Physiology & Behavior 105 (2012) 1194–1201

Contents lists available at SciVerse ScienceDirect

Physiology & Behavior

journal homepage: www.elsevier.com/locate/phb

The effect of a covert manipulation of ambient temperature on heat storage and voluntary exercise intensity

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ARTICLE INFO

Article history:
Received 1 August 2011
Received in revised form 16 December 2011
Accepted 16 December 2011

Keywords:
Exercise control
Heat stress
Power output
Thermal behavior
ARIMA

ABSTRACT

The modulation of sub-maximal voluntary exercise intensity during heat stress has been suggested as a behavioral response to maintain homeostasis; however, the relationship between thermophysiological cues and the associated response remains unclear. Awareness of an environmental manipulation may influence anticipatory planning before the start of exercise, making it difficult to isolate the dynamic integration of thermophysiological afferents during exercise itself. The purpose of the present study was to examine the direct real-time relationship between thermophysiological afferents and the behavioral response of voluntary exercise intensity. Participants were tasked with cycling at a constant rating of perceived exertion while ambient temperature (T_a) was covertly changed from 20 °C to 35 °C and then back to 20 °C at 20-minute intervals. Overall, power output (PO) and heat storage, quantified using repeated measures ANOVA, changed significantly over 20-minute intervals (135 ± 39 W, 133 ± 46 W, 120 ± 45 W; 52.35 ± 36.15 W·m^{-2}, 66.34 ± 22.02 W·m^{-2}, -66.53 ± 56.01 W·m^{-2}). The synchronicity of PO fluctuations with changes in thermophysiological status was quantified using Auto-Regressive Integrated Moving Average (ARIMA) time series analysis. Fluctuations in PO were not synchronized in real time with changes in T_a; heat storage; rectal, skin, or mean body temperature; or sweat rate (stationary-$r^2 \leq 0.10$ and Ljung–Box statistic > 0.05 for all variables). We conclude that, while the thermal environment affects physiological responses and voluntary power output while cycling at a constant perceived effort, the behavioral response of voluntary exercise intensity did not depend on a direct response to real-time integration of thermal afferent inputs.

1. Introduction

It is widely accepted that ambient temperature (T_a) can dramatically alter maximal performance capacity, with Galloway and Maughan [1] demonstrating a significant and progressive reduction in tolerance time to exhaustion with increasing ambient temperatures from 11 to 31 °C. Many thermophysiological factors can directly alter physiological capacity and elicit this earlier onset of fatigue during exercise to exhaustion [2,3]. For example, elevated core temperature directly reduces voluntary neuromuscular activation [4,5] independent of local muscle temperature. In turn, such reduced physiological capacity may also work in conjunction with reduced mental arousal [6] and altered brain neurochemistry [7]. Similarly, cardiovascular strain is elevated, with increased cutaneous vasculature conductance [8] and decreased plasma volume [9], combined with an increased demand in nutrient supply and waste removal from working musculature [10]. Thus, the accumulation of additional cardiovascular strain caused by these physiological events disrupts homeostasis compared to similar exercise in cooler environments and can contribute to premature fatigue.

Increased ambient temperature can also reduce voluntary and self-paced exercise intensity in submaximal exercise situations where individuals do not exercise to exhaustion or reach physiological collapse, with warmer temperatures reducing marathon running times across all fitness levels [11] and degrading performance in a short time trial even without significant hyperthermia [12]. This suggests that voluntary fluctuations in PO in the heat is a behavioral response to maintain homeostasis [13], but what remains unknown is how thermophysiological cues are integrated, if at all, into this behavioral response of voluntary exercise. Much of the research in this field has focused on isolating and testing a single thermophysiological afferent. Thus, one model argues that changes in mean body temperature or perturbations in the rate of heat storage ($\dot{S}$) mediates an anticipatory down-regulation in exercise intensity so that excessive heat accumulation does not occur and physiological homeostasis is maintained [14]; however, this conclusion may be confounded by the use of thermometric calculations for $\dot{S}$ [15]. Other proposed

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doi:10.1016/j.physbeh.2011.12.017

afferents include skin temperature changes eliciting thermal discomfort and causing a decrease of voluntary exercise, thus defending core temperature [16–18]. For example, voluntary power output during a time trial was increased with lower skin temperatures even though core temperature remained similar [16].

The majority of research utilizes an approach that permits the individual to self-pace their exercise in different environmental conditions, and then to see whether any thermophysiological afferents control the observed changes. While intriguing, the primary design consideration that has not been met to date involves eliminating psychological mechanisms such as prior knowledge of the task or knowledge of a thermal manipulation, which may cause the adoption of an exercise strategy based on a preconceived performance template that may override physiological cues. In turn, this will limit an experiment's potential towards quantifying the true conscious and real-time control of exercise intensity. Although it is extremely difficult to blind subjects to the presence of an environmental manipulation, deception can be employed to minimize any possible psycho-physiological mechanisms of prior knowledge involved with this model [19].

