC-312, 9000).

Experimental protocol. Subjects performed one incremental maximal $V\dot{O}_2$ test on a cycle ergometer on the first day. The two experimental trials were conducted in either the morning (3 subjects) or midafternoon (3 subjects) after a 24-h period without heavy or prolonged physical activity, the last 12 h of which included abstinence from stimulants and alcohol, 8 h of sleep, and a minimum of 0.25 liters of water during each waking hour. On each study day, care was taken to avoid major thermal stimuli or substantial increase in metabolic rate between awakening and the start of the experiment.

On arrival at the laboratory, subjects were clothed in shorts and running shoes and were instrumented appropriately. They were then outfitted with a water-perfusion suit and seated in a climatically controlled chamber. Baseline data were collected over 20 min at an ambient temperature ($T_a$) of 24°C (Fig. 1). Warm water (~40°C) was then circulated through the water-perfused suit until cutaneous vasodilation occurred (~10 min). $T_{sk,avg}$ was then decreased at a constant rate of ~7.0°C/h as the temperature of the water perfusing the suit was progressively lowered until vasoconstriction and vigorous shivering occurred. $T_{es}$ and $T_{sk,avg}$ were subsequently increased to near-baseline values by perfusing the suit with warm water (~15 min) and increasing $T_a$ to 29°C. Water circulation in the suit was then stopped (until the second warming period), and $T_a$ was reduced to 24°C for the duration of the trial. Subjects then either exercised on a cycle ergometer (65% maximal $V\dot{O}_2$) for 15 min (Exercise) followed by 30-min recovery, or remained seated for 45 min (Control). Warm water (~40°C) was then circulated through the water-perfused suit until cutaneous vasodilation occurred (~10 min). Subjects were then cooled a second time until vasoconstriction and vigorous shivering occurred.

The threshold for vasoconstriction was defined as the point at which fingertip blood flow reached a minimum (14). The shivering threshold was indicated by a sustained 40% increase in $V\dot{O}_2$ above the baseline level (17). This method for determination of the shivering threshold has been validated in our laboratory against the subjects’ self-report of shivering onset and an increase in electromyographic activity (unpublished data). To compare thresholds between conditions in which both esophageal and $T_{sk,avg}$ were changing, the following equation (16) was used to correct the $T_{es}$ ($T_{es,calc}$) for a designated skin temperature ($T_{sk,des}$)

$$T_{es,calc} = T_{es} + (\beta/(1 - \beta))(T_{sk,avg} - T_{sk,des})$$

where $T_{sk,des}$ was set as the $T_{sk,avg}$ of Pre- and Postcontrol and Pre- and Postexercise Cooling conditions (30°C), and $\beta$ is the fractional contribution of the skin to the vasoconstriction and shivering response (0.2) (3).

Analysis of results. For the purpose of comparison, thermoregulatory response thresholds were identified as 1) Pre- and Postcontrol Cooling and 2) Pre- and Postexercise Cooling for the Control and Exercise conditions, respectively. ANOVA for repeated measures was used to test for significant intra- and intercondition differences in $T_{sk,avg}$, $T_{es}$, and $T_{es,calc}$ at each cold response threshold. In the Control trial, mean data were compared for the final 5 min of the following periods: baseline 1; warming before baseline 2; and baseline 2. In the Exercise trial, mean data were compared for the final 5 min of baseline 1, Preexercise warming, and baseline 2 periods as well as the final minute of Exercise. In the event of statistical significance ($P < 0.05$), Tukey’s test was used to identify significant differences. Data are presented as means ± SD.

![Fig. 1. Protocols for Control (A) and Exercise (B) trials identified by esophageal temperature ($T_{es}$) changes in 1 subject. Small open bars, periods of data compared in analyses; VC and SH, onset of vasoconstriction and shivering, respectively. Note that, although length of cooling periods varies, time between end of 1st cooling period and start of 2nd period is similar in Control and Exercise protocols. Ex, exercise.](image-url)
RESULTS

Suit perfusate was cooled at the same rate in both Pre- and Postexercise Cooling periods (Table 1). Thus $T_{sk,avg}$ and $T_{es}$ decreased at the same respective rates in both conditions.

