HIGH-INTENSITY INTERVAL TRAINING IN THE HEAT

by

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ABSTRACT

High-intensity interval training (HIIT) is an effective form of exercise; however, it remains unclear how heat stress, exercise intensity prescription, and recovery mode influence work rate adjustments, thermal and cardiovascular strain, and maximal aerobic capacity ($\dot{V}O_{2max}$). Three studies investigated these outcomes between the first and fifth round of HIIT (8-min warm-up and rounds of 4-min work×3-min recovery). $\dot{V}O_{2max}$ was measured on separate occasions after the first and final round of HIIT. In Study 1, HIIT was completed in hot (35 °C) and temperate (22 °C) environments using target heart rate (THR) [warm-up and recovery=70% maximal heart rate (HR_{max}), work=90% HR_{max}]. To maintain target intensity, considerable decreases in work rate occurred in both conditions, but they were nearly twice as large in the heat, accompanied by elevated recovery HR, higher mean skin temperature, and greater increases in rectal temperature. Following the final round of HIIT, $\dot{V}O_{2max}$ decreased marginally.

In Study 2, the HIIT workout was completed in 35 °C at the same THR as Study 1 or a target rating of perceived exertion (RPE) (warm-up and recovery=RPE of 12, work=RPE of 17). To maintain target intensity, work rate decreased 46 W and 30 W in the HR- and RPE-based trials, respectively. Thermal strain was similar between conditions, but elevated cardiovascular strain during RPE-based HIIT corresponded to a larger decrease (15.6%) in $\dot{V}O_{2max}$ compared to HR-based HIIT (6.5%). In Study 3, the HIIT workout occurred in 35 °C and included work at RPE=17 and passive (rest) or active recovery (RPE=12). Thermal strain was similar, but active recovery increased cardiovascular strain and resulted in a larger percent decrease in work rate. $\dot{V}O_{2max}$ declined 11.5% between the first and fifth work intervals regardless of recovery mode.

These studies demonstrated all variations of HIIT necessitated meaningful reductions in work rate over time except HIIT based on RPE with passive recovery. Participants ranged from low fit to high fit, but all completed the HIIT sessions. Nonetheless, work rate adjustments and thermal and cardiovascular strain were dependent on the method of exercise intensity prescription and recovery mode and should be considered when prescribing HIIT in the heat.

DEDICATION

It is with genuine gratitude that I dedicate this to my parents Kim and Denis Yoder and sister Alli Katz for their love and support.

LIST OF ABBREVIATIONS AND SYMBOLS

Δ	difference, change
α	alpha
α'	alpha prime
$\%HR_{max}$	percent of maximal heart rate
15_{ACT}	15 min of exercise based on target rating of perceived exertion with active recovery in a hot environment (CHAPTER 4)
15_{HOT}	15 min of exercise based on target heart rate in a hot environment (CHAPTER 2)
15_{HR}	15 min of exercise based on target heart rate in a hot environment (CHAPTER 3)
15_{PASS}	15 min of exercise on target heart rate in a hot environment (CHAPTER 4)
15_{RPE}	15 min of exercise based on target rating of perceived exertion in a hot environment (CHAPTER 3)
15_{TEMP}	15 min of exercise based on target heart rate in a temperate environment (CHAPTER 2)
43_{ACT}	43 min of exercise based on target rating of perceived exertion with active recovery in a hot environment (CHAPTER 4)
43_{HOT}	43 min of exercise based on target heart rate in a hot environment (CHAPTER 2)
43_{HR}	43 min of exercise based on target heart rate in a hot environment (CHAPTER 3)
43_{PASS}	43 min of exercise based on target heart rate in a hot environment (CHAPTER 4)
43_{RPE}	43 min of exercise based on target rating of perceived exertion in a hot environment (CHAPTER 3)
43_{TEMP}	43 min of exercise based on target heart rate in a temperate environment (CHAPTER 2)
ANOVA	analysis of variance
CI	confidence interval

ec	caloric equivalent per liter of oxygen for oxidation of carbohydrates
ef	caloric equivalent per liter of oxygen for fat oxidation
ES	effect size
GXT	graded exercise test
Hb	hemoglobin
HCT	hematocrit
HIIT	high-intensity interval training {PAGE iv}
HR	heart rate
kg	kilogram
$M - W$	metabolic heat production
PV	plasma volume
RER	respiratory exchange ratio
RH	relative humidity
RPE	rating of perceived exertion
SD	standard deviation
SE_d	standard error of the differences
SV	stroke volume
$\overline{T}_b$	mean body temperature
T_{calf}	skin temperature of calf
T_{chest}	skin temperature of calf
t_{cv}	critical t
T_{delt}	skin temperature of calf
THR	target heart rate
T_{re}	rectal temperature
$\overline{T}_{sk}$	mean skin temperature

T_{thigh}	skin temperature of calf
U_{SG}	urine specific gravity
$\dot{V}_E$	minute ventilation
$\dot{V}O_2$	oxygen uptake
$\dot{V}O_{2max}$	maximal oxygen uptake
W	absolute rate of metabolic heat production
W	work rate (mechanical power)

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My committee members were instrumental in completing this dissertation as well. Dr. Leeper provided helpful guidance for the statistical analyses, especially during the writing process. Drs. Richardson and Mota provided useful feedback to improve the project during the proposal and project development. Dr. MacDonald provided invaluable guidance during this project and encouraged her students to assist with data collection. Lastly, I would like to thank Dr. Wingo for teaching me the necessary skills to complete this project and his support developing the ideas, executing the project, and writing this document. I am thankful I had the opportunity to learn from him.

Outside of the University of Alabama, Dr. Johnson encouraged me to pursue a PhD and has been an encouraging mentor. Buster, my family dog, gets my final acknowledgement for being a great companion, along with my family, when I was home during COVID and for

Buster's inspirational attitude. Despite his failures, he chases rabbits with the belief that he will catch one.

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CHAPTER 1

INTRODUCTION

High intensity interval training (HIIT)—a popular mode of exercise for those aiming to improve their health and fitness (Esfarjani and Laursen 2007; Helgerud et al. 2007; Tjønna et al. 2008)—involves alternating between high-intensity and lower-intensity exercise. A current limitation of research involving HIIT and its practical application is that most studies have been completed in controlled temperate environments (Buchheit and Laursen 2013b), even though HIIT workouts are performed in a variety of environments, including in the heat [out of necessity or intentionally for acclimation (Sunderland et al. 2008)]. Since cardiovascular responses [e.g., heart rate (HR)] are differentially affected by heat stress (Lafrenz et al. 2008; Tucker et al. 2006), and because these responses are related to exercise intensity prescription (Wingo and Cureton 2006; American College of Sports Medicine 2021; Tucker et al. 2006), they must be given special consideration in order to optimize HIIT.

For example, exercise in hot conditions results in greater cardiovascular drift, a progressive increase in HR and decrease in stroke volume that occurs over time despite no change in work rate (Wingo 2015). While cardiovascular drift is traditionally considered to occur during continuous exercise, it may also occur during HIIT by "accumulating" over the course of the workout, i.e., HR may progressively increase over successive work bouts even though work rate remains constant. As such, some methods of exercise intensity prescription may have unintended consequences during HIIT in the heat. For instance, if cardiovascular drift occurs when using target HR (THR), workload will have to be lowered throughout the workout to

maintain THR. If using rating of perceived exertion (RPE) to prescribe intensity, a higher subjective workload may be maintainable compared to using THR, but this could lead to increased thermal and cardiovascular strain.

Besides exercise prescription considerations involving HR and RPE during HIIT in a hot environment, the recovery interval mode—active, in which exercise continues at a lower intensity, or passive, in which exercise ceases—may also differentially affect the achievable workload during work intervals and the magnitude of cardiovascular and thermal strain. In general, during HIIT with work intervals > 1 min in duration, passive recovery is recommended if the recovery interval is < 2–3 min, but active recovery is recommended if the recovery interval is > 3–4 min (Buchheit and Laursen 2013a). However, these recommendations do not account for increased thermal strain expected with active recovery in hot conditions (Bishop et al. 2007), which could necessitate lower work rates during the high-intensity intervals.

To further complicate matters, cardiovascular drift is associated with reduced maximal oxygen uptake ($\dot{V}O_{2max}$) during continuous exercise in hot conditions (Lafrenz et al. 2008; Wingo and Cureton 2006; Wingo et al. 2020). Temporary decreases in $\dot{V}O_{2max}$ cause any given level of work to be perceived as more taxing (i.e., requiring a higher $\%\dot{V}O_{2max}$), which could further muddle exercise prescription during HIIT. However, the extent to which $\dot{V}O_{2max}$ is affected during HIIT in the heat is unknown.

Outcomes related to modifications of HIIT prescription based on HR, RPE, and form of recovery in hot conditions remain unknown. Therefore, the overall aim of this dissertation was to evaluate work rate adjustments and cardiovascular and thermal strain as well as changes in $\dot{V}O_{2max}$ using different methods of exercise prescription during HIIT in a hot environment.

Hypotheses

Study 1. The hypotheses being tested are:

1) A single bout of HIIT in a hot environment will result in increased cardiovascular and thermal strain compared to HIIT in a temperate environment.
2) Work rate must be lowered to a greater extent during a single bout of HIIT in a hot environment compared to HIIT in a temperate environment in order to maintain THR.
3) $\dot{V}O_{2max}$ will decrease to a greater extent after a single bout of HIIT in the heat compared to HIIT in a temperate environment when exercise intensity during HIIT is based on THR.

Study 2. The hypotheses being tested are:

1) Work rate must be lowered to a greater extent to maintain THR than to maintain target RPE during a single bout of HIIT in a hot environment.
2) Greater thermal and cardiovascular strain will result from maintaining target RPE compared to THR during a single bout of HIIT in the heat.
3) $\dot{V}O_{2max}$ will decrease to a greater extent after a single bout of HIIT in the heat when exercise intensity during HIIT is based on target RPE compared to THR.

Study 3. The hypotheses being tested are:

1) Active recovery will increase thermal and cardiovascular strain compared to passive recovery during a single bout of HIIT in the heat.
2) Progressive declines in work rate during successive high-intensity intervals will be greater with active recovery compared to passive recovery during a single bout of HIIT in a hot environment.

3) $\dot{V}O_{2max}$ will decrease to a greater extent after a single bout of HIIT in the heat with active recovery compared to HIIT with passive recovery.

CHAPTER 2

WORK RATE ADJUSTMENTS DURING HEART RATE-BASED HIGH-INTENSITY INTERVAL TRAINING IN HOT AND TEMPERATE ENVIRONMENTS

INTRODUCTION

World class athletes have incorporated high-intensity interval training (HIIT), exercise that involves alternating between high-intensity and lower intensity exercise, into their training regimes for over a century (Buchheit and Laursen 2013; Billat 2001). More recently, HIIT has become a popular mode of exercise for recreational athletes aiming to improve their health and fitness. Indeed, the American College of Sports Medicine has ranked HIIT in the top 5 Worldwide Fitness Trends from 2014 – 2021 (Thompson 2021), and it is effective. HIIT programs improve cardiorespiratory fitness in trained (Esfarjani and Laursen 2007) and untrained populations (Helgerud et al. 2007) and reduce risk factors related to metabolic syndrome (Tjønna et al. 2008).

For HIIT to elicit desired training outcomes and to be most effective, intensity must be carefully prescribed. A common method of prescribing exercise intensity is to set work rate based on a corresponding target heart rate (THR). During continuous exercise, a progressive increase in heart rate (HR) can occur over time despite no change in work rate, a phenomenon known as cardiovascular drift (Wingo and Cureton 2006). Cardiovascular drift may also occur during HIIT by "accumulating" over the course of the workout, i.e., HR may progressively increase over successive work bouts and recovery bouts even though work rate remains constant (Falz et al. 2019). When using THR to gauge exercise intensity, cardiovascular drift is

problematic because the work rate must be lowered to maintain THR, which can compromise the training stimulus (Wingo 2015; Morales-Palomo et al. 2017; Wingo et al. 2012; Wingo and Cureton 2006). Indeed, in a study comparing HIIT to continuous exercise in a temperate environment, greater reductions in work rate were necessary during HIIT compared to continuous exercise in order to maintain THR because of greater cardiovascular drift during HIIT (Morales-Palomo et al. 2017). Using THR to prescribe exercise intensity in hot conditions may be especially problematic because heat stress amplifies cardiovascular drift (Lafrenz et al. 2008), which necessitates large reductions in work rate to maintain THR (Wingo and Cureton 2006). Despite the potential limitations of using THR to prescribe exercise intensity of HIIT in hot environments because of cardiovascular drift, it has not been evaluated and the effect of cardiovascular drift on exercise intensity prescription during HIIT in the heat remains unknown.