Therefore, the purpose of the present study was to examine the direct and real-time relationship between multiple thermophysiological afferent inputs and the behavioral effector response of voluntary exercise intensity while attempting to control for psychological mechanisms. This was accomplished by deceiving participants to the true purpose of the study and the presence of temperature changes in order to remove the conscious pre-planning of performance, and then covertly manipulating ambient temperature (T_a) during self-paced cycling at a set rating of perceived exertion. We hypothesized that the change in T_a would force subconscious changes in one or more thermophysiological afferents, such as heat storage, core or skin temperature, sweat rate, or heart rate, and that these changes would elicit synchronous alterations in voluntary power output (PO) in order to maintain thermal homeostasis.

2. Methods

2.1. Subjects

The experimental protocol and instrumentation conformed to the standards set by the *Declaration of Helsinki* and was approved by the Research Ethics Board of Brock University. Twenty healthy participants (fifteen male and five female) were recruited from the local cycling club and university community, and provided initial written consent prior to the experiment. The mean (±SD) age, height, weight, body fat percentage, peak oxygen uptake and peak PO of all participants was 33.7 ± 14 years, 174.7 ± 10.2 cm, 73.1 ± 13.5 kg, 12.8 ± 6.1%, 56.8 ± 9.3 $mL \cdot kg^{-1} \cdot min^{-1}$, and 329 ± 73 W, respectively.

2.2. Experimental design

Each participant was required to report to the laboratory on three separate occasions with each session separated by at least 48 h. Twenty-four hours prior to each experimental session, participants were asked to refrain from strenuous exercise and the consumption of caffeine or alcohol. The first experimental session consisted of preliminary anthropometric testing. The second session was considered a familiarization session. The third session was the experimental session, which was conducted in a controlled environmental chamber.

2.3. Deception

To deter performance pre-planning within the experimental design through removing conscious awareness of the existence of heat manipulations prior to the exercise, participants were deceived as to the true purpose of the experiment and were not informed of a T_a manipulation. Although participants were undoubtedly able to sense the 15 °C change in T_a as the experiment occurred, the important design consideration was that participants did not develop an initial strategy based on the prior knowledge of the performance requirements. This method simulates conditions where individuals make conscious (or subconscious) decisions with respect to performance based on current environmental conditions without any information regarding future thermal load. Therefore, upon recruitment, participants were informed that the title of the project was *"Power output variability while cycling at a constant rating of perceived exertion."* Specifically, the participants were told that the overall goal of the experiment was to test their ability to maintain a steady PO based only on perceived exertion and without external feedback, adjusting the workload of the ergometer if needed. During the experimental session, participants were positioned in an isolated corner of the chamber to minimize confounds of any cues presented by the investigator during the T_a manipulation. They also wore a mouthpiece throughout the experimental session, such that verbal communication with the investigators about the changing environment was prevented. Following the completion of data collection by all participants, they were informed to the presence of the change in T_a and were enlightened to the necessity of deception. Once this information was disclosed, participants reviewed a document outlining the true purpose and methodology of the study, then provided post-consent, allowing the use of their data in the final analysis. No subjects declined post-consent participation.

2.4. Preliminary testing

Peak aerobic capacity and peak power output were quantified during the initial preliminary testing session. Participants began by completing a 5-minute warm-up on an electronically braked cycle ergometer. Males performed a 5-minute warm-up at 100 W, followed by 25 W increments in workload every minute until exhaustion, while females performed a 5-minute warm-up at 50 W, with 20 W increments in workload every minute until exhaustion. Peak power output was recorded as the highest level of power output achieved during the last one-minute interval. Body fat percentage was measured using the skin fold measurement technique and calculated using the Jackson and Pollock equation [20,21].

Following the initial anthropometric testing session, participants reported to the lab for the familiarization session in which they completed a cycling protocol that was identical to that employed in the experimental session, except that ambient temperature was maintained at 20 °C throughout. In addition to the benefits of becoming accustomed to the experimental procedures, the familiarization session provided the opportunity to examine the PO response while riding at a constant RPE without any thermal manipulations. Furthermore, anecdotal evidence suggests that previous experience is necessary to ride at a constant perceived exertion without significant fluctuations in PO.

2.5. Experimental protocol

All experimental sessions were conducted in a controlled environmental chamber capable of temperature control within 0.5 °C across a range from −30 to +50 °C (Can-Trol Environmental Systems, Markham, Ontario). The air circulation created by the chamber's heating and cooling systems corresponded to a wind speed of approximately 1.2 $m \cdot s^{-1}$, measured at the height of the participant's head. Throughout the experimental protocol, T_a was covertly manipulated at 20-minute intervals in an A–B–A fashion from 20 °C (the A1 manipulation) to 35 °C (the B manipulation), then returning to 20 °C (A2). Relative humidity was maintained within the range of 40–60% throughout the session. Participants were asked to cycle for a total of 60 min on a cycle ergometer (Pro 300PT,

Saris Cycling Group, Madison, Wisconsin) while maintaining a rating of perceived exertion (RPE) of 14 on a 6–20 scale, which corresponds to an intensity between 'somewhat hard' and 'hard' [22]. Given the disclosure of session duration, exercise was considered as closed-loop control; however, the use of this cycle ergometer allowed for an open mode of exercise intensity in which participants had free control of both resistance and cadence, and therefore PO. The main display monitor was shielded from both the subject and the experimenter during the session to remove feedback and experimental bias, and the only feedback the participant received during the experimental session was a reminder at approximately 5 min intervals to maintain an RPE of 14.