Baseline $T_{es}$ (37.1 ± 0.2 and 37.2 ± 0.1°C for Control and Exercise, respectively) and $T_{sk,avg}$ (33.2 ± 0.6 and 33.9 ± 0.5°C for Control and Exercise, respectively) remained stable and consistent during the 15-min period before surface warming. $T_{es}$ during surface warming remained stable in both conditions and showed a gradual decrease during the initial surface cooling. $T_{es}$, $T_{sk,avg}$, and cutaneous blood flow returned to resting values within ~15 min of surface warming. These values remained stable for the duration of the 45-min seated rest during the Control trial. For the Exercise trial, $T_{es}$ increased to an end-of-exercise value of 38.0 ± 0.2°C during Exercise and decreased to an elevated value of 37.5 ± 0.1°C within 30 min of the Postexercise period. This value was significantly higher (0.4°C) than the Preexercise value ($P < 0.05$). $T_{sk,avg}$ and fingertip blood flow returned to baseline values within 15–20 min of the 30-min Postexercise recovery period. $T_{es}$ during surface warming remained stable in both conditions and showed a gradual decrease during the second surface cooling.

Control: effect of time on cold response thresholds. $T_{sk,avg}$, $T_{es}$, and $T_{es,calc}$, at the onset of vasoconstriction and shivering, are presented in Table 1. Figure 2 shows the individual and mean corrected core temperatures for Pre- and Postcontrol Cooling at the thresholds for vasoconstriction (36.78 ± 0.13 and 36.68 ± 0.14°C, respectively) and shivering (35.84 ± 0.15 and 35.72 ± 0.23°C, respectively). On average, vasoconstriction and shivering onset occurred successively at 36 ± 5 and 45 ± 5 min after initiation of cooling, respectively. In all of our subjects, onset of vasoconstriction occurred at a slightly lower (0.10°C) corrected core temperature during the second period of cooling ($P < 0.05$). Shivering onset showed a comparable decrease (0.12°C) in four of the six subjects.

Table 1. Cooling parameters

<table>
<thead>
<tr>
<th>Precontrol Cooling</th>
<th>Postcontrol Cooling</th>
<th>Preexercise Cooling</th>
<th>Postexercise Cooling</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rate of cooling, °C/h</td>
<td></td>
<td></td>
</tr>
<tr>
<td>$T_{water}$</td>
<td>20 ± 1</td>
<td>19 ± 2</td>
<td>19 ± 1</td>
</tr>
<tr>
<td>$T_{es}$</td>
<td>0.42 ± 0.19</td>
<td>0.43 ± 0.23</td>
<td>0.42 ± 0.21</td>
</tr>
<tr>
<td>$T_{sk,avg}$</td>
<td>6.67 ± 0.41</td>
<td>7.50 ± 0.49</td>
<td>6.87 ± 0.25</td>
</tr>
<tr>
<td></td>
<td>Onset of vasoconstriction, °C</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Actual $T_{sk,avg}$</td>
<td>31.58 ± 0.26</td>
<td>31.48 ± 0.23</td>
<td>31.28 ± 0.26</td>
</tr>
<tr>
<td>Actual $T_{es}$</td>
<td>37.14 ± 0.13</td>
<td>37.06 ± 0.10*</td>
<td>37.07 ± 0.12</td>
</tr>
<tr>
<td>$T_{es,calc}$</td>
<td>36.78 ± 0.13</td>
<td>36.68 ± 0.14*</td>
<td>36.64 ± 0.20</td>
</tr>
<tr>
<td></td>
<td>Onset of shivering, °C</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Actual $T_{sk,avg}$</td>
<td>28.88 ± 0.09</td>
<td>28.88 ± 0.28</td>
<td>28.82 ± 0.30</td>
</tr>
<tr>
<td>Actual $T_{es}$</td>
<td>36.87 ± 0.15†</td>
<td>36.75 ± 0.15*</td>
<td>36.78 ± 0.13*</td>
</tr>
<tr>
<td>$T_{es,calc}$</td>
<td>35.84 ± 0.15</td>
<td>35.72 ± 0.23</td>
<td>35.73 ± 0.13</td>
</tr>
</tbody>
</table>

Values are means ± SD; n = 6 subjects. $T_{water}$, $T_{es}$, $T_{sk,avg}$, and $T_{es,calc}$: Water, esophageal, average skin, and calculated esophageal temperature, respectively. *Significantly different temperature from Precontrol Cooling, $P < 0.05$. †Significantly different temperature from Preexercise Cooling, $P < 0.05$.