In addition to the aforementioned considerations regarding cardiovascular drift, a consequence of cardiovascular drift is that it corresponds to reduced maximal oxygen uptake ($\dot{V}O_{2max}$) during continuous exercise in hot conditions (Wingo et al. 2005; Lafrenz et al. 2008). This has implications for how exercise is perceived (e.g., if $\dot{V}O_{2max}$ declines, a given work rate momentarily represents a greater proportion of $\dot{V}O_{2max}$ and therefore will be perceived as more taxing). Even though heat stress amplifies the decline in $\dot{V}O_{2max}$ associated with cardiovascular drift during continuous exercise (Lafrenz et al. 2008), it is unclear how maintaining THR during HIIT, and thereby preventing cardiovascular drift, affects $\dot{V}O_{2max}$. Other factors associated with exercise in hot conditions, such as elevated core and skin temperatures, also reduce aerobic capacity in the heat (Cheuvront et al. 2010). Therefore, HIIT performed in hot conditions, even without cardiovascular drift, may still result in compromised $\dot{V}O_{2max}$ compared to temperate

conditions because of higher core and skin temperatures in the heat, but this has not been investigated.

Given the preceding notions, the purposes of this study were to test the hypotheses that 1) HIIT in a hot environment will result in increased cardiovascular (i.e., elevated recovery HR) and thermal strain compared to HIIT in a temperate environment, 2) work rate must be lowered to a greater extent during HIIT in a hot environment compared to HIIT in a temperate environment in order to maintain THR, and 3) $\dot{V}O_{2max}$ will decrease to a greater extent after HIIT in the heat compared to HIIT in a temperate environment when exercise intensity is gauged by THR.

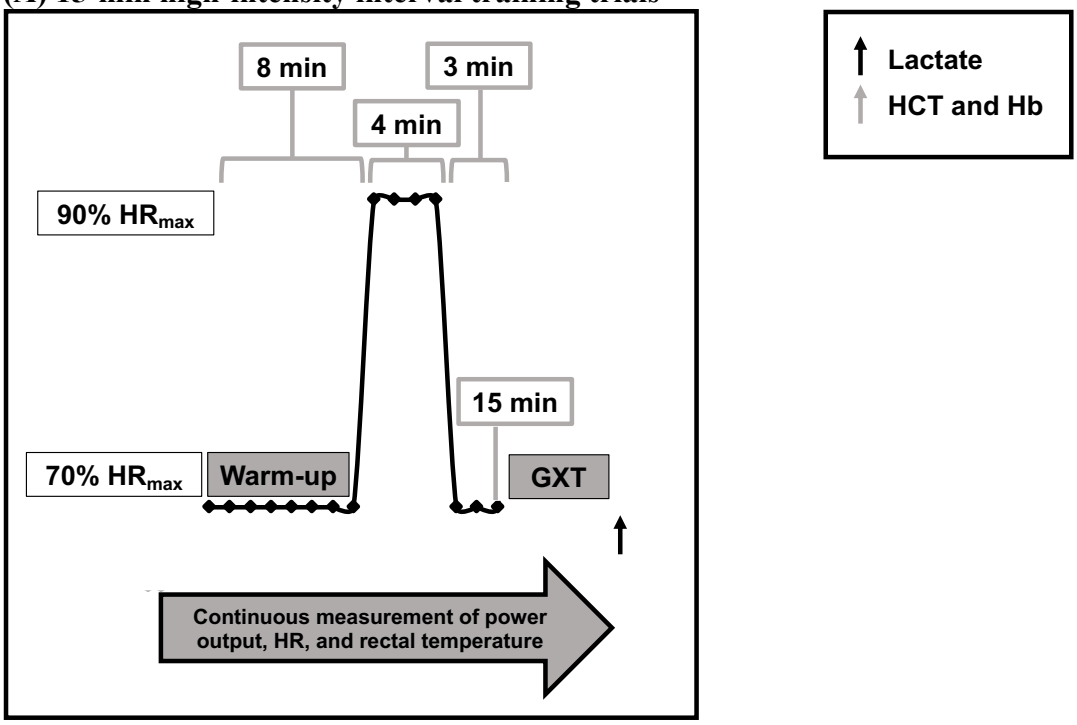
METHODS

Experimental design

Participants completed 5 trials (1 control trial, two 15-min experimental trials, and two 43-min experimental trials) on a cycle ergometer (LC6 Novo, Monark Exercise, Vansbro, Sweden) during summer to mid-fall in the southeastern United States. A repeated measures crossover design was used. For the control trial, participants completed a graded exercise test (GXT) to measure maximal HR (HR_{max}) and $\dot{V}O_{2max}$ in a temperate environment [~ 22 °C, ~ 40% relative humidity (RH)]. At least 24 h separated the control trial and the first experimental trial. The 4 remaining experimental trials were performed in a counterbalanced order and counterbalanced treatment orders were randomly assigned to participants. Experimental trials were separated by at least 48 h; each consisted of an 8-min warm-up at 70% HR_{max} followed by 1 or 5 rounds of HIIT (4 min at 90% HR_{max} and 3 min at 70% HR_{max}) in a temperate (~ 22 °C, ~40% RH) or hot (~ 35 °C, ~ 40% RH) environment: 15_{TEMP} = warm-up and 1 round of HIIT in a temperate environment; 15_{HOT} = warm-up and 1 round of HIIT in a hot environment; 43_{TEMP} =

warm-up and 5 rounds of HIIT in a temperate environment; 43_{HOT} = warm-up and 5 rounds of HIIT in a hot environment. An overview of the HIIT protocol is shown in Figure 2.1. During the recovery intervals, work rate was lowered to achieve 70% HR_{max}, but not below 30 W even if HR was still greater than 70% HR_{max} to ensure the intensity could be distinguished from passive recovery. Each experimental trial was immediately followed by a GXT to measure $\dot{V}O_{2max}$. The purpose of the separate 15- and 43-min trials was to evaluate $\dot{V}O_{2max}$ before (15-min trials) and after (43-min trials) work rate adjustments had been made in order to maintain THR. All trials for a given participant took place over ≤ 8 weeks.

(A) 15-min high-intensity interval training trials



(B) 43-min high-intensity interval training trials

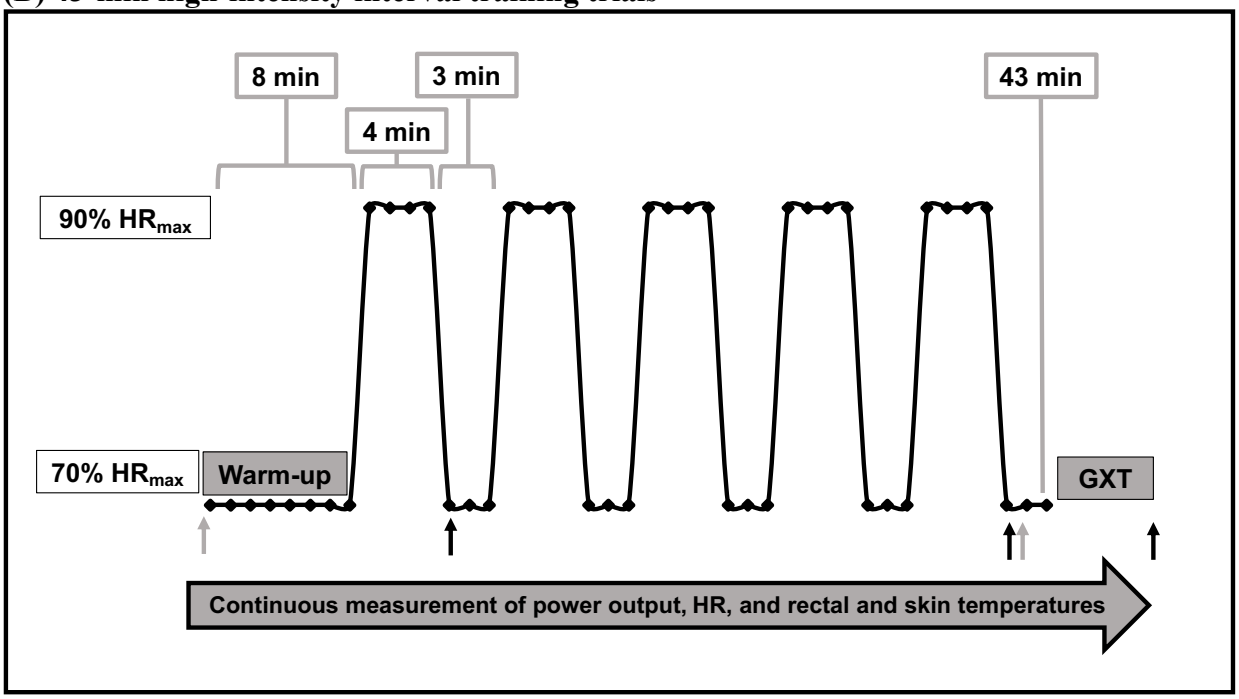


Figure 2.1. General exercise protocols for the (A) 15- and (B) 43-min experimental trials. GXT, graded exercise test; $\dot{V}O_2$, oxygen uptake; HR, heart rate. Arrows indicate blood samples drawn for analysis of hematocrit (HCT), hemoglobin (Hb), and lactate concentration.

Participants

An a priori power analysis (G*power 3.1.9.6) revealed a sample size of 7 would be sufficient to detect a 25-W (effect size = 1.4) difference between the change score in power output from the first to the final work interval in 43_{TEMP} versus 43_{HOT}, assuming $\alpha = 0.05$ and power ≈ 0.80. Nine participants consented and 7 completed all study procedures. One participant withdrew following the first visit another after the second visit. Participants included 4 men and 3 women, 6 were recreationally active (participating in ≥ 30 min of aerobic exercise per day, ≥ 3 times per week for the past ≥ 3 months) (American College of Sports Medicine 2021) and 1 was an endurance athlete, all free of cardiovascular, renal, and metabolic disease. Physical activity levels and health history were confirmed through questionnaires; all participants reported engaging in physical activity outdoors. Physical characteristics of participants were age (mean ± SD) = 27 ± 6 y, body mass = 74.1 ± 8.3 kg, height = 183 ± 8 cm, and percent body fat = 21.4% ± 6.6%, HR_{max} = 186 ± 7 b·min^{-1}, 70% HR_{max} = 130 ± 5 b·min^{-1}, 90% HR_{max} = 167 ± 6 b·min^{-1}.

Female participants had a regular menstrual cycle lasting 21–35 days (Elliott-Sale et al. 2021) and self-reported the first and last day of previous menses. Self-reported information was used to attempt to schedule women during the same phase of their menstrual cycle, although the specific phase was not expected to affect study outcomes (Stone et al. 2021). Based on baseline rectal temperature, it is likely 1 woman completed the 43_{HOT} trial in the follicular phase and the remaining 3 experimental trials in the luteal phase of her menstrual cycle. None of the women were taking oral contraceptives and phase of the menstrual cycle was not confirmed with a hormonal assay.

Procedures

All trials

For each of the 5 trials, participants were instructed to arrive at the laboratory after a 2-h fast, well rested, euhydrated, and having refrained from ingesting non-prescription drugs and caffeine on the day of testing. Additionally, participants were instructed to avoid consuming alcohol or participating in strenuous exercise during the 24 h prior to testing. Adherence to pre-test instructions was confirmed using a 24-h history questionnaire. Upon arrival, participants provided a urine sample that was analyzed for urine specific gravity (U_{SG}) using a digital refractometer (ATAGO PAL-10S digital refractometer, Tokyo, Japan). U_{SG} had to be ≤ 1.020 for a participant to be considered adequately hydrated (Sawka et al. 2007). Participants whose U_{SG} values were > 1.020 were given fluids to ingest for 20–30 min and reevaluated. Then participants donned padded cycling shorts, a mesh tank top, and a chest strap HR monitor (H10, Polar Electro, Kempele, Finland). All trials were completed at a similar time of day (± 2 h) to control for fluctuations in core body temperature associated with circadian rhythm (Moore-Ede et al. 1983). During all GXTs, researchers provided verbal encouragement to participants.

Control $\dot{V}O_{2max}$ trial

At the first visit, participants completed a questionnaire about their readiness to participate in exercise, filled out a general health history form, and provided written informed consent. Following completion of paperwork, height was measured using a stadiometer (SECA 213, Seca Ltd., Hamburg, Germany), body mass was measured with a digital scale (Tanita WB-800S plus, Tanita Corp., Tokyo Japan) while wearing cycling clothes and socks, and body fat percentage was calculated from the sum of 3 skinfolds (Jackson and Pollock 1985).