2.6. Perceived exercise intensity protocol

The perceived exercise intensity utilized during the present experimental protocol was specifically selected so that, when coupled with the stress of the environmental conditions, caused sufficient strain to elicit voluntary changes in PO; however, not stressful enough to cause volitional fatigue prior to the completion of the 60-minute protocol. Extensive pilot testing was performed to find the highest RPE in a thermoneutral environment that would permit completion of 60 min of exercise without premature fatigue, which we defined as a major drop in power output during the final 20 min or voluntary termination prior to 60 min. A RPE of 14 on a 6–20 scale (between 'somewhat hard' and 'hard') was selected based on the above criteria. In support, a similar protocol has been used previously [22] and the results illustrate that riding at a higher level of perceived exertion (such as 16 on the Borg scale), even in 15 °C, would cause continued decrease in PO and volitional fatigue in less than 60 min. Prior to each session, participants were carefully instructed on how to rate their perceived exertion on the 6–20 Borg scale. RPE was defined as an equal combination of central (dyspnea and tachycardia) and local (active musculature) fatigue [23]. RPE training was conducted based on a scripted set of instructions to ensure consistency among participants.

2.7. Instrumentation

Height (cm) and weight (kg) were measured prior to each session using standard laboratory equipment, while euhydration, defined as a urine specific gravity of 1.02 or lower, was ensured using a refractometer (Atago, PAL-10S, USA). Throughout each session, rectal temperature (T_{re}) was measured using a thin and flexible core temperature thermistor (Mon-A-Therm Core, Mallinkrodt Medical, St Louis, MO), inserted 15 cm beyond the anal sphincter. Heart rate (HR) was measured throughout each session using a telemetric heart rate monitor (RS800CX, Polar Electro, Kempele, Finland). Local sweat rate (SR) was measured using a 13.19 cm^2 ventilated capsule placed over the trapezius muscle of the upper back. Anhydrous compressed air was passed through the capsule and over the skin surface at a constant flow rate of 0.24 $L \cdot min^{-1}$. The vapor density of the effluent air was calculated from the relative humidity and temperature measured using the Omega HX93 humidity and temperature sensor (Omega Engineering, Stanford, CT, USA). Sweat rate was defined as the product of the difference in water content between effluent and influent air and the flow rate. The sweat rate value was adjusted for skin surface area under the capsule (expressed in $mg \cdot min^{-1} \cdot cm^{-2}$).

2.8. Heat storage measurements and calculations

Residual body heat storage was measured using partitional calorimetric techniques. Skin temperature was quantified using thermistors (Concept Engineering, Old Saybrook, Connecticut) placed on the forehead, abdomen, forearm, hand, quadriceps, shin and foot surfaces [24]. Using a seven point weighted average equation, as described by Hardy and DuBois [24], mean skin temperature ($\bar{T}_{sk}$) was calculated:

$$\bar{T}_{sk} = 0.07(\text{head}) + 0.14(\text{forearm}) + 0.05(\text{hand}) + 0.07(\text{foot}) + 0.13(\text{shin}) + 0.19(\text{quadriceps}) + 0.35(\text{abdomen}).$$

Humidity at the surface of the skin was measured using small humidity probes (HMP50 RH/T, Vaisala Inc., Vantaa, Finland), taped parallel to the surface of the skin of the upper back, abdomen, and upper thigh. Metabolic data were collected using open-circuit spirometry (Moxus, AEI Technologies, Naperville, Illinois) to determine oxygen uptake and ventilation data during exercise. All data were collected at 15-second intervals throughout the entire experimental session.

The rate of heat storage ($\dot{S}$), was calculated in $W \cdot m^{-2}$ using the following heat balance equation:

$$\dot{S} = \left(\dot{M} - \dot{W}\right) - \left(\dot{E}_{res} + \dot{C}_{res} + \dot{E}_{sk} + \dot{K}_{sk} + \dot{C}_{sk} + \dot{R}_{sk}\right)$$

where $\dot{M}$ represents the heat created by metabolism, specifically the transport of oxygen throughout the body, and was calculated using the following equation [25]:

$$\dot{M} = 352 \cdot (0.23 \cdot RQ + 0.77)\left(\dot{V}_{O_2} \cdot A_D^{-1}\right)$$

where RQ represents the respiratory quotient, and A_D is the body surface area, which was calculated by [27]:

$$A_D = 0.007184 \cdot Weight^{0.425} \cdot Height^{0.725}$$

$\dot{W}$ is the mechanical work of the human body. $\dot{E}_{res}$ represents the transfer of heat though the evaporative process of respiration and $\dot{C}_{res}$ represents the transfer of heat through convective processes of respiration; $\dot{E}_{res}$ and $\dot{C}_{res}$ were modeled by the following equations, respectively [27]:

$$\dot{E}_{res} = 0.0023 \cdot \dot{M} \cdot (6.51 - P_a)$$

$$\dot{C}_{res} = 0.0014 \cdot \dot{M} \cdot (37 - T_a)$$

where P_a represents the partial pressure of water in ambient air (kPa) and T_a represents ambient temperature (°C). Evaporative heat loss through the skin, as indicated by $\dot{E}_{sk}$, was modeled by the following equation [28]:

$$\dot{E}_{sk} = (P_{sk} - P_a) \cdot v^{0.6} \cdot 124$$

where P_{sk} is the partial pressure of water vapor on the skin surface (kPa) and v represents the air velocity circulating in the environmental chamber ($m \cdot s^{-1}$). P_a and P_{sk} can be derived based on the relationship between relative humidity (ϕ) and the saturated vapor pressure of water (P_{sa}) using the following equations:

$$P_a = (\phi \cdot 0.01) \cdot P_{sa}$$

$$P_{sa} = \exp\left(18.956 - \frac{4030.18}{T_a + 235}\right)$$

$\dot{K}$, $\dot{C}$ and $\dot{R}$ represent conductive, convective and radiative heat exchange, respectively. In the present study, $\dot{K}$ is deemed to be negligible due minimal A_D in direct contact with high thermally conductive objects. $\dot{C}$ and $\dot{R}$ were calculated based on the temperature gradient between T_a and T_{sk} using the following equations:

$$\dot{C} = 8.3 \cdot v^{0.6} \cdot (T_{sk} - T_a)$$

$$\dot{R} = 4.7 \cdot (T_{sk} - T_a)$$

2.9. Statistical analysis

For PO, T_a, and physiological ($\dot{S}$, T_{re}, $\bar{T}_{sk}$, SR and HR) data, analyses focused on two separate components. First, a comparison of mean values over each 20-min interval was performed, coinciding with the A1, B, and A2 thermal manipulations, to establish the global effects of the thermal manipulation on physiological responses and power output. This was performed using a one-way (time = A1, B, A2) repeated measures ANOVA, with pair-wise comparisons with Bonferroni correction for multiple comparisons utilized to identify significant differences between manipulations. PO over the same 20 min intervals during the familiarization session was also compared. Second, synchronicity between changes in the 15-second time series data for PO with T_a, $\dot{S}$, T_{re}, $\bar{T}_{sk}$, SR and HR were compared using an Auto-Regressive Integrated Moving Average (ARIMA) time series analysis. ARIMA combines three statistical processes, autoregression, integration/differencing, and moving averages, to mathematically describe the association in a disturbance in one time series (in this case, T_a, $\dot{S}$, T_{re}, $\bar{T}_{sk}$, SR and HR) and the possible associated perturbation in a second time series (PO), and has been used successfully in previous physiological studies to assess the temporal association between changing thermal afferents (e.g. heat storage) and effector responses such as sweat rate and finger blood flow over time [29–31]. All statistical analyses were conducted using SPSS 17.0 (SPSS Inc., Chicago, Illinois), with statistical significance set at $p<0.05$.

Preliminary ARIMA analysis consisted of graphing all time series data (PO, T_a, $\dot{S}$, T_{re}, $\bar{T}_{sk}$, SR and HR) against time to determine any systematic seasonal patterns as illustrated by rhythmic fluctuations in a variable. An autocorrelation analysis was conducted, indicating a progressive decline in the autocorrelation scores throughout the time series and therefore, that the time series is non-stationary. To comply with the ARIMA assumptions, the data were transformed using differencing procedures, rendering the data stationary.

Using specific model building procedures, an appropriate ARIMA [a, d, q] model was specified based on the calculated autoregression (a), differencing (d) and moving averages (q) integers. Given the stringent nature of an ARIMA analysis, data must be collected in time series with adequate resolution to construct a viable model. To analyze potential synchronicity between PO (the dependent variable) and T_a, $\dot{S}$, T_{re}, $\bar{T}_{sk}$, SR and HR (the independent variables), an ARIMA model was constructed based on the combined time series data from all participants. Therefore, ARIMA was used to determine whether changes in PO across time were caused by any fluctuations in any of the thermo-physiological variables measured. The overall integrity of the ARIMA model was determined using the Ljung–Box test in which a probability level of $p>0.05$ implies that the model is correctly specified while a probability level of $p<0.05$ suggests that there is structure in the observed series which is not accounted for by the model. The correlation between the dependent variable and any of the independent was quantified by the stationary r^2 value (given the stationary nature of the data) while significant models were accepted at $p>0.05$.

3. Results

PO did not change significantly throughout the familiarization session at a RPE = 14 in a 20 °C environment ($p=0.227$, partial-$\eta^2=0.322$, $F=2.696$, $df=2$). Mean PO (±SD) averaged over the three 20-minute intervals was 135 ± 33 W, 95%CI = 106–164 W; 131 ± 40 W, 95%CI = 95–166 W; and 124 ± 45 W, 95%CI = 85–165 W, respectively.

During the experimental session, T_a was manipulated in a deliberate and repeatable pattern. After a 2-minute equilibrium period, T_a reached 20 °C and remained at steady state for the remainder of the first A1 (20.0 ± 0.6 °C) manipulation. Upon initiation of the B manipulation, T_a increased gradually at a rate of 1.2 °C·min^{-1}, with a mean value of 31.3 ± 0.9 °C over the entire B interval. Following the initiation of the second A2 manipulation 40 min into the experimental protocol, T_a decreased sharply at a rate of 3.2 °C·min^{-1}, with a mean of 19.0 ± 1.8 °C over the entire A2 interval.

