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# Thermal behavior remains engaged following exercise despite autonomic thermoeffector withdrawal

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ABSTRACT

We tested the hypothesis that thermal behavior during the exercise recovery compensates for elevated core temperatures despite autonomic thermoeffector withdrawal. In a thermoneutral environment, 6 females and 6 males (22 ± 1 y) cycled for 60 min (225 ± 46 W metabolic heat production), followed by 60 min passive recovery. Mean skin and core temperatures, skin blood flow, and local sweat rate were measured continually. Subjects controlled the temperature of their dorsal neck to perceived thermal comfort using a custom-made neck device. Neck device temperature provided an index of thermal behavior. Mean body temperature, calculated as the average of mean skin and core temperatures, provided an index of the stimulus for thermal behavior. To isolate the independent effect of exercise on thermal behavior during recovery, data were analyzed post-exercise the exact minute mean body temperature recovered to pre-exercise levels within a subject. Mean body temperature returned to pre-exercise levels 28 ± 20 min into recovery (Pre: 33.5 ± 0.2, Post: 33.5 ± 0.2 °C, P = 0.20), at which point, mean skin temperature had recovered (Pre: 29.6 ± 0.4, Post: 29.5 ± 0.5 °C, P = 0.20) and core temperature (Pre: 37.3 ± 0.2, Post: 37.5 ± 0.3 °C, P = 0.01) remained elevated. Post-exercise, skin blood flow (Pre: 59 ± 78, Post: 26 ± 25 PU, P = 0.10) and local sweat rate (Pre: 0.05 ± 0.25, Post: 0.13 ± 0.14 mg/cm$^2$ min$^{-1}$, P = 0.09) returned to pre-exercise levels, while neck device temperature was depressed (Pre: 27.4 ± 1.1, Post: 21.6 ± 7.4 °C, P = 0.03). These findings suggest that thermal behavior compensates for autonomic thermoeffector withdrawal in the presence of elevated core temperatures post-exercise.

## 1. Introduction

During the transition from rest to exercise, autonomic heat loss thermoeffectors (e.g., skin blood flow and sweating) are recruited in proportion to the rate of metabolic heat production and subsequent increases in core and skin temperatures [1]. During low intensity, steady state exercise in a moderate thermal environment, increases in skin temperature are modest (increase of ~1 °C) and occur mostly as a result of increases in skin blood flow [2]. Similarly, thermal behavior is believed to be recruited in proportion to the magnitude of thermal discomfort arising from changes in skin and core temperatures [3–5]. During rest, skin and core temperatures are equally weighted for producing thermal discomfort [6,7]. Because thermal discomfort is the perceptual mediator of thermal behavior [8] it is often posited that skin and core temperatures equally contribute to the decision to behaviorally thermoregulate [6,7]. Notably however, the relative contributions of skin and core temperatures to thermal behavior during exercise have never been quantified.

During the transition from exercise to recovery, autonomic thermoeffectors and skin temperature rapidly return to pre-exercise levels [9]. Paradoxically, core temperature remains elevated during this time [9]. The sustained elevation of core temperature is largely attributed to the rapid withdrawal of autonomic thermoeffectors, including reduced blood flow, which ultimately results in a reduction in skin temperature post exercise [9,10]. This response, however, eliminates heat loss, thereby trapping heat in the core and musculature, and occurs independent of exercise intensity and ambient conditions [9]. To the best of our knowledge, the effect of exercise recovery on thermal behavior is unknown. It is possible that thermal behavior compensates for the disconnect between autonomic thermoeffector deactivation and prevailing elevations in core temperature post-exercise. On the other hand, thermal behavior may parallel the autonomic thermoeffector response

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with quick restoration to pre-exercise levels. Moreover, the relative contributions of skin and core temperatures to thermal behavior during recovery from exercise is also unknown.

The purposes of this study were twofold. First, we aimed to determine the effects of exercise on thermal behavior post-exercise. We hypothesized that thermal behavior during recovery would compensate for a sustained elevation in core temperature in the absence of autonomic thermoeffector activation. Second, we aimed to identify the relative contributions of skin and core temperatures to thermal behavior during exercise and recovery. We hypothesized that during exercise, skin and core temperatures would contribute equally to thermal behavior, but during recovery, core temperature would contribute more to thermal behavior. The testing of these hypotheses will provide unique insights regarding interactions between autonomic and behavioral thermoeffectors during and following exercise. Such an understanding can potentially improve comfort and safety during and following exercise in both healthy populations and/or those demonstrating abnormal thermoregulation. This should be considered particularly important given the virtually unlimited ability of behavior to restore and maintain thermal comfort.

## 2. Methods

### 2.1. Subjects

Twelve young, healthy adults participated in this study (6 females). The subject characteristics are listed in Table 1. All subjects were physically active, normotensive, non-smokers, not taking medications, cognitively normal, and free from any known cardiovascular, metabolic, neurological or psychological diseases. Female subjects were not pregnant, which was confirmed via a urine pregnancy test, and reported to be normally menstruating. Each subject was fully informed of the experimental procedures and possible risks before giving informed written consent. The study was approved by the Institutional Review Board at the University at Buffalo, and performed in accordance with the standards set by the latest revision of the declaration of Helsinki.