Participants then began a 5–10-min warm-up on the cycle ergometer at a self-selected moderate intensity in a temperate environment (22.6 ± 0.6 °C, 36% ± 4% RH). After the warm-up, a GXT to determine $\dot{V}O_{2max}$ and HR_{max} began at a moderate intensity based on the self-selected work rate, HR responses, and rating of perceived exertion (RPE) during the warm-up. Every 2 min power output was increased by 25 W until volitional exhaustion was reached or cadence fell below 30 rev·min^{-1}. During the test, $\dot{V}O_2$ was measured continuously using open circuit spirometry (Parvo Medics Metabolic Measurement System, model TrueOne 2400, Salt Lake City, UT, USA). HR was also measured continuously using a smartphone application (Polar Beat, version 3.5.0, Polar Electro, Kempele, Finland) that was integrated with the chest strap. RPE was obtained at the end of each 2-min stage and immediately upon completion of the test using a numerical perception scale with verbal anchors (Borg and Noble 1974). Approximately 3–5 min after the end of the GXT, a 2-mL blood sample was drawn from a superficial forearm vein into a Vacutainer tube containing EDTA (BD Vacutainer, Becton, Dickinson and Co., Franklin Lakes, NJ, USA) for measurement of blood lactate in duplicate using a benchtop analyzer (YSI 2300 STAT Plus, Yellow Spring Instruments, OH, USA).

Participants completed a $\dot{V}O_{2max}$ plateau verification 20 min after the GXT to ensure a plateau in $\dot{V}O_2$ occurred. Participants exercised at the final power output achieved if they completed < 1 min of the final stage of the initial GXT or 25 W higher than the final power output achieved if they completed ≥ 1 min of the final stage of the initial GXT (Wingo et al. 2005). All subjects achieved a plateau using the verification protocol. All participants had to exhibit a $\dot{V}O_{2max}$ value ≥ 20th percentile for cycle ergometer-based testing for their sex and age (American College of Sports Medicine 2021) to remain in the study.

Experimental Trials

All experimental trials

The 4 remaining experimental trials were performed in an environmental chamber maintained at the appropriate temperature and RH based on the trial (15_{TEMP} = 21.9 ± 0.4 °C, 39% ± 3% RH; 15_{HOT} = 35.1 ± 0.4 °C, 38% ± 5% RH; 43_{TEMP} = 21.7 ± 0.4 °C, 39% ± 6% RH; 43_{HOT} = 34.9 ± 0.2 °C, 40% ± 2% RH). In addition to the aforementioned initial procedures for all trials, for the experimental trials, participants measured nude body mass and inserted a flexible rectal thermistor (MEAS 401, Measurement Specialties, Andover, MN, USA) 10 cm beyond the anal sphincter. The thermistor was integrated with wireless amplifiers (BioNomadix Wireless SKT Transmitter, Biopac Systems, Inc., Goleta, CA, USA) set to a sampling frequency of 1000 Hz and used to measure and record rectal temperature (T_{re}). T_{re} and ambient temperature were recorded continuously using a data acquisition system (MP150, Biopac Systems, Inc., Goleta, CA, USA) powered by data analysis software (AcqKnowledge 5.0, Biopac Systems, Inc., Goleta, CA, USA). Blood samples were drawn from a superficial forearm vein at the timepoints shown in Figure 2.1.

Participants began the HIIT session with an 8-min warm-up at 70% of HR_{max}. For the 15-min trials, after the warm-up, participants completed 1 round of HIIT (4 min at 90% HR_{max} and 3 min at 70% HR_{max}); THR was calculated based on the HR_{max} achieved during the control trial. During all experimental trials a member of the research team monitored HR and adjusted the work rate to maintain HR within 5 $b \cdot min^{-1}$ of THR during the entire workout. Participants were not able to see HR or power output during the HIIT session and were asked RPE at the end of the first and final (for 43-min trials) work and recovery intervals and at the end of the GXT. For the 43-min trials, participants completed 5 rounds of HIIT. All experimental trials were followed by

a GXT to determine $\dot{V}O_{2max}$ with no cessation of exercise; power output was increased to approximately half of the maximal power output observed during the control trial, with 25-W increases every 2 min thereafter until volitional exhaustion.

15_{TEMP} and 15_{HOT}

Participants entered an environmental chamber maintained at the appropriate temperature for the given trial and mounted the cycle ergometer. Next, remaining instrumentation was connected and baseline measurements were taken (~15 min). After baseline measurements, participants completed 1 of the 15-min trials (warm-up, 1 round of HIIT, and a GXT to measure $\dot{V}O_{2max}$).

43_{TEMP} and 43_{HOT}

For the 43_{TEMP} and 43_{HOT} trials, a flexible catheter was placed into a forearm vein for 2-mL blood sample collection before, during exercise at the end of the first (min 12) and final (min 40) high intensity bouts, and after the GXT as shown in Figure 2.1. Blood lactate concentrations were measured upon completion of the first and final work interval and the GXT while hematocrit (HCT) and hemoglobin (Hb) concentrations were measured prior to exercise and at min 40. HCT microcapillary tubes were centrifuged (Autocrit Ultra 3 Microhematocrit Centrifuge, model 420575, Becton, Dickinson and Co., Franklin Lakes, NJ, USA) and then assessed in triplicate using a microcapillary reader (Model 3201, International Equipment Co., Boston, MA, USA); Hb was measured in duplicate with a Hb analyzer (HemoPoint H2, EKF Diagnostics, Inc., Boerne, TX, USA). Together, HCT and Hb were used to calculate plasma volume change (Dill and Costill 1974).

After the indwelling catheter placement, four iButtons (model DS1921H, Embedded Data Systems, KY, USA) were secured to each participant's right side on their upper chest, lateral

deltoid, anterior thigh, and lateral calf to measure skin temperature with elastic therapeutic tape. Mean skin temperature ($\overline{T}_{sk}$) was calculated using the following equation (Ramanathan 1964):

$$\overline{T}_{sk} = 0.3(T_{chest} + T_{delt}) + 0.2(T_{thigh} + T_{calf}),$$

where T_{chest}, T_{delt}, T_{thigh}, and T_{calf} are the skin temperatures at the chest, deltoid, thigh, and calf, respectively. Mean body temperature ($\overline{T}_b$) was calculated using a weighted average of T_{re} and $\overline{T}_{sk}$ using the following equation (Stolwijk and Hardy 1966):

$$\overline{T}_b = 0.8(T_{re}) + 0.2(\overline{T}_{sk}).$$

The core-to-skin thermal gradient was calculated as the difference between rectal temperature and mean skin temperature ($T_{re} - \overline{T}_{sk}$). $\dot{V}O_2$ was measured during the first and final work interval as well as during the GXT. Metabolic rate was calculated for the first and final work intervals using the following equation (Kenny and Jay 2013):

$$M = (\dot{V}O_2[(((RER - 0.7)0.3^{-1})e_c) + (((1 - RER)0.3^{-1})e_f)]60^{-1},$$

where $\dot{V}O_2$ is the rate of oxygen uptake in $L \cdot min^{-1}$, e_c = 21,130 J (caloric equivalent per liter of oxygen for carbohydrate oxidation), e_f = 19,630 J (caloric equivalent per liter of oxygen for fat oxidation), and RER is respiratory exchange ratio. The difference between *M* and the external work rate on the cycle ergometer was calculated as the rate of metabolic heat production (*M – W)* and expressed in W (Kenny and Jay 2013).

Following the placement of the iButtons, participants entered an environmental chamber and mounted the cycle ergometer. Remaining instrumentation was then connected, baseline measurements were taken (~15 min) and participants completed 1 of the 43-min trials (warm-up, 5 rounds of HIIT and a GXT). At min 12 and 40 of exercise (i.e., at the start of recovery intervals) thermal sensation was obtained using a numerical scale (Young et al. 1987).

Participants were asked to rate the session RPE ~20 min following the completion of the exercise session (Foster et al. 2001).

Data analysis

All statistical analyses were performed using SPSS for Mac v.28.0.0.0 (IBM Corporation, Somers, NY). Mean (± SD) data were generated on the indicated outcome measures. To test the significance of mean differences in power output, a 2-way [condition × time (work intervals 1 and 5)] repeated measures analysis of variance (ANOVA) was used. Power output was also assessed by comparing the change in power output from the first work interval (min 9–11) to the final work interval (min 37–40) between the 43_{TEMP} and 43_{HOT} trials using a paired samples t-test. Likewise, paired samples t-tests were used to evaluate ΔT_{re} from baseline to min 43, from the final min of interval 1 to the final min of interval 5, and from baseline to the end of the GXT, as well as to compare differences in $\overline{T}_{sk}$, $\overline{T}_{b}$, $T_{re} - \overline{T}_{sk}$, and session RPE at the end of the GXT of the 43-min trials.

Baseline data for control and experimental trials were analyzed using a 1-way repeated ANOVA. Planned contrasts were performed to compare $\dot{V}O_{2max}$ from each of the experimental trials to the control trial using a Bonferroni correction to control for family-wise α. To evaluate if changes in $\dot{V}O_{2max}$ differed between experimental trials over time, a 2-way repeated measures ANOVA [condition × time (after 15-min and 43-min)] was conducted. To evaluate if $\dot{V}O_{2max}$ decreased by a greater magnitude in a hot or temperate environment, a paired samples t-test was used to compare change in $\dot{V}O_{2max}$ (from after 15 min to after 43 min). For other variables, such as HR, T_{re}, $\overline{T}_{sk}$, power output, RPE, and $\dot{V}O_2$, 2-way repeated measures ANOVAs [condition × time (work intervals 1 and 5 and/or recovery intervals 1 and 5)] were also conducted. For

relevant repeated measures ANOVAs, if sphericity was violated then the Greenhouse-Geisser correction was used.

In the event of a significant omnibus test, paired samples t-tests with a Bonferroni-adjusted α level (α′) were used for post hoc comparisons as appropriate. Effect sizes (ES) for pairwise comparisons were calculated using the following formula (Lakens 2013) for Cohen's d_{av} (Cohen 1988) adjusted for positive bias using Hedges's correction (g_{av}):

$$\text{ES} = \frac{Mean\ difference}{\frac{SD_1 + SD_2}{2}} \times \left(1 - \frac{3}{4(n \times 2) - 9}\right),$$

where SD_1 and SD_2 are the standard deviations of the respective time points or conditions and n is the number of pairs. Effect size values of 0.20 were interpreted as small, 0.50 as medium, and 0.80 as large (Caldwell and Cheuvront 2019; Fritz et al. 2012).

For select variables, a 95% confidence interval (CI) was calculated using a critical t (adjusted for multiple comparisons, if applicable) as follows (Weir and Vincent 2020):

$$\text{CI} = \text{Mean difference} \pm t_{cv}(SE_d),$$

where t_{cv} is the critical t value and SE_d is the standard error of the differences.

For power output, $\dot{V}O_2$, and $(M - W)$, the average over the entire interval was used for data analysis; for T_{re} and $\overline{T}_{sk}$, the average of the final min of the interval was used for data analysis; for HR, both the average over the entire interval and the average of the final min were analyzed. All statistical tests used an α level of 0.05.

RESULTS

Hydration

Based on U_{SG}, participants arrived adequately hydrated for all trials (control =1.005 ± 0.002, 15_{TEMP} = 1.004 ± 0.003, 43_{TEMP} = 1.005 ± 0.002, 15_{HOT} = 1.007 ± 0.006, 43_{HOT} = 1.007 ± 0.006; p = 0.65). Additionally, pre-exercise body mass was comparable among trials (control =

74.1 ± 8.3 kg, 15_{TEMP} = 74.1 ± 8.6 kg, 43_{TEMP} = 74.2 ± 8.9 kg, 15_{HOT} = 73.8 ± 7.8 kg, 43_{HOT} = 74.4 ± 9.1 kg; $p = 0.54$). Greater changes in body mass from before to after exercise were observed in the hot trials (15_{HOT} = –0.7% ± 0.4%, 43_{HOT} = –1.3% ± 0.8%) compared to temperate (15_{TEMP} = –0.4% ± 0.3%, 43_{TEMP} = –0.9% ± 0.6%; $p < 0.001$ for main effect of condition) and in the 43-min trials versus the 15-min trials ($p = 0.01$ for main effect of time). Although change in body mass was larger in the hot trials this did not result in a difference between the 43-min trials in percent change of plasma volume from pre- to post-exercise (43_{HOT} = –9.8% ± 2.5%, 43_{TEMP} = –8.4% ± 2.4%, $p = 0.31$, ES = 0.54).

Cardiovascular, metabolic, and perceptual responses during HIIT exercise

Heart rate responses

Given that exercise intensity was prescribed based on THR it is unsurprising that during the final min of the work intervals HR was the same during the first and final intervals in both environmental conditions ($p = 0.07$ for interaction effect; Table 2.1). However, from the first to final recovery interval, based on the final min of the respective intervals, HR increased 120% more in the hot (11 b·min^{-1}) compared to the temperate (5 b·min^{-1}) environment and resulted in HR being 10 b·min^{-1} higher in the hot environment at the start of the GXT ($p = 0.01$ for interaction effect; Table 2.1 and Figure 2.2A).