Table 1
Mean power output (PO), ambient temperature (T_a), heat storage ($\dot{S}$), rectal temperature (T_{re}), mean skin temperature ($\bar{T}_{sk}$), sweat rate (SR) and heart rate (HR) during the A1 (20 °C), B (35 °C), and A2 (20 °C) manipulation of T_a.

	Manipulation		
	A1	B	A2
PO (W)	135 ± 39	133 ± 46	120 ± 45[†]
T_a (°C)	20.0 ± 0.6	31.3 ± 0.9[#]	19.0 ± 1.8
$\dot{S}$ (W·m^{-2})	52.35 ± 36.15	66.34 ± 22.02	−66.53 ± 56.01[†]
T_{re} (°C)	37.5 ± 0.1[*]	37.7 ± 0.1[#]	38.0 ± 0.1[†]
$\bar{T}_{sk}$ (°C)	34.2 ± 0.4	36.5 ± 1.3[#]	35.0 ± 1.9
SR (mg·min·cm^{-2})	0.268 ± 0.058[*]	0.381 ± 0.047[#]	0.338 ± 0.083[†]
HR (b·min^{-1})	137 ± 5	143 ± 3[#]	137 ± 4

Values are mean ± SD. N = 20.
[*] Significantly different from B and A2.
[#] Significantly different from A1 and A2.
[†] Significantly different from A1 and B ($p<0.05$) using repeated measures ANOVA.

Table 1 presents the mean values (±SD) of PO, T_a, $\dot{S}$, T_{re}, $\bar{T}_{sk}$, SR and HR during the A1, B and A2 manipulations, representing the ANOVA analysis. PO decreased over the 60-minute session, with a significant decrease ($p=0.004$, partial-$\eta^2=0.448$, $F=10.548$, $df=2$) being observed during the A2 manipulation (120 ± 45 W, 95%CI = 95–146 W) when compared to both the A1 (135 ± 39 W, 95%CI = 112–157 W) and B (133 ± 46 W, 95%CI = 107–160 W) manipulations. Significant differences ($p<0.001$, partial-$\eta^2=0.661$, $F=25.320$, $df=2$) in $\dot{S}$ were observed during the A2 (−66.53 ± 56.01 W·m^{-2}, 95%CI = 232.91–404.98 W·m^{-2}) manipulation when compared to manipulations A1 (52.35 ± 36.15 W·m^{-2}, 95%CI = 253.97–449.86 W·m^{-2}) and B (66.34 ± 22.02 W·m^{-2}, 95%CI = 319.64–585.64 W·m^{-2}). Heat loss via dry heat exchange ($\dot{C}$ and $\dot{R}$) occurring during the A2 manipulation was caused by a high temperature gradient between $\bar{T}_{sk}$ and T_a. $\bar{T}_{sk}$ remained elevated following the initiation of the A2 manipulation (from 35 °C to 20 °C); however, T_a decreases quickly, creating a gradient (and therefore, mechanism for heat exchange) that is much greater than during the A1 and B manipulations. Similarly, water vapor pressure at the skin and sweating rate remained elevated for the first half of A2, sustaining greater evaporative heat loss than during A1. The trend in T_{re} throughout the 60-minute session exhibited a significant increase ($p<0.001$, partial-$\eta^2=0.675$, $F=26.959$, $df=2$) during each successive manipulation (37.5 ± 0.1 °C, 95%CI = 37.4–38.0 °C; 37.7 ± 0.1 °C, 95%CI = 37.8–38.4 °C; 38.0 ± 0.1 °C, 95%CI = 38.0–38.5 °C during the A1, B and A2 manipulations, respectively). Both $\bar{T}_{sk}$ ($p<0.001$, partial-$\eta^2=0.828$, $F=62.773$, $df=2$) and HR ($p<0.001$, partial-$\eta^2=0.134$, $F=12.187$, $df=2$) fluctuated throughout the 60-minute session, reaching significantly higher values during the B (36.5 ± 1.3 °C, 95%CI = 25.49–28.96 °C; 143 ± 3 b·min^{-1}, 95%CI = 143–151 b·min^{-1}) manipulation in comparison to both A1 (34.2 ± 0.4 °C, 95%CI = 24.75–27.66 °C; 137 ± 5 b·min^{-1}, 95%CI = 137–145 b·min^{-1}) and A2 (35.0 ± 1.9 °C, 95%CI = 26.74–29.63 °C; 137 ± 4 b·min^{-1}, 95%CI = 139–148 b·min^{-1}) manipulations, respectively. SR was significantly different ($p<0.022$, partial-$\eta^2=0.632$, $F=22.338$, $df=2$) during all 3 manipulations; peaking during the B (0.381 ± 0.047 mg·min·cm^{-2}, 95%CI = 0.359–0.403 mg·min·cm^{-2}) manipulation compared to the A1 (0.268 ± 0.058 mg·min·cm^{-2}, 95%CI = 0.241–0.295 mg·min·cm^{-2}) and A2 (0.338 ± 0.083 mg·min·cm^{-2}, 95%CI = 0.322–0.358 mg·min·cm^{-2}) manipulations.