### 2.2. Study design

Subjects visited the laboratory on two occasions. Visit one was a screening and familiarization visit and visit two was the experimental trial. During the experimental trial, subjects arrived at the laboratory euhydrated, confirmed via urine specific gravity $< 1.020$ (actual urine specific gravity: $1.010 \pm 0.007$) and having refrained from strenuous exercise, alcohol and caffeine for 12 h, and food for 2 h. To control for menstrual cycle hormones, females were tested during the first 10 days following self-identified menstruation or during the placebo phase of their oral contraceptives, a period in which estrogen and progesterone are at their lowest levels. All experimental testing was conducted during the summer months in Buffalo, NY (mean outside temperature during experimental trials: $20 \pm 4$ °C). Subjects wore a standard short sleeved t-shirt and shorts (lululemon, Vancouver, BC, Canada), and their own socks and athletic shoes (~0.4 clo).

**Table 1**
Subject characteristics, mean ± SD (range).

| Number of subjects | 12 (6 females) |
|---|---|
| Age (y) | 23 ± 2 (20–29) |
| Height (cm) | 170 ± 8 (152–180) |
| Weight (kg) | 70.5 ± 9.1 (55.1–88.7) |
| Body surface area ($m^2$) | 1.8 ± 0.2 (1.5–2.0) |
| Body fat (%) | 22 ± 6 (6–34) |
| International Physical activity Questionnaire (high/moderate/low)[a] | 7/5/0 |
| Montreal Cognitive Assessment Score[b] | 29 ± 1 (26–30) |

[a] Stratified according to Craig et al. [15].
[b] All subjects were in the normal range for age group (≥25 points) [43].

### 2.3. Instrumentation and measurements

Height and weight were measured with a stadiometer and scale (Sartorius Corp. Bohemia, NY, USA), and body surface area was calculated accordingly [11]. Skinfold thickness was measured in triplicate at the chest, axilla, triceps, subscapula, abdomen, suprailliac, and thigh (Harpenden, Baty International, UK), and percent body fat was estimated from body density [12], which was calculated from the sum of skinfolds for males [13] and females [14]. Physical activity level was estimated using the validated International Physical Activity Questionnaire [15]. Cognitive ability was measured using the Montreal Cognitive Assessment [16].

All measures of temperature were obtained using thermocouples (Omega Engineering, Inc. Stamford, CT, USA), unless otherwise noted. Mean skin temperature was measured as the equally weighted average of ten thermocouples attached to the right side of the body on the shin, posterior calf, posterior thigh, anterior thigh, abdomen, chest, scapula, hand, triceps and forehead [17]. This ten site mean skin temperature calculation was chosen based on the recent recommendation that ten sites are most appropriate for studies examining thermal comfort [18]. Approximately 60 min prior to experimental testing, subjects swallowed a telemetry pill (HQ Inc., Palmetto, FL, USA) for measurement of core temperature. Mean skin and core temperatures are evenly weighted for producing thermal discomfort [6,7]. Thus, mean body temperature was calculated as $0.5 \times$ core temperature $+ 0.5 \times$ mean skin temperature. This provided an index of thermal afferent feedback stimulating thermal behavior, as has been recently employed [19].

Heart rate was continually measured via a 3-lead ECG (DA100C, Biopac Systems, Inc. Goleta, CA, USA). Skin blood flow was measured continually via integrated laser Doppler flowmetry (Periflux System 5010, Perimed, Stockholm, Sweden) on the dorsal surface of the right forearm. Skin blood flow was measured at this location and not under the clothing to remove any potential influence of the laser Doppler probes and wires from interfering with the skin to clothing interface. The arm was stabilized and held in the same position throughout the protocol. Blood pressure was measured using electro-sphygmomanometry (Suntech, Tango M2, Raleigh, NC, USA) on the left arm every 10 min. Mean arterial pressure was calculated as double the diastolic blood pressure value added to the systolic blood pressure value and divided by three. Skin blood flow data are presented as the change in absolute values and were also normalized to blood pressure, providing an index of cutaneous vascular conductance, which is indicative of changes in cutaneous vasomotor tone.

Local sweat rate was measured by securing a plastic capsule that covers 3.9 $cm^2$ of skin on the axilla and upper arm on the right side of the body. These capsules were perfused with dry nitrogen at a flow rate of 0.5 L/min. The water vapor of the gas exiting the capsules was continuously measured by capacitance hygrometry (HMT130, Vaisala, Woburn, WA, USA), and local sweat rate was calculated by multiplying the humidity output by flow rate and dividing that value by the surface area of the capsule [20]. The local sweat rates are reported as an average of these two locations.

Metabolic data were taken every 30 min during the pre-exercise, exercise and post-exercise periods, and were obtained via a mouth piece and one-way non-rebreathing valve (Han Rudoph, Inc., Shawnee, KS, USA). Minute ventilation was calculated from expired airflow that was measured via a heated pneumotachometer (Hans Rudolph, Inc. Shawnee, KS, USA), which was continually integrated over 30 s and corrected to standard temperature pressure, dry (STPD). The fraction of expired oxygen and carbon dioxide (Vacumed, Ventura, CA, USA) was continually measured from a 3 L mixing chamber. Oxygen uptake and carbon dioxide production were calculated using the Haldane Transformation. The rate of metabolic heat production was calculated

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