DISCUSSION

This is the first human study to address the residual effects of moderate exercise on thermoregulatory cold response thresholds. As long as 1–1.5 h Postexercise, there was a parallel increase in thresholds for vasoconstriction and shivering by 0.3 and 0.4°C, respectively. This effect is similar to the 0.3°C increase in the Postexercise threshold for sweating (11). Our results also agree with studies in mice that demonstrated that 1 h of exercise, before 3-h exposure to 6°C air, increases cold tolerance through an increase in peripheral vasoconstriction (this is consistent with an increase in the vasoconstriction threshold) (23). Although this exercise-related effect was only seen in the mice tested in the afternoon, our Postexercise increase in cold response thresholds was seen in subjects tested in both morning and afternoon trials. In contrast to the Postexercise increase in thermal cold response thresholds, sequential measurements demonstrated a time-dependent decrease in both onset of vasoconstriction and shivering (0.1°C) responses comparable to the decrease in sweating thresholds (0.1°C) demonstrated by Brengelmann et al. (1).

Possible mechanisms for results. The increase in thresholds was not caused by differences in rate of change of $T_{es}$ or $T_{sk,avg}$ because cooling rates were
virtually the same for the Pre- and Postexercise Cooling periods. Cold response thresholds could increase if the total integrated thermal signal at a given core temperature decreased. This is unlikely in the present protocol because total heat content of the body after exercise would actually be greater than during the Preexercise period.

The prolonged nature of the protocol was also unlikely to contribute to the elevated cold response thresholds during the second cooling period. Brengelmann et al. (1) made sequential measurements and actually demonstrated a time-dependent decrease in sweating thresholds over a 2-h period. The similar time-dependent increase that we demonstrated for cold responses would result in an underestimation of the increase in cold response thresholds in the present study.

Our laboratory has previously demonstrated that $T_{es}$ remains elevated by ~0.5°C for at least 65 min after intense exercise, even though $T_{sk,avg}$ and skin blood flow rapidly returned to preexercise values (27). These data are consistent with an elevated vasodilation threshold during this period. It has also been shown that an increase in heat content alone (i.e., with warm-water immersion) was not the primary mechanism for an elevated vasodilation threshold and persistent elevation of $T_{es}$ (12). Although it is unlikely that heat content was responsible for the increase in the thresholds for vasoconstriction and shivering, this factor cannot be discounted without further study. However, it is plausible that some exercise-related factor(s), such as endocrine changes (5), endogenous pyrogen, metabolic by-products (2, 5), or baroreflex activity (7, 9), likely exerts the residual thermoregulatory effects.

Reflex control of the cutaneous circulation involves both sympathetic active vasoconstrictor and active vasodilatory systems (8). The degree to which either system affects skin blood flow at rest and exercise differs greatly. Whole body heating during rest increases skin blood flow by decreasing active vasoconstriction (20), whereas an increase in skin blood flow during exercise is due to an increase in the active vasodilatory response (10). It remains unclear how skin blood flow is controlled during recovery. It is known, however, that the cutaneous vasodilator system is under baroreceptor control (9). Acute bouts of exercise have been shown to cause postexercise hypotension (4), likely due to a decrease in baroreflex sensitivity (24). A decrease in skin blood flow (i.e., vasoconstriction) would help maintain adequate filling pressure in response to the decrease in systemic vascular resistance. Thus this nonthermal factor may influence the elevated threshold for vasoconstriction during exercise recovery.

An increase in the postexercise threshold for sweating has previously been demonstrated (11). Our present
data demonstrate parallel increases in both cold response thresholds. These data are not consistent with an exercise-induced general inhibition of thermoregulatory control, as often occurs with general anesthesia, where warm response thresholds increase but cold response thresholds decrease (22). The parallel increase in cold and warm response thresholds is consistent with either 1) an increase in set-point control with a similar tolerance for core temperature displacement before heat loss or heat gain responses are initiated (6, 18, 21, 26); or 2) separate but parallel increases in each individual response threshold (15).

We conclude that a residual exercise-related factor(s) increases the postexercise vasoconstriction and shivering thresholds. The increased vasoconstriction threshold would result in the retention of heat produced during exercise for a longer period of recovery. This could be a teological advantage when recovery occurs in a cold environment. These findings have a practical implication in thermoregulation studies that employ exercise for manipulation of core temperature. Consideration should be given to other methods, such as surface heating and cooling, or IV infusion of saline at different temperatures (22).

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