Table 2.1. Responses during the first (1) and fifth (5) work and recovery intervals.

	43_{TEMP}		43_{HOT}	
Work Interval	**1**	**5**	**1**	**5**
ΔPower output (%)	—	–18 ± 12	—	–33 ± 9†
Average HR (b·min^{-1}) §‡	157 ± 5	163 ± 7	160 ± 4	166 ± 8
Average %HR_{max} §‡	85 ± 1	88 ± 1	86 ± 1	89 ± 1
Final min HR (b·min^{-1})	168 ± 6	170 ± 6	170 ± 5	168 ± 7
$\dot{V}O_2$ (L·min^{-1}) §‡	2.4 ± 0.9	2.1 ± 1.0	2.3 ± 1.0	2.0 ± 0.7
Blood lactate (mmol·L^{-1})	3.6 ± 0.8	3.0 ± 1.0	3.5 ± 1.1	2.5 ± 1.1
RPE	16 ± 1	16 ± 1	17 ± 1	16 ± 1
Thermal sensation ‡	4.5 ± 1.0	5.0 ± 0.5	6.0 ± 0.5	7.0 ± 0.5
$M - W$ (W) §‡	677 ± 237	597 ± 272	650 ± 293	562 ± 207
Recovery interval	**1**	**5**	**1**	**5**
ΔPower output (%)	—	–23 ± 15	—	–15 ± 27†
Average HR (b·min^{-1}) §‡	141 ± 9	145 ± 9	146 ± 8	151 ± 10
Average %HR_{max} §‡	76 ± 3	78 ± 3	79 ± 2	81 ± 4
Final min HR (b·min^{-1})	130 ± 10	135 ± 11*	134 ± 11†	145 ± 12*†
RPE	11 ± 0	11 ± 1	11 ± 1	12 ± 2
ΔT_{re} (°C)	—	0.6 ± 0.2	—	0.8 ± 0.2†
$\overline{T}_{sk}$ (°C) §‡	33.0 ± 1.3	33.8 ± 1.4	36.2 ± 0.4	36.6 ± 0.6
$T_{re} - \overline{T}_{sk}$ (°C) ‡	4.6 ± 1.4	4.4 ± 1.4	1.2 ± 0.4	1.5 ± 0.6

ΔPower output = change in power output from the first to final interval; HR = heart rate either averaged over the interval or during the final minute of the interval as indicated; %HR_{max} = percent of maximal HR either averaged over the interval; $\dot{V}O_2$ = oxygen uptake; RPE = rating of perceived exertion; $M - W$ = rate of metabolic heat production; ΔT_{re} = change in rectal temperature from the final min of interval 1 to interval 5; $\overline{T}_{sk}$ = mean skin temperature during the final min of intervals 1 and 5; $T_{re} - \overline{T}_{sk}$ = core-to-skin thermal gradient during the final min of intervals 1 and 5. § $p < 0.05$ for main effect of time; ‡ $p < 0.05$ for main effect of condition; * $p < 0.05$ compared with interval 1 within the same condition; † $p < 0.05$ compared with temperate during the same interval. Target HR during work intervals = 167 ± 6 b·min^{-1}; Target HR during recovery intervals = 130 ± 5 b·min^{-1}.

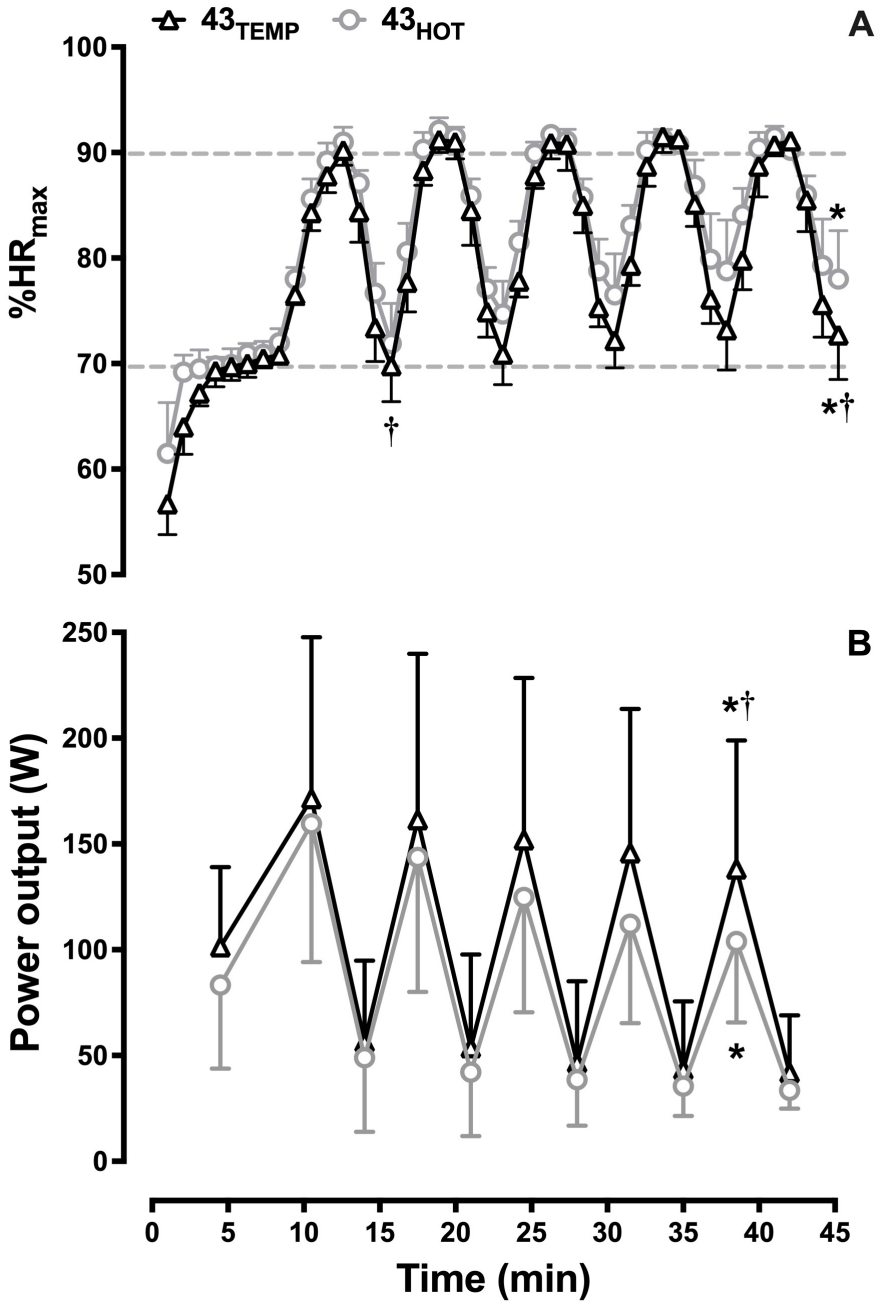


Figure 2.2. Percent maximal heart rate (mean ± SD) (%HR_{max}; Panel A) and power output (Panel B) during the 43 min trials. 43_{TEMP} = 43-min trial in a temperate environment; 43_{HOT} = 43-min trial in a hot environment. Peaks represent work intervals and nadirs represent recovery intervals. Gray dashed horizontal lines show the target heart rate for the work (90%) and recovery (70%) intervals. For power output, time points (x coordinates) represent the middle of the interval and values (y coordinates) were averaged over the entire interval. $^{*}p < 0.05$ compared with interval 1 (final min for panel A and average of the interval for panel B) in respective condition; $^{\dagger}p < 0.05$ compared to the same time in hot.

In the temperate environment, 6 participants were able to achieve the THR during the first 3-min recovery interval. However, during the last recovery interval, 3 participants were able to reach the THR. In the hot environment, 4 participants achieved the THR during the first interval but only 1 participant was able to achieve the THR during the 3rd, 4th, and 5th recovery intervals (Table 2.2, Figure 2.2A).

Table 2.2. Number of participants who achieved the targeted 70% maximum heart rate (HR) during the recovery intervals, the time in seconds it took to achieve the target HR (THR), the average power output and HR during each recovery interval, and the average THR for each interval.

	Recovery interval				
Variable	**1**	**2**	**3**	**4**	**5**
43_{TEMP} (n)	6	6	3	3	4
Time (s)	99 ± 23	138 ± 22	124 ± 23	135 ± 18	111 ± 19
Power (W)	61 ± 40	58 ± 47	68 ± 57	60 ± 49	50 ± 36
HR ($b \cdot min^{-1}$)	138 ± 6	141± 6	139 ± 3	141 ± 5	139 ± 5
THR ($b \cdot min^{-1}$)	130 ± 4	130 ± 4	128 ± 3	130 ± 1	129 ± 3
43_{HOT} (n)	4	2	1	1	1
Time (s)	120 ± 20	154 ± 13	120	133	97
Power (W)	55 ± 46	71 ± 56	88	68	53
HR ($b \cdot min^{-1}$)	142 ± 5	141 ± 4	136	134	134
THR ($b \cdot min^{-1}$)	129 ± 3	127 ± 3	125	125	125

Data are summarized as mean ± SD. 43_{TEMP} = 43-min trial in a temperate environment; 43_{HOT} = 43-min trial in a hot environment; HR = Heart rate; Target heart rate = THR.

Power output

As shown in Figure 2.2B, power output had to be lowered between the first and final work intervals to maintain THR in 43_{TEMP} (33 ± 20 W, $p = 0.005$, ES = 0.48) and in 43_{HOT} (56 ± 30 W, $p = 0.003$, ES = 1.07; $p = 0.008$ for interaction effect). The mean decrease was 70% greater in 43_{HOT} (95% CI for mean difference = 8, 36; $p = 0.008$, ES = 0.86). These changes in

power output resulted in a 25% (95% CI for mean difference = 12, 56; $p = 0.009$, ES = 0.64) lower power output in 43_{HOT} compared to 43_{TEMP} during the final work interval. During the recovery intervals, power output was not different across conditions and intervals ($p = 0.90$ for interaction effect).

Perceptual responses

Neither condition nor time affected RPE during the work ($p = 0.32$ for interaction effect) or recovery intervals ($p = 0.23$ for interaction effect). As expected, rating of thermal sensation was higher in the heat ($p < 0.01$). Lastly, session RPE was not different between the 43_{TEMP} and 43_{HOT} trials (both 8 ± 1; $p = 0.22$).

Thermoregulatory responses to HIIT exercise

T_{re} at baseline was not different among the 4 experimental trials (15_{TEMP} = 37.2 ± 0.3 °C, 43_{TEMP} = 37.3 ± 0.2 °C, 15_{HOT} = 37.3 ± 0.3 °C, 43_{HOT} = 37.1 ± 0.3 °C; $p = 0.22$). Although there was a significant interaction ($p = 0.01$), T_{re} was not different at min 15 ($p = 0.15$) or 43 ($p = 0.83$) between environmental conditions, but as expected it increased over time in both the hot ($p < 0.001$) and temperate ($p < 0.001$) environments (Figure 2.3). Furthermore, the ΔT_{re} from the end of the first to final recovery interval was 0.2 °C higher in the hot compared to temperate environment ($p = 0.01$ for t-test comparing change scores, ES = 0.94; 95% CI for mean difference = 0.1, 0.3; Table 2.1).

$\overline{T}_{sk}$ increased by 0.6 °C from the end of the first to the end of the final recovery interval across both conditions ($p = 0.009$ for main effect of time) and it was ~3.0 °C higher in the hot compared to temperate environment ($p < 0.001$ for main effect of condition; Table 2.1). The core-to-skin thermal gradient was 3.2 °C larger in the temperate compared to the hot environment across both time points (i.e., first and final recovery intervals; $p < 0.001$).

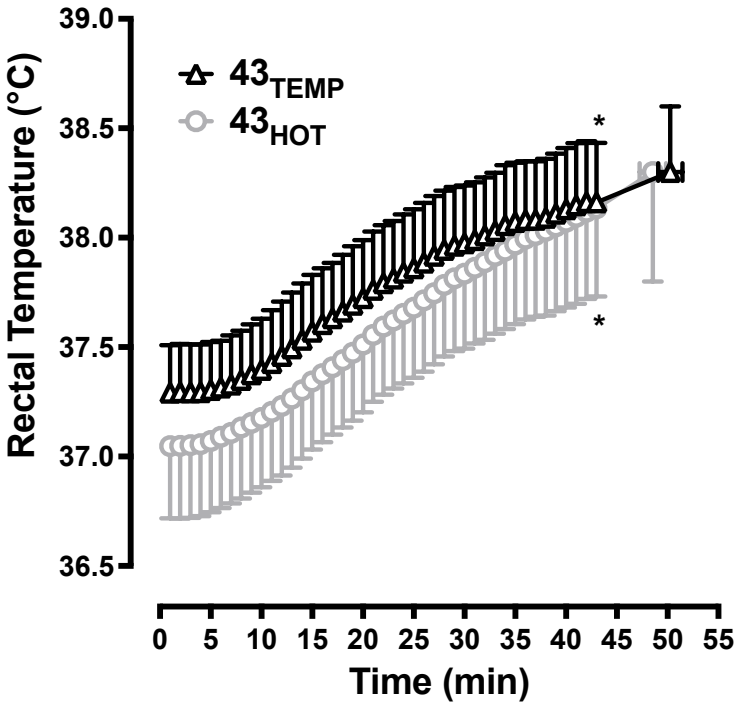


Figure 2.3. Rectal temperature (mean ± SD) from the start of exercise to the end of the graded exercise test. 43_{TEMP} = 43-min trial in a temperate environment; 43_{HOT} = 43-min trial in a hot environment. * $p < 0.05$ compared with min 15 of the same condition.