Fig. 1 is a visual representation of the mean (±SD) 15-second time series data and therefore, the temporal association quantified using ARIMA analysis between PO and T_a, $\dot{S}$, T_{re}, $\bar{T}_{sk}$, SR and HR throughout the 60-minute session. Table 2 outlines the specific Ljung–Box

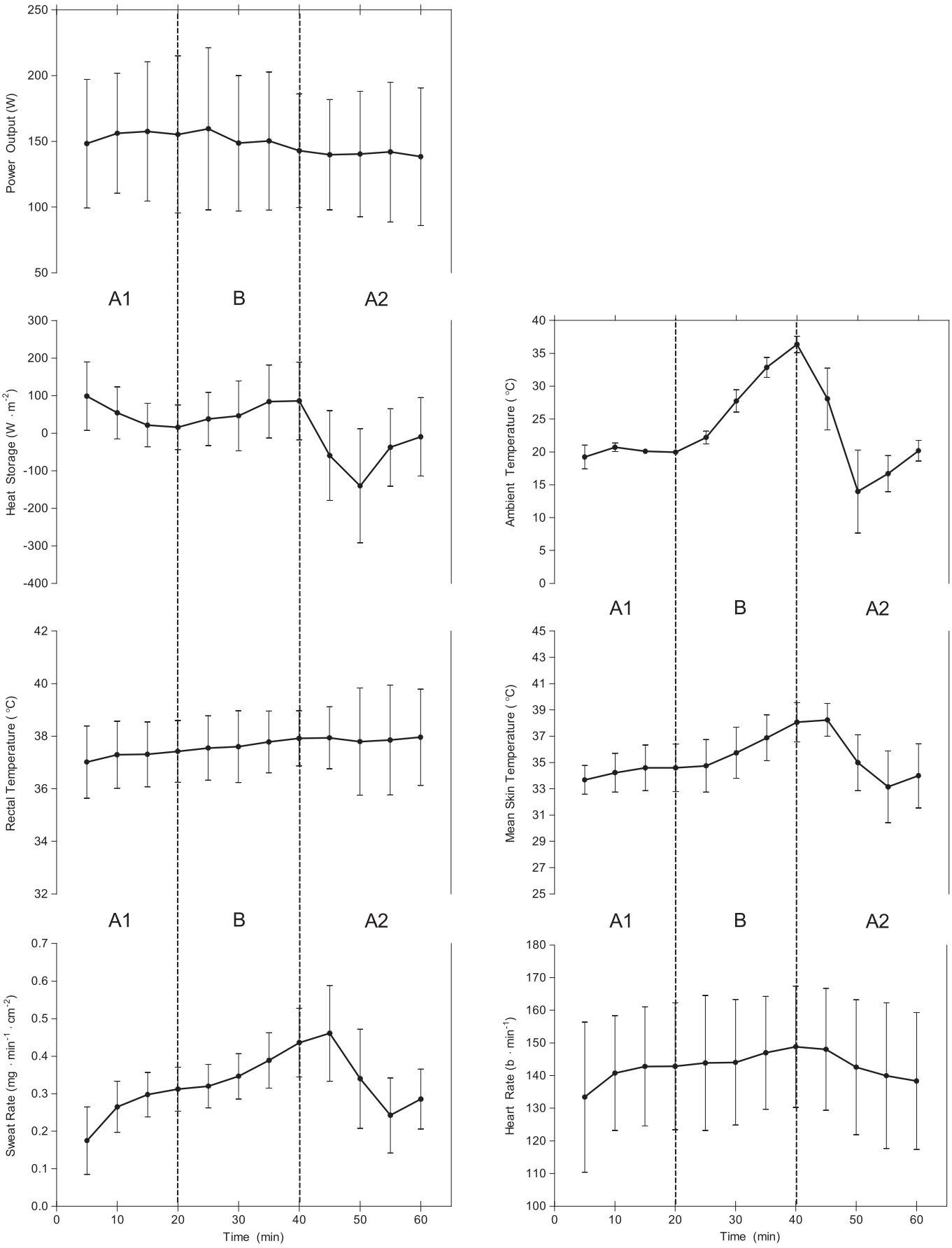
Power Output (W)
250
200
150
100
50
A1
B
A2
Heat Storage (W · m-2)
300
200
100
0
-100
-200
-300
-400
Ambient Temperature (°C)
40
35
30
25
20
15
10
5
0
A1
B
A2
Rectal Temperature (°C)
42
40
38
36
34
32
Mean Skin Temperature (°C)
45
43
41
39
37
35
33
31
29
27
25
A1
B
A2
Sweat Rate (mg · min-1 · cm-2)
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0.0
Heart Rate (b · min-1)
180
170
160
150
140
130
120
110
100
0
10
20
30
40
50
60
Time (min)
Time (min)

Table 2
Independent predictor variables for PO and associated stationary r^2 and Ljung–Box statistics resultant from the ARIMA analysis.