Maximal responses

Maximal responses to the 4 experimental trials are shown in Table 2.3 and Figure 2.4. Planned comparisons of control $\dot{V}O_{2max}$ (3.1 ± 1.2 L·min^{-1}) with $\dot{V}O_{2max}$ after each experimental trial revealed no differences [($\alpha' = 0.0125$); $p = 0.24$ for 15_{TEMP}; $p = 0.50$ for 15_{HOT}; $p = 0.42$ for 43_{TEMP}; $p = 0.014$ for 43_{HOT}]. $\dot{V}O_{2max}$ was 0.2 L·min^{-1} lower in the heat across time points (p = 0.001 for main effect of condition, ES = 0.17) and 0.2 L·min^{-1} lower over time across conditions ($p = 0.02$ for main effect of time, ES = 0.17). The decrease over time was not different between conditions ($p = 0.29$ for interaction; 95% CI for mean difference in change scores between 43_{HOT} and 43_{TEMP} = -0.1, 0.3; $p = 0.29$ for the t-test comparing change scores). Maximal power output was 21 W lower following the 43-min trials compared to the 15-min trials ($p = 0.006$ for main effect of time).

ΔT_{re} from baseline to end of GXT was 0.3 °C greater during 43_{HOT} versus 43_{TEMP} (p = 0.005, ES = 0.71). $\overline{T}_b$ was ~ 0.6 °C higher at the end of 43_{HOT} (37.9 ± 0.4 °C) compared to 43_{TEMP} (37.3 ± 0.4 °C; $p < 0.001$, ES = 1.40), which was mostly attributable to differences in $\overline{T}_{sk}$ since T_{re} was not different between 43-min trials at the end of the GXT (p = 0.91, ES = 0.00; Figure 2.3). At the end of the GXT, $\overline{T}_{sk}$ was 3.1 °C higher on average (36.6 ± 0.7 °C) in 43_{HOT} versus 43_{TEMP} (33.5 ± 1.4 °C; $p < 0.001$, ES = 2.62). The lower ambient temperature in 43_{TEMP} resulted in a larger $T_{re} - \overline{T}_{sk}$ (4.7 ± 1.4 °C) at the end of the GXT compared to 43_{HOT} (1.7 ± 0.7 °C; $p < 0.001$; ES = 2.68).

Table 2.3. Maximal responses during a graded exercise test following 15 min (after 1 work and recovery interval) and 43 min (after 5 work and recovery intervals) of high-intensity interval training in temperate and hot environments.

	Condition			
Variable	**15_{TEMP}**	**43_{TEMP}**	**15_{HOT}**	**43_{HOT}**
$\dot{V}_E$ (STPD, L·min^{-1}) $^{\#\dagger}$	93.4 ± 28.3	85.2 ± 23.5	85.3 ± 24.0	78.3 ± 23.2
$\dot{V}O_2$ (mL·kg^{-1}·min^{-1}) $^{\#\dagger}$	42.4 ±13.0	40.5 ±12.1	40.9 ± 13.4	37.5 ± 11.9
RER $^{\#\dagger}$	1.10 ± 0.04	1.04 ± 0.04	1.03 ± 0.04	1.01 ± 0.04
RPE	20 ± 0	20 ± 0	20 ± 0	20 ± 0
HR (b·min^{-1})	185 ± 8	185 ± 6	186 ± 7	185 ± 6
Blood lactate (mmol·L^{-1}) $^{\#}$	6.3 ± 2.4	5.3 ± 1.8	5.2 ± 1.3	3.9 ± 1.2
Test duration (min) $^{\#\dagger}$	8.7 ± 1.9	7.3 ± 1.2	7.2 ± 1.3	5.6 ± 1.3
Power output (W) $^{\#}$	211 ± 90	193 ± 76	200 ± 72	175 ± 65

15_{TEMP} = 15-min trial in a temperate environment; 15_{HOT} = 15-min trial in a hot environment; 43_{TEMP} = 43-min trial in a temperate environment; 43_{HOT} = 43-min trial in a hot environment; $\dot{V}_E$ = minute ventilation; $\dot{V}O_2$ = oxygen uptake; RER = respiratory exchange ratio; RPE = rating of perceived exertion; HR = heart rate. $^{\#}p < 0.05$ main effect of time; $^{\dagger}p < 0.05$ main effect of condition.

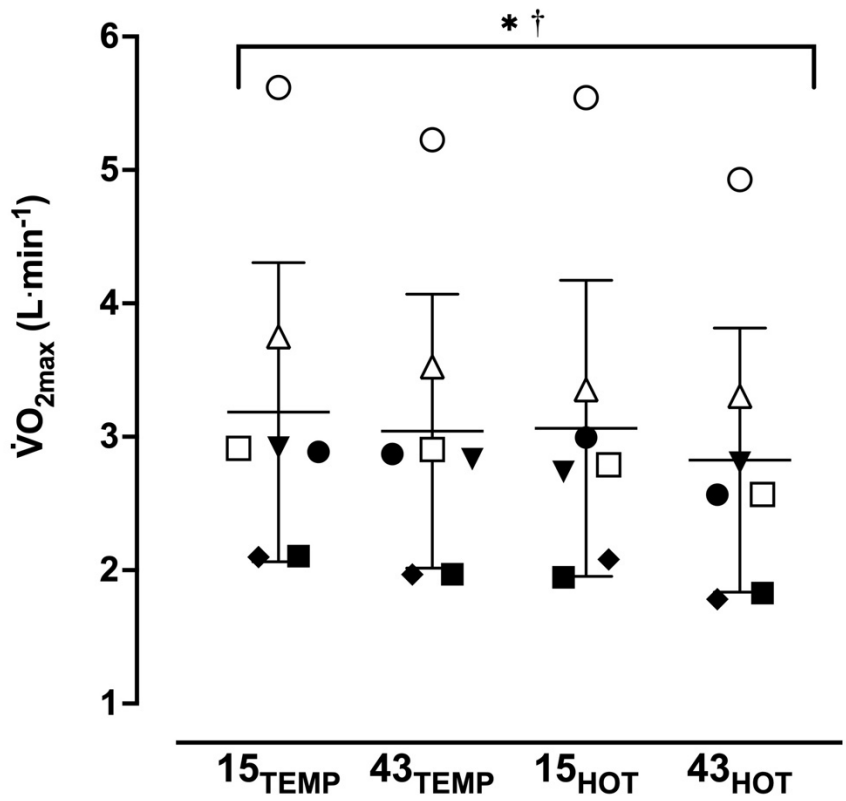


Figure 2.4. Vertical scattergram of maximal oxygen uptake ($\dot{V}O_{2max}$) during the experimental trials. Symbols represent data from individual participants and horizontal bars and accompanying error bars represent mean ± SD. * $p < 0.05$ main effect of time; † $p < 0.05$ main effect of condition.

DISCUSSION

The primary objectives of this study were to evaluate the magnitude of thermal and cardiovascular strain and accompanying work rate adjustments during HIIT when THR is used to prescribe exercise intensity in a temperate versus hot environment. A secondary objective was to evaluate changes in maximal aerobic capacity following 43-min of HIIT in the different environmental conditions. The main finding was that in order to maintain THR, power output had to be reduced by 18% in a temperate environment and nearly double that—33%—in a hot environment. T_{re} was not different between conditions at the end of the first and fifth recovery intervals, but the increase between intervals was larger in the heat. Even though THR was used to prescribe the exercise intensity, increased cardiovascular strain was observed during the

recovery intervals in the heat. Lastly, $\dot{V}O_{2max}$ decreased by a small amount following both 43-min trials. The large work rate adjustments attenuated thermal and cardiovascular strain but did not completely preserve $\dot{V}O_{2max}$.

By design the present study clamped HR over time by prescribing intensity based on THR. THR was successfully achieved and maintained during the work intervals, but as hypothesized, increased cardiovascular strain was observed during the recovery intervals in 43_{HOT} compared to 43_{TEMP} (Figure 2.2). This was evidenced by an 8% (11 b·min^{-1}) higher HR during the final minute of the last recovery interval compared to the first recovery interval in 43_{HOT}, while over the same time during 43_{TEMP}, HR increased only 4% (5 b·min^{-1}), despite similar work rates during recovery. For most participants, THR was unattainable during recovery intervals in the heat, even with work rate at 30 W (Table 2.2). Others using HIIT protocols that are not based on THR have also found that HR drifts upward during recovery intervals in temperate environments, but they found it drifts upward during work intervals too (Seiler and Hetlelid 2005; Thomas et al. 2020; Fennell and Hopker 2021). An increase in HR over time in hot conditions is often observed during constant intensity exercise as well (Stone et al. 2021; Wingo et al. 2019; Lafrenz et al. 2008). HR not only increases over time but is also elevated in the heat at a given intensity, just like we observed during the recovery intervals. The intensity of the recovery intervals may interact with the environmental temperature to influence the amount by which HR drifts upward. For example, during a HIIT protocol (5-s sprint, 115-s recovery) with a fixed recovery intensity of 50% peak $\dot{V}O_2$ ($\dot{V}O_{2peak}$), HR increased at a faster rate in a hot (5 b·min^{-1}) compared to temperate (3 b·min^{-1}**)** environment; however, at a fixed recovery intensity of 35% $\dot{V}O_{2peak}$, there was no difference in rate of change of HR between

environmental conditions (Maxwell et al. 2008). If the minimal work rate could have been lowered below 30 W in 43_{HOT}, recovery HR might have been similar between conditions.

Like cardiovascular strain, thermal strain (elevated core and skin temperatures) is observed during exercise in hot environments (Lafrenz et al. 2008; Galloway and Maughan 1997). Even though T_{re} did not differ statistically between the temperate and hot conditions at the respective time points, the increase over time was larger during 43_{HOT}. Another sign of thermal strain during 43_{HOT} was the elevated $\overline{T}_{sk}$ and smaller thermal gradient ($T_{re} - \overline{T}_{sk}$). Maxwell et al. (2008) also observed a greater rate of increase in core temperature during HIIT in a hot compared to a temperate environment, although THR was not used to prescribe the intensity. Unlike our results, cycle-based sprints (Drust et al. 2005) and shuttle run performance (Morris et al. 1998) in the heat resulted in higher T_{re} compared to a temperate environment. Furthermore, the ending T_{re} in the present study was less than what others have observed during HIIT in hot environments where participants had to stop exercising prematurely because core temperatures exceeded the laboratory safety limit (Maxwell et al. 2008; Morris et al. 1998). In our study, 1 participant had a core temperature exceed 38.5 °C during the HIIT session, indicating most participants experienced only modest hyperthermia (core temperature < 38.5 °C) (Ely et al. 2010); moreover, no participant had to end the exercise session prematurely.

The changes in work rate that limited thermal strain during both work and recovery intervals and cardiovascular strain during work intervals are comparable to what others have observed when using THR to prescribe cycle ergometer exercise intensity. During a similar HIIT protocol in a temperate environment, work rate had to be lowered by 21% (Morales-Palomo et al. 2017) and during a continuous 45-min exercise session in the heat, work rate had to be lowered by 37% (Wingo and Cureton 2006) in order to maintain THR. Périard and Racinais

(2015) also observed larger declines in power output in a hot (25%) compared to temperate (10%) environment during a repeated time trial performance lasting 60 min. The reductions in work rate in the present study were large in both conditions, but all participants—regardless of fitness level ($\dot{V}O_{2max}$ range = 29.7 $mL \cdot kg^{-1} \cdot min^{-1}$ to 68.2 $mL \cdot kg^{-1} \cdot min^{-1}$)—were able to complete the prescribed protocols. If a constant intensity had been used, it is likely participants would not have been able to complete the protocols, which is consistent with our pilot testing.

Despite work rate adjustments preventing a rise in HR over time during the work intervals, $\dot{V}O_{2max}$ decreased following 43 min of HIIT compared to 15 min by an average of 6.0% across both conditions (7.5% in the heat and 4.4% in the temperate environment). The 7.5% decrease in aerobic capacity in the hot condition is equivalent to the results of Wingo and Cureton (2006) where $\dot{V}O_{2max}$ also declined by 7.5% immediately following a 45-min continuous exercise session using THR to prescribe intensity in the heat. In contrast to our results, following 45 min of continuous exercise at 60% $\dot{V}O_{2max}$ in which HR was free to drift upward over time, the decline in maximal aerobic capacity was 15% in a hot and 5% (non-statistically significant) in a temperate environment (Lafrenz et al. 2008). Périard and Racinais (2015) also observed larger declines in maximal aerobic capacity in a hot compared to temperate environment during repeated time trials. Differences in the nature of HITT and continuous exercise or how the exercise was prescribed may explain the discrepant results with the studies cited above that used continuous exercise and different methods of exercise prescription.

The large decreases in work rate necessary to maintain THR during work intervals in the hot environment lowered ($M - W$) and likely resulted in the comparable T_{re} between conditions. In the first few min of 43_{HOT}, $\overline{T}_{sk}$ was lower than ambient temperature, so the ambient-to-skin thermal gradient favored heat gain by the skin, which resulted in higher $\overline{T}_{sk}$. As exercise

prolonged and the cutaneous vasculature dilated as part of thermoregulation, increased cutaneous blood flow likely also amplified $\overline{T}_{sk}$. Given the influence of ambient temperatures and $\overline{T}_{sk}$ on thermal perception, the elevated ambient and $\overline{T}_{sk}$ likely explains why participants reported feeling hotter in the heat (Gagge et al. 1968).

Why was the THR achieved at a lower work rate in the heat? Increased thermal strain in the hot condition was evidenced by elevated $\overline{T}_{sk}$, a narrowed $T_{re} - \overline{T}_{sk}$ thermal gradient, and greater perception of heat in 43_{HOT}. As mentioned above, greater thermal strain likely resulted in cutaneous vasodilation and increased skin blood flow (Brengelmann et al. 1977; Sawka et al. 2011). The peripheral displacement of blood volume (Rowell et al. 1969), potentially combined with direct effects of heat on sinoatrial node firing (Bolter and Atkinson 1988), increased sympathetic nervous system activity (Gorman and Proppe 1984), and increased catecholamine release (Kim et al. 1979), resulting in an increased HR at a given work rate both during work and recovery intervals.

Interestingly, the perceptual responses were similar between the two 43-min trials even though the work rate during the final work interval was 25% lower in the hot compared to temperate environment. This is similar to the results of Tucker et al. (2006) who found that to maintain an RPE of 16 during 30 min of constant cycling, work rate had to be lowered by a larger amount in a hot (≈ 27%) versus temperate (≈ 19%) environment, meaning RPE was the same despite different work rates. However, Glass et al. (1994) observed an upward drift in RPE during 30 min of exercise when intensity was held constant, but RPE was similar across differing environmental conditions (15 °C, 21 °C and 27 °C). The lower ambient temperature and shorter duration of the exercise session in the present study may explain the contrasting results. The equal perception of intensity we observed likely occurred because RPE is directly correlated to

HR in young healthy individuals and HR was maintained within 1 unit (10 b·min^{-1}) on the scale in both conditions (Borg and Noble 1974). Additionally, one of the consequences of exercise in the heat is an increased perception of intensity (Wingo et al. 2005; Wingo et al. 2020; Galloway and Maughan 1997) which could explain why RPE was similar despite lower work rates in the heat.

While cardiovascular drift (indexed as the rise in HR) was prevented during the work intervals of both conditions, this did not completely prevent declines in maximal aerobic capacity. Participants gave maximal efforts for each GXT because maximal HR and RPE were not different among trials. Furthermore, maximal (a-$\bar{v}$)O_2 was likely unaffected (Gonzalez-Alonso and Calbet 2003). While stroke volume (SV) was not measured, there may have been a decline in submaximal SV which was not overcome during maximal exercise and ultimately decreased $\dot{V}O_{2max}$ (Coyle and Gonzalez-Alonso 2001; Turkevich et al. 1988). At the end of HIIT in a temperate environment with a comparable protocol to the one in this study, Morales-Palomo et al. (2017) found that SV had declined. While we predicted the decline in $\dot{V}O_{2max}$ would be greater in the heat, it was only 0.1 L·min^{-1} lower. The larger reduction in work rate that limited cardiovascular and thermal strain in the heat was apparently sufficient to mitigate declines in $\dot{V}O_{2max}$, which is consistent with findings involving continuous exercise (Wingo and Cureton 2006).

The decline in aerobic capacity following the 43-min trials may have important implications for exercise performance and perception. During the GXT after the 43-min trials, power output was 10% lower compared to the 15-min trials. In terms of perception of intensity, during the final work interval, participants rated their perceived exertion about the same in the hot as in the temperate environment, but this was only achieved by lowering work rate by an

additional 23 W in the heat in order to maintain THR. If no work rate adjustments had been made, perception of intensity would have been higher, and perhaps more importantly, HR would likely have drifted to maximum, preventing participants from being able to complete the protocol.

Limitations

Although exercise in the heat can lead to heat acclimation, it is unlikely this occurred during this study. Only 2 trials took place in a hot environment, and they were performed on non-consecutive days which is below the amount of heat stress needed for short term heat acclimation [~7 days of consecutive heat stress (Pryor et al. 2018)]. Any systematic effects a change in acclimatization or acclimation status may have had were avoided because experimental trials were completed in a counterbalanced order.

Another limitation is that all of the participants were young and healthy individuals free of disease; as such, it is unclear if similar magnitudes of work rate adjustments and cardiovascular strain would have been observed in different populations, so generalizations should be made with caution. Notably, the results from the temperate environment were similar to those of Morales-Palomo et al. (2017) who also evaluated work rate adjustments and cardiovascular drift during a HIIT workout in middle aged adults with metabolic syndrome.

HIIT can be manipulated into many unique exercise sessions of varying durations and intensities. This study implemented longer duration work intervals (4 min) that were submaximal, so the results are likely not applicable to all types of HIIT where shorter durations and/or higher intensities are used. Additionally, it may not always be practical or possible to adjust the work rate to elicit the desired THR (Buchheit and Laursen 2013). Despite these limitations to using THR, it is still used to monitor and prescribe HIIT exercise training and in

the case of the present study, resulted in the exercise intensity being attainable across the varying fitness levels of the participants.

CONCLUSIONS

To maintain THR during a HIIT session, work rate had to be lowered by 18% and 33% in temperate and hot environments, respectively. This method of exercise prescription during HIIT may reduce the training stimulus, especially in hot environments, but simultaneously may limit thermal strain and allow participants to complete the prescribed workout. Although the work rate was lower in the heat, RPE was similar. Despite maintaining THR, a small decline in maximal aerobic capacity was observed following 43-min of HIIT in both conditions. Had an absolute intensity been used (e.g., a target power output corresponding to a target %$\dot{V}O_{2max}$) to prescribe the exercise intensity—in the hot or cool environment—cardiovascular drift would have likely rendered the target intensity unattainable. During constant rate submaximal exercise in the heat at a lower intensity (e.g., 60% $\dot{V}O_{2max}$), if cardiovascular drift is ignored, heat strain will increase and relative metabolic intensity will increase in conjunction with a decrease in $\dot{V}O_{2max}$, but exercise can nonetheless be sustained for a prolonged period (Wingo et al. 2020; Wingo and Cureton 2006). However, based on the results of this study, if cardiovascular drift is ignored during HIIT exercise, even in a temperate environment, it is unlikely participants could complete the session. Exploring different methods of exercise prescription during HIIT, such as using RPE to gauge intensity or implementing passive recovery periods, could allow for maintaining higher intensities during the work intervals while also allowing the exercise protocols to remain physically possible. These methods of exercise prescription will need to be balanced by the understanding that a higher work rate will likely result in increased cardiovascular and thermal strain, possibly increasing the risk of heat illness if the session is prolonged. When prescribing

HIIT in the heat using THR, consideration should be given to the magnitude of expected work rate adjustments and the accompanying decreases in training stimulus.

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CHAPTER 3

WORK RATE ADJUSTMENTS DURING HEART RATE- AND RPE-BASED HIGH-INTENSITY INTERVAL TRAINING IN THE HEAT

INTRODUCTION

The intensity of work and rest intervals during high-intensity interval training (HIIT) can be prescribed using work rate (speed or power output), oxygen uptake ($\dot{V}O_2$), metabolic equivalents, heart rate (HR), or rating of perceived exertion (RPE). Each method has different advantages and disadvantages. For instance, using work rate can be problematic because a single velocity can represent varying metabolic demands depending on the terrain and environmental conditions, and some speeds may not be attainable in certain conditions such as high winds, steep hills, or oppressive heat. Furthermore, outside of a laboratory, prescribing intensity using $\dot{V}O_2$ is impractical because of expensive and cumbersome equipment needed if $\dot{V}O_2$ is to be measured directly and general unfamiliarity with using metabolic equations if $\dot{V}O_2$ is to be estimated.

Because of these limitations and as a result of its ease of use and linear relationship with $\dot{V}O_2$ (Swain et al. 1994), target HR (THR) is often used for prescribing intensity of HIIT (Morales-Palomo et al. 2017; Arazi et al. 2017; Helgerud et al. 2007). However, using THR to gauge exercise intensity is complicated by a phenomenon known as cardiovascular drift whereby a progressive increase in HR occurs over time despite no change in work rate. Under conditions in which cardiovascular drift occurs, work rate must be lowered to maintain THR, which can compromise the training stimulus and adaptations (Wingo 2015; Morales-Palomo et al. 2017;

Wingo and Cureton 2006b). Historically cardiovascular drift has been applied to conditions of prolonged, continuous, moderate-intensity exercise but more recently it has been observed during HIIT in a temperate environment (24 °C) (Morales-Palomo et al. 2017). Using THR to prescribe exercise intensity during HIIT in hot conditions may be especially problematic because heat stress may amplify cardiovascular drift during HIIT like it does during continuous exercise (Lafrenz et al. 2008).

A simple alternative to using THR to prescribe intensity of a HIIT session is to use RPE, a subjective measure of intensity (Borg and Noble 1974). RPE is an appealing method of prescribing exercise intensity because it requires no equipment, allows the individual to adjust the intensity based on how the intensity of exercise is perceived, and in young healthy individuals, is directly related to HR (Borg 1982). RPE has been repeatedly shown to be a valid method to gauge exercise intensity in temperate conditions (Eston and Williams 1988; Edwards et al. 1972; Dunbar et al. 1992). During constant-intensity exercise in the heat, however, RPE is elevated compared to cooler environments (Maw et al. 1993), and, like HR, it may progressively increase over time despite no change in work rate (Pandolf 1998; Wingo and Cureton 2006a; Wingo et al. 2005; Wingo et al. 2020). Therefore, like THR, to maintain target RPE in hot environments, work rate must be lowered to a larger extent compared to that in cooler environments (Tucker et al. 2006; Roussey et al. 2018). Nonetheless, the magnitude by which work rate needs to be lowered to maintain target RPE in the heat appears to be less than that to maintain THR (Tucker et al. 2006). Consequently, using target RPE to gauge exercise intensity during HIIT in the heat may be advantageous compared to using THR because of maintenance of a higher work rate and thereby training stimulus, but this has not been evaluated. Additionally, this will likely result in the unintended consequences of higher core body temperature and

amplified cardiovascular strain (indexed as cardiovascular drift), but no study has addressed the extent to which this may occur.

In addition to the aforementioned considerations regarding cardiovascular drift, a consequence of cardiovascular drift is that it corresponds to reduced maximal oxygen uptake ($\dot{V}O_{2max}$) during continuous exercise in hot conditions (Wingo et al. 2005; Lafrenz et al. 2008). This has implications for how exercise is perceived (e.g., if $\dot{V}O_{2max}$ declines during an exercise bout, a given work rate momentarily represents a greater proportion of $\dot{V}O_{2max}$ and therefore will be perceived as more taxing). Since elevated core and skin temperatures and accompanying cardiovascular drift are associated with declines in $\dot{V}O_{2max}$ (Wingo et al. 2005; Nybo et al. 2001; Cheuvront et al. 2010), and since HIIT based on RPE is expected to result in higher core and skin temperatures—and thereby greater cardiovascular drift—then HIIT based on RPE is expected to also result in larger declines in $\dot{V}O_{2max}$ compared to HIIT based on HR, but this has not been tested.

Given the preceding notions, the purposes of this study were to test the hypotheses that 1) work rate must be lowered to a greater extent to maintain THR than to maintain target RPE during HIIT in a hot environment, 2) greater thermal and cardiovascular strain will result from maintaining target RPE compared to THR during a HIIT workout in the heat, and 3) $\dot{V}O_{2max}$ will decrease to a greater extent after HIIT in the heat when exercise intensity during HIIT is based on target RPE compared to THR.