Predictor variable	Stationary r^2 (p value)	Ljung–Box statistic (p value)
Ta	0.052 (0.036)	19.085 (0.162)
$\dot{S}$	0.042 (0.019)	28.181 (0.058)
T_{re}	0.001 (0.001)	15.569 (0.569)
$\bar{T}_{sk}$	0.091 (0.018)	24.380 (0.059)
SR	0.053 (0.031)	20.847 (0.106)
HR	0.054 (0.036)	18.320 (0.193)

statistic and stationary r^2 values statistics associated with each predictor variable for PO. All models passed the Ljung–Box test ($p > 0.05$) confirming that they were correctly specified in the ARIMA process. The results suggest that perturbations in PO were not associated with changes in any of the collected thermo-physiological variables, specifically, changes T_a, $\dot{S}$, T_{re}, $\bar{T}_{sk}$, SR and HR (Table 2).

4. Discussion

Initial ANOVA analysis clearly illustrates that, while no significant changes in PO were evident during the familiarization session, a progressive reduction in PO occurred during the experimental session that appears inversely related with elevated thermal status, specifically SR, T_{re}, and $\bar{T}_{sk}$. The subsequent ARIMA time-series analyses indicated that, regardless of the increase in SR, T_{re}, and $\bar{T}_{sk}$ caused by the initiation of the B manipulation, PO was not down-regulated in a synchronized manner. Collectively, these observations suggest that voluntary submaximal exercise is not directly mediated by a change in thermal afferents, and therefore, the behavioral response to heat stress during exercise involves a more complex interplay of psychological and physiological factors.

Using ANOVA, PO was found to decrease during the A2 manipulation, despite a significant reduction in $\dot{S}$ compared to A1 and B manipulation values, suggesting a potential linkage between PO and $\dot{S}$. However, ARIMA analysis indicates that no repeatable patterns in $\dot{S}$ were observed and also that changes did not occur in synchrony with PO. Together, these analyses suggests that, during a dynamic manipulation of T_a, $\dot{S}$ is not a physiological afferent that directly influences a conscious reduction in exercise intensity, though it may indirectly contribute to the overall perception of exercise intensity and psycho-physiological regulation of exercise intensity. This contrasts with previous research [14] utilizing a similar RPE clamp protocol that proposes that voluntary work output is consciously controlled to a constant $\dot{S}$ in anticipation of optimizing performance while delaying the onset of hyperthermia. The different methods used in calculating heat storage may help to explain the contrasting findings on the relationship between heat storage and voluntary exercise intensity. Tucker et al. [14,32] utilized a thermometric two-compartment "shell" and "core" model to calculate heat storage every 1 min during exercise. However, thermometry has been demonstrated to consistently underestimate heat storage in both heat [33] and cold [34]. Furthermore, this thermometric model is compromised by theoretical limitations when applied to exercise. The limitations of using the two-compartment (T_{re} and $\bar{T}_{sk}$) calculation of mean body temperature, especially during dynamic exercise, to quantify $\dot{S}$ are twofold [15]. Primarily, estimations of $\dot{S}$ during the initial phase of exercise are drastically under-estimated due to the delayed activation of heat loss mechanisms. Additionally, the large thermal inertia of the pelvic region causes a gradual change in rectal temperature, and does not adequately reflect the minute-by-minute changes in total body heat content. Rather than relying on rectal temperature, the present study employed partitional calorimetry to directly calculate the individual components of heat production and exchange with the environment, providing a more accurate method of quantifying heat exchange during dynamic exercise and short measurement intervals [15]. Supporting the advantages of partitional calorimetry, we recently showed that partitional calorimetric indices explained more effectively the thermoregulatory responses observed during rest and exercise in the heat, as compared with thermometric indices [30].

Intriguing in the theory of an integrated psycho-physiological regulation of voluntary submaximal exercise is the association between thermal afferents and their effect on perceptions of heat stress, and ultimately how this integrated information may influence work output during exercise. Core temperature will rise during exercise even in cool or thermoneutral environments, such that increasing core temperature reflects both changes in work intensity in addition to extra heat strain from the environment that needs to be counteracted. This can be seen with differences in self-selected running speed between smaller African and larger Caucasian runners being evident only in hot (35 °C) but not cool (15 °C) temperatures [35]. Skin temperature is another focus of research in this paradigm, and skin temperature alterations appear to act on behavior to prevent or 'buffer' changes in core temperature [18,36]. Schlader et al. [37] indicate that changes in $\bar{T}_{sk}$ during passive thermal exposure drives changes in thermal comfort, and therefore initiate conscious behavioral actions to modulate one's thermal status. Similarly, self-selected exercise intensity appears to be controlled in response to changing $\bar{T}_{sk}$ and the accompanying thermal perception [16]. Tatterson et al. [17] reported a 6.5% decrease in PO while cycling in hot (32 °C) when compared to cool (23 °C), conditions; however, the compelling finding was that, despite the 9 °C difference in T_a, core temperature remained similar. It is thus believed that the increase in $\bar{T}_{sk}$ during the hot condition causes a decrease in neural drive [17], and in an anticipatory, feed forward fashion, the body was protected from accumulating excessive heat.