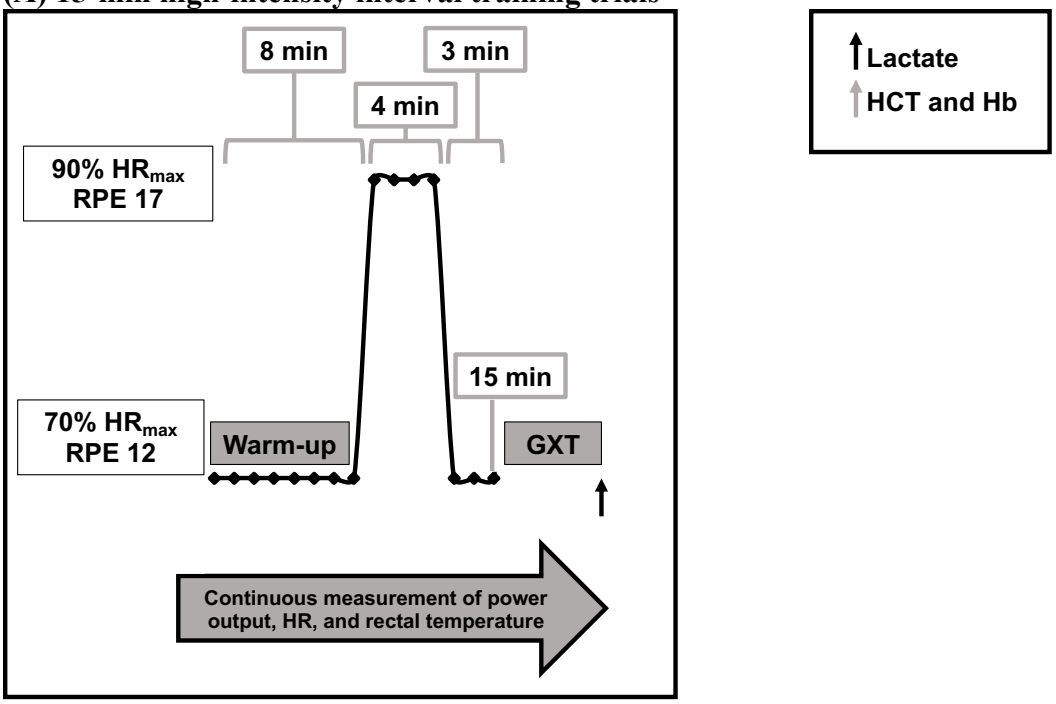
METHODS

Experimental design

Participants visited the laboratory on 5 separate occasions and completed 1 trial at each visit (1 control trial, two 15-min experimental trials, and two 43-min experimental trials) on a

cycle ergometer (LC6 Novo, Monark Exercise, Vansbro, Sweden). The first visit was a control trial; participants completed a graded exercise test (GXT) to measure maximal HR (HR_{max}) and $\dot{V}O_{2max}$ in a temperate environment [22.6 ± 0.6 °C, 37% ± 6% relative humidity (RH)]. The remaining 4 experimental trials were completed in a counterbalanced order and in a hot environment (35.1 ± 0.3 °C, 40% ± 4% RH). Counterbalanced treatment orders were randomly assigned to the participants. Figure 3.1 depicts a general overview of the experimental trials. The experimental trials consisted of an 8-min warm-up at 70% HR_{max} or an RPE of 12 followed by 1 (for 15-min trials) or 5 (for 43-min trials) rounds of HIIT (4 min at 90% HR_{max} or RPE of 17 and 3 min at 70% HR_{max} or RPE of 12); 15_{HR} = warm-up and 1 round of HIIT based on HR, 15_{RPE} = warm-up and 1 round of HIIT based on RPE, 43_{HR} = warm-up and 5 rounds of HIIT based on HR, and 43_{RPE} = warm-up and 5 rounds of HIIT based on RPE. If 70% HR_{max} or an RPE of 12 could not be achieved during the rest intervals (because of thermal and cardiovascular strain), participants cycled at 30 W with a cadence ≥ 30 $rev \cdot min^{-1}$ to ensure the intensity was distinguishable from passive recovery. Upon the completion of each experimental trial participants immediately began a GXT at approximately half of maximal power output observed during the control trial to measure $\dot{V}O_{2max}$. The purpose of the separate 15- and 43-min trials was to evaluate $\dot{V}O_{2max}$ before (15-min trials, after 1 round of HIIT) and after (43-min trials, after 5 rounds of HIIT) work rate adjustments had been made to maintain target intensity.

(A) 15-min high-intensity interval training trials



(B) 43-min high-intensity interval training trials

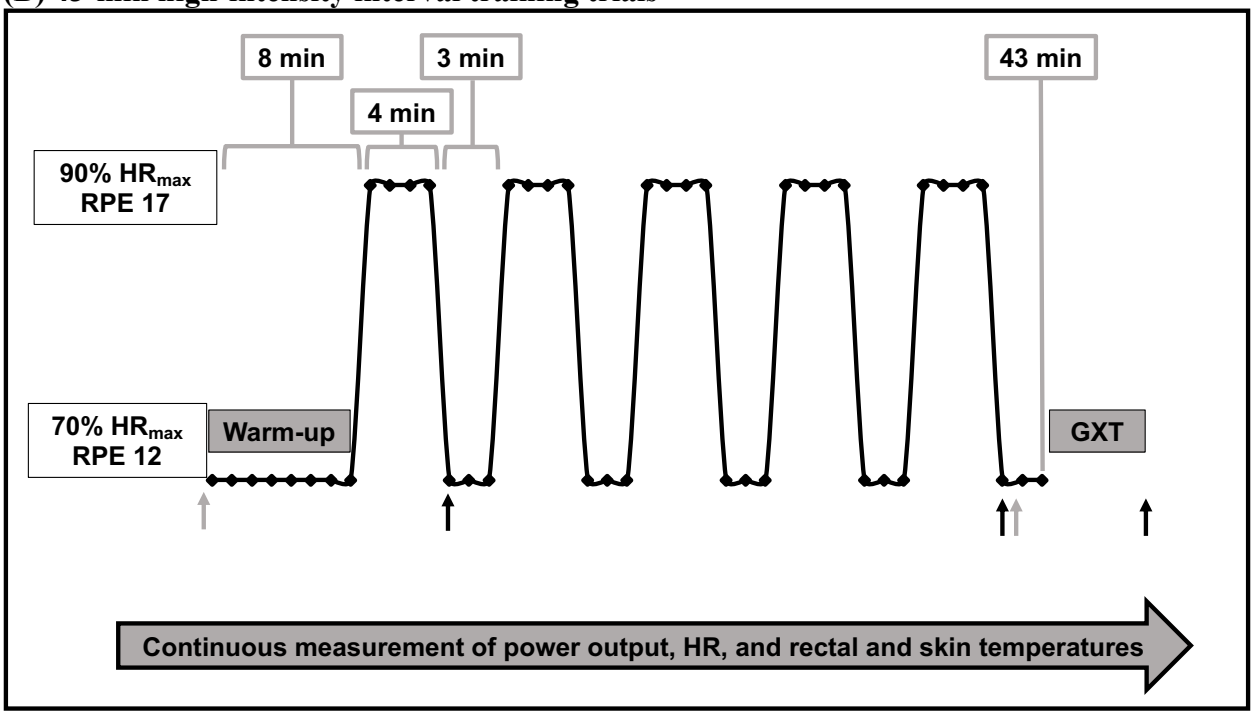


Figure 3.1. General exercise protocols for the (A) 15- and (B) 43-min experimental trials. GXT, graded exercise test; $\dot{V}O_2$, oxygen uptake; HR, heart rate; RPE, rating of perceived exertion; HCT, hematocrit; Hb, hemoglobin.

Participants

An a priori power analysis (G*power 3.1.9.6) revealed a sample size of 7 would be sufficient to detect a 25-W (effect size = 1.4) difference between the change score in power output from the first to the final work interval in 43_{HR} versus 43_{RPE}, assuming $\alpha = 0.05$ and power ≈ 0.80 (Faul et al. 2009; Faul et al. 2007). Nine participants provided written informed consent and 1 participant withdrew from the study following the first visit. Eight healthy adults (4 men and 4 women; 18–38 y) free of disease completed all 5 visits. Seven were recreationally active as defined by the American College of Sports Medicine (2021) (i.e., exercising aerobically ≥ 30 min per day, ≥ 3 times per week, for the past ≥ 3 months) and 1 was a competitive endurance athlete. Physical characteristics of participants were age (mean ± SD) = 25 ± 7 y, body mass = 74.1 ± 8.3 kg, height = 181 ± 10 cm, percent body fat = 21.4% ± 8.4%, HR_{max} = 185 ± 5 $b \cdot min^{-1}$, 70% HR_{max} = 130 ± 5 $b \cdot min^{-1}$, 90% HR_{max} = 166 ± 7 $b \cdot min^{-1}$.

Women with a regular menstrual cycle lasting 21–35 days were included (Elliott-Sale et al. 2021). They were asked to self-report the first and last day of previous menses and contraceptive use for data analysis and scheduling. All experimental trials were scheduled during the same phase of their menstrual cycle (luteal phase or follicular phase), although the specific phase was not expected to affect study outcomes (Stone et al. 2021). Two of the 4 women completed the experimental trials in the luteal phase of their menstrual cycle. Although we did not confirm phase of menstrual cycle via hormonal assay, based on rectal temperature it is likely 1 woman completed the 43-min trials in the follicular phase and the 15-min trials in the luteal phase. One woman who was using an oral contraceptive [norgestimate (0.25 mg) and ethinyl estradiol (0.035 mg)] was tested in her follicular phase.

Procedures

All trials

For each of the 5 trials, participants were instructed to abstain from consuming alcohol or participating in strenuous exercise during the 24 h prior to testing. Additionally, participants were instructed to report to the laboratory after a 2-h fast, well rested, euhydrated, and having refrained from ingesting non-prescription drugs and caffeine on the day of testing. Adherence to pre-testing instructions were confirmed using a 24-h history questionnaire.

Upon arrival, participants provided a urine sample that was analyzed for urine specific gravity (U_{SG}) using a digital refractometer (ATAGO PAL-10S digital refractometer, Tokyo, Japan). U_{SG} had to be ≤ 1.020 for a participant to be considered adequately hydrated (Sawka et al. 2007). Participants whose U_{SG} values were > 1.020 were given water to ingest for 20–30 min and then reevaluated. Then participants donned padded cycling shorts, a mesh tank top, and a chest strap HR monitor (H10, Polar Electro, Kempele, Finland) that paired with a smart phone application (Polar Beat, version 3.5.0, Polar Electro, Kempele, Finland). Prior to beginning exercise, the Borg 6–20 RPE scale was explained using standardized instructions (Borg and Noble 1974). Approximately 3–5 min after the end of each GXT a 2-mL blood sample was drawn from a superficial forearm vein into a Vacutainer tube containing EDTA (BD Vacutainer, Becton, Dickinson and Co., Franklin Lakes, NJ, USA) for the measurement of blood lactate (YSI 2300 STAT Plus, Yellow Spring Instruments, OH, USA). During the trials, researchers provided verbal encouragement to participants.

A minimum of 24 h separated control trials from subsequent experimental trials and at least 48 h separated experimental trials from one another. All trials for a given participant took

place over ≤ 8 weeks, and each was completed at a similar time (± 2 h) of day to control for fluctuations in core body temperature associated with circadian rhythm (Moore-Ede et al. 1983).

Control trial

At the first visit, participants completed a questionnaire about their readiness to participate in exercise, a general health history form, and they provided written informed consent. Next, height was measured using a stadiometer (SECA 213, Seca Ltd., Hamburg, Germany) and body mass was measured with a digital scale (Tanita WB-800S, Tanita Corp., Tokyo Japan). Body fat percentage was calculated from the sum of 3 skinfolds (Jackson and Pollock 1985).

Participants then began a self-selected warm-up for 5-10 min on the cycle ergometer. Next, the GXT started at a moderate intensity selected by the researcher based on RPE and HR observed during the warm-up. Every 2 min the power output on the cycle ergometer was increased 25 W until volitional exhaustion was reached or pedal cadence fell below 30 rev·min^{-1}. $\dot{V}O_2$ was measured continuously using open circuit spirometry (Parvo Medics Metabolic Measurement System, model TrueOne 2400, Salt Lake City, UT, USA). The highest 1-min average was considered $\dot{V}O_{2max}$. During the GXT, HR was measured continuously and HR_{max} was the highest 1-s value achieved during the test. This value was then used to calculate the THR for the experimental trials.