Unique to this study is the use of an A–B–A manipulation, in which the effect of a stimulus response (in this case an increased T_a during the B manipulation) on baseline values (PO during the first A1 manipulation), followed by the removal of the stimulus (the return to 20 °C during the A2 manipulation) was examined. Specifically, the inferential power of examining the stimulus and removal of a treatment variable is much greater when compared to examining only the treatment variable itself [38]. Furthermore, the use of an A–B–A design allows observation of the voluntary PO response during two different environmental conditions without introducing inter-trial variability, especially as psychological factors such as motivation may vary between sessions. The integrity of our experimental design also relies on the assumption that all participants were able to effectively "clamp" their RPE. Theoretically, the correlation between RPE and HR [22] suggests that the two variables can be used analogously, such that an RPE score of 14 should correlate to a heart rate of 140 $b \cdot min^{-1}$. Mean HR was consistently within the 140 to 150 $b \cdot min^{-1}$ range, suggesting that the magnitude of perceived exertion was within the acceptable range throughout the 60-minute session. To supplement the A–B–A manipulation, ARIMA statistical analysis provides a significantly improved research design when compared to previous studies. Typically, the cause and effect relationship between PO and thermo-physiological status is statistically determined using a repeated measures ANOVA [14,32], which examines the mean response of a variable at a few time intervals.

Fig. 1. Mean (±SD) 5-minute time series data for power output (PO), ambient temperature (T_a), heat storage ($\dot{S}$), rectal temperature (T_{re}), mean skin temperature ($\bar{T}_{sk}$), sweat rate (SR) and heart rate (HR) throughout the 60-minute A–B–A experimental session (N = 20, stationary-$r^2 \leq 0.10$ and Ljung–Box statistic > 0.05 for all variables in ARIMA analyses when correlated with power output).

Although the ANOVA methodology may indicate that the mean difference in PO during each manipulation is significantly different – which is the case in the present study – it does not evaluate dynamic correlation across time between two variables, and therefore, accurate cause-and-effect relationships cannot be inferred.

Although very insightful, previous studies [14,32] have not addressed the psycho-physiological mechanisms that may influence voluntary exercise intensity when the presence of an environmental stressor is known prior to the start of exercise, where psychological factors such as experience, prior knowledge of manipulations [19], and motivation may override physiological afferents. Therefore, our use of deception and a single constant RPE experimental session aimed to ensure that any possible changes in PO associated with each thermal manipulation would have occurred as a result of a modulation in afferent feedback, not psychological factors associated with prior knowledge of environmental manipulation or performance requirements. Although the mechanism(s) related to the perception of thermal stress and the resultant influence on exercise performance remains an intricate issue, the impact of heat stress on thermobehavior is undoubted [39]. Maw et al. [40] report a higher thermal discomfort and subjective RPE while cycling at a constant workload in the heat, which suggests that this psychological process modulates the resultant behavioral response. Furthermore, Mauger et al. [41] demonstrated the importance of prior knowledge when developing a pacing strategy during athletic competition, as subjects rapidly learned the optimal pacing required for a 4 km time trial within four trials performed over the course of a day, despite not receiving any prior knowledge of distance, nor any external feedback throughout the time trials. Similarly, Metsios et al. [42] suggest that performance feedback during tests of predicted maximal oxygen uptake results in superior performance. Collectively, this evidence suggests that without adequate deception and blinding, subjects are likely to adopt a strategy based on the conscious knowledge of a stressor or performance requirements, rather than a conscious feed-forward regulation of exercise intensity.

Although the present study utilizes novel methodologies to address the research question, several inherent design limitations may influence the results. A potential confound to this project is that an RPE of 14 may have elicited a workload too low to require behavioral thermoregulation (i.e., a decrease in PO) during the 35 °C 'B' manipulation. Although this is a legitimate issue that must be considered, evidence of an increased HR during the 'B' manipulation suggests that the combination of increased T_a and workload elicited by a RPE of 14 was sufficient to initiate behavioral thermoregulation. An important variable that was not measured during the present study is the subjective measures of thermal comfort and thermal sensation. Previous research [16,39] suggests that there is a strong association between perceived thermal comfort and exercise intensity selection. Although a viable design consideration, the present study specifically did not incorporate any measurement of thermal comfort/sensation in an attempt to isolate the influence of thermal afferents on voluntary exercise intensity, rather than subjective evaluations of the thermal environment.

In summary, the present study aimed to directly investigate the relationship between thermal afferents and voluntary exercise intensity control during environmental stress. When cycling at a constant RPE, the observed changes in PO did not correspond to any observed perturbations in thermophysiological homeostasis. This observation conflicts with previous research [14,32], in that we saw fluctuating heat storage with no apparent synchronous effect on voluntary exercise intensity. Furthermore, there was no discernible synchronicity between the changes in T_{re}, $\bar{T}_{sk}$, SR and HR and any changes in PO. These results suggest that the mechanism(s) that control PO during self-paced exercise involves a more complex interplay of psychological and physiological factors than simply an abrupt change in ambient temperature.

Acknowledgments

We express our gratitude to the participants for their efforts throughout the study. The study was supported by the Natural Science and Engineering Research Council of Canada through a Discovery Grant (#227912-07, S.S. Cheung).

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