Twenty min following the GXT, participants remounted the cycle ergometer and completed a $\dot{V}O_{2max}$ plateau verification protocol in which they cycled to volitional exhaustion. Those who completed < 1 min of the final stage of the initial GXT performed the verification protocol at the final power output achieved during the initial GXT; those who completed ≥ 1 min of the final stage of the initial GXT performed the verification protocol at a power output 25 W

higher than that achieved during the final stage of the initial GXT (Wingo et al. 2005). To remain in the study all participants had to exhibit a $\dot{V}O_{2max}$ value ≥ 20th percentile for cycle ergometer-based testing for their sex and age (American College of Sports Medicine 2021).

Experimental trials – general procedures

At least 24 h following the control trial, participants returned to the laboratory for the first experimental trial. In addition to the procedures outlined under "all trials," for the experimental trials, participants measured nude body mass and inserted a flexible rectal thermistor 10 cm beyond the anal sphincter for measurement of rectal temperature (T_{re}). The thermistor was integrated with wireless amplifiers (BioNomadix Wireless SKT Transmitter, Biopac Systems, Inc., Goleta, CA, USA) set to a sampling frequency of 1000 Hz. T_{re} and ambient temperature were recorded continuously using a data acquisition system (MP150, Biopac Systems, Inc., Goleta, CA, USA) powered by data analysis software (AcqKnowledge 4.2, Biopac Systems, Inc., Goleta, CA, USA). During trials based on HR, a member of the research team monitored HR and adjusted the workload to maintain HR within 5 b·min^{-1} of THR during the entire workout. During trials based on RPE the participant adjusted the power output (without being able to view the value) to match the given RPE. The RPE scale was continuously visible to participants throughout the exercise sessions and participants were frequently reminded to adjust resistance to remain at the prescribed intensity. During the HR-based trials, participants were instructed to point to a value on the chart that matched their RPE at the end of each interval; the value was verbally confirmed by a member of the research team.

All blood samples taken before, during, and after the experimental trials were drawn from a superficial forearm vein into a Vacutainer containing EDTA for measurement of either lactate concentration, hematocrit (HCT) and hemoglobin (Hb), or both, as specified in Figure 3.1. HCT

was assessed in triplicate using a microcapillary reader (Model 3201, International Equipment Co., Boston, MA, USA); Hb was assessed in duplicate using a Hb analyzer (HemoPoint H2, EKF Diagnostics, Inc., Boerne, TX, USA). HCT and Hb were then used to calculate plasma volume change (Dill and Costill 1974).

After the last round of HIIT recovery, participants immediately began a GXT to determine $\dot{V}O_{2max}$. Power output was set to approximately half of the maximal power output achieved during the control GXT. Thereafter, power output was increased by 25 W every 2 min until participants could no longer continue.

15_{HR} and 15_{RPE}

Participants entered the environmental chamber and mounted the cycle ergometer. Next, instrumentation was connected and baseline measurements were taken (~15 min). After baseline measurements, participants completed 1 of the 15-min trials, which included a warm-up and 1 round of HIIT followed by a GXT to determine $\dot{V}O_{2max}$.

43_{HR} and 43_{RPE}

For the 43_{HR} and 43_{RPE} trials, skin temperature was measured using 4 iButtons (model no. DS1921H, Embedded Data Systems, KY, USA) taped to each participant's upper chest, lateral deltoid, anterior thigh, and lateral calf with elastic therapeutic tape. Skin temperatures from these sites were then used to calculate mean skin temperature ($\overline{T}_{sk}$) using the following equation (Ramanathan 1964):

$$\overline{T}_{sk} = 0.3(T_{chest} + T_{delt}) + 0.2(T_{thigh} + T_{calf}),$$

where T_{chest}, T_{delt}, T_{thigh}, and T_{calf} are the skin temperatures at the chest, deltoid, thigh, and calf, respectively. Mean body temperature ($\overline{T}_b$) was calculated using a weighted average of T_{re} and $\overline{T}_{sk}$ using the following equation (Stolwijk and Hardy 1966):

$$\overline{T}_b = 0.8(T_{re}) + 0.2(\overline{T}_{sk}).$$

The core-to-skin thermal gradient was calculated as the difference between rectal temperature and mean skin temperature ($T_{re} - \overline{T}_{sk}$). $\dot{V}O_2$ was measured during the first and final work interval and the GXT. Metabolic rate was estimated for the first and final work intervals using the following equation (Kenny and Jay 2013):

$$M = (\dot{V}O_2[(((RER - 0.7)0.3^{-1})e_c) + (((1 - RER)0.3^{-1})e_f)]60^{-1},$$

where $\dot{V}O_2$ is the rate of oxygen uptake in $L \cdot min^{-1}$, e_c = 21,130 J (caloric equivalent per liter of oxygen for carbohydrate oxidation), e_f = 19,630 J (caloric equivalent per liter of oxygen for fat oxidation), and RER is respiratory exchange ratio. The difference between *M* and the external work rate on the cycle ergometer was estimated as the rate of metabolic heat production (*M – W)* and expressed in W (Kenny and Jay 2013).

Next, a flexible catheter was placed into a forearm vein for 2-mL blood sample collection before, during the trial at time points corresponding to the end of the high intensity bouts (min 12 and 40), and after exercise. Blood lactate concentration was measured at the end of the first and fifth work interval and upon completion of the GXT. HCT and Hb were measured at baseline and at the end of the final work interval.

Following the placement of the catheter, participants entered the environmental chamber and mounted the cycle ergometer. Remaining instrumentation was then connected, a 2-mL blood sample was drawn, and other baseline measurements were taken (~15 min). Participants then began one of the 43-min trials (warm-up, 5 rounds of HIIT, and GXT). At min 12 and 40 participants were asked to report their thermal sensation on a numerical scale (Young et al. 1987). Approximately 20 min after the exercise session, participants were asked to rate the session RPE (Foster et al. 2001).

Data analysis

Mean (± SD) data were generated on the indicated outcome measures. To test the significance of mean differences in power output, a 2-way [condition × time (work intervals 1 and 5)] repeated measures analysis of variance (ANOVA) was used. Power output was also assessed by comparing the change in power output from the first work interval (min 9–11) to the final work interval (min 37–40) between the 43_{HR} and 43_{RPE} trials using a paired samples t-test. Paired samples t-tests were also used to evaluate the difference in T_{re}, $\overline{T}_{sk}$, $\overline{T}_{b}$, $T_{re} - \overline{T}_{sk}$, and session RPE at the end of the GXT of the 43-min trials.

Baseline data for control and experimental trials were analyzed using a 1-way repeated measures ANOVA. Planned contrasts were performed to compare $\dot{V}O_{2max}$ from each experimental trial to the control trial with a Bonferroni correction to control for family-wise α. Two-way repeated measures ANOVAs [condition × time (after 15 and 43 min)] were used to analyze $\dot{V}O_{2max}$ and other variables at the completion of the GXT after 15 min (1 round of HIIT) compared to after 43 min (5 rounds of HIIT). To evaluate if $\dot{V}O_{2max}$ decreased by a larger amount depending on method of exercise prescription (THR or RPE), a paired samples t-test was used to compare change in $\dot{V}O_{2max}$ (from after 15 min to after 43 min).

For hematological variables, 2-way (condition × time) repeated measures ANOVAs were conducted. For other variables, such as HR, T_{re}, $\overline{T}_{sk}$, power output, and $\dot{V}O_{2}$, 2-way repeated measures ANOVAs [condition × time (work intervals 1 and 5) and/or condition × time (recovery intervals 1 and 5)] were conducted.

In the event of a significant omnibus test, paired samples t-tests with a Bonferroni-adjusted α level (α′) were used for post hoc comparisons as appropriate. Effect sizes (ES) for

paired samples t-tests were calculated using the following formula (Lakens 2013) for Cohen's d_{av} (Cohen 1988) adjusted for positive bias using Hedges's correction (g_{av}):

$$\text{ES} = \frac{Mean\ difference}{\frac{SD_1 + SD_2}{2}} \times \left(1 - \frac{3}{4(n \times 2) - 9}\right),$$

where, SD_1 and SD_2 are the standard deviations of the respective time points or conditions and n is the number of pairs. ES were interpreted as: 0.20 = small, 0.5 = medium, and 0.80 = large (Caldwell and Cheuvront 2019; Fritz et al. 2012).

For select variables, a 95% confidence interval (CI) was calculated for the mean difference between conditions (for pairwise comparisons of interest) using a critical t (adjusted, if applicable, to keep the family-wise α at 0.05) in the following formula (Weir and Vincent 2020):

$$\text{CI} = \text{Mean difference} \pm t_{cv}(SE_d),$$

where t_{cv} is the critical t value (adjusted for multiple comparisons, if applicable) and SE_d is the standard error of the differences.

For power output, $\dot{V}O_2$, and $(M - W)$, the average over the entire interval was used for data analysis; for T_{re} and $\overline{T}_{sk}$, the average of the final min of the interval was used for data analysis; for HR, both the average over the entire interval and the average of the final min were analyzed. All statistical tests used an α level of 0.05 and analyses were performed using SPSS for Mac v.28.0.0.0 (IBM Corporation, Somers, NY).

RESULTS

Hydration

Participants were adequately hydrated prior to all trials (U_{SG}, control = 1.005 ± 0.002, 15_{HR} = 1.007 ± 0.005, 15_{RPE} = 1.005 ± 0.004, 43_{HR} = 1.006 ± 0.006, 43_{RPE} = 1.006 ± 0.003; p = 0.82). Additionally, pre-exercise body mass was comparable among trials (control = 73.9 ± 7.9

kg, 15_{HR} = 73.9 ± 7.6 kg, 15_{RPE} = 74.4 ± 8.8 kg, 43_{HR} = 74.3 ± 8.9 kg, 43_{RPE} = 74.0 ± 8.0 kg; p = 0.68). Percent change in body mass from before to after exercise for each experimental trial was 15_{HR} = –0.7% ± 0.4%, 15_{RPE} = –0.6% ± 0.4%, 43_{HR} = –1.3% ± 0.8%, and 43_{RPE} = –1.3% ± 0.8% (p = 0.004 for main effect of time). There were no differences in percent change in plasma volume pre- to post-HIIT exercise between 43-min trials (43_{HR} = –9.0% ± 3.3%, 43_{RPE} = –9.5% ± 3.9%, p = 0.67, ES = 0.13).

Cardiovascular, work rate, metabolic, and perceptual responses during HIIT exercise

Heart rate responses

As designed, during the work intervals of the HR-based trial, HR during the final min did not increase from the first to final interval (p = 0.36), and THR was achieved. In contrast, during 43_{RPE}, HR increased by 12 b·min^{-1} (p < 0.001; p = 0.01 for interaction effect) from the first to final work interval. During the final min of recovery intervals across both 43-min trials, HR increased by 11 b·min^{-1} from the first to final recovery interval (p < 0.001 for main effect of time), and HR was 13 b·min^{-1} higher during the RPE-based trial (p = 0.005 for main effect of condition; p = 0.08 for interaction). HR during the final min of the first and fifth recovery intervals increased from 72% to 78% HR_{max} in the HR trial and from 79% to 85% HR_{max} in the RPE trial (p < 0.001 for main effect of time; p = 0.006 for main effect of condition). Similar patterns were observed for $\%HR_{max}$ averaged over the entire work and recovery intervals and are shown in Figure 3.2A.

During 43_{HR}, 4 participants were able to reach target HR during the first recovery interval, 3 in the second, and the same 2 participants for the final 3 recovery intervals. During the 43_{RPE} trial, 2 participants cycled at the minimum 30 W for the final 2 intervals. Two participants

reached or surpassed the HR_{max} observed in the control trial during the HIIT portion of the 43_{RPE} trial.

Power output

Across both 43-min trials, power output had to be lowered by 38 W (ES = 0.59) between the first and fifth work intervals ($p = 0.002$ for main effect of time) to maintain the target intensity, but conditions were not statistically different ($p = 0.35$ for main effect of condition; $p = 0.26$ for interaction; Figure 3.2B). Likewise, the t-test comparing the change score between the first and fifth work interval for 43_{HR} (–46 ± 29 W) and the change score between the first and fifth work interval for 43_{RPE} (–30 ± 28 W) was not statistically significant ($p = 0.26$; 95% CI for mean difference = –45, 15), but the magnitude of difference between these change scores was moderate (16 ± 36 W; ES = 0.53). During the recovery intervals power output was 22 W lower during the fifth compared to first interval in both conditions ($p = 0.01$ for main effect of time; ES = 0.79) and 28 W lower (ES = 1.00) in the HR-based trial across the first and final recovery intervals ($p = 0.001$ for main effect of condition; $p = 0.16$ for interaction).

Metabolic responses

Absolute $\dot{V}O_2$ was not different over time ($p = 0.16$ for interaction; $p = 0.09$ for main effect of time; Table 3.1). Furthermore, even though the experimental conditions were based on different methods of gauging exercise intensity, they elicited comparable $\dot{V}O_2$ ($p = 0.19$ for main effect of condition).

Perceptual responses

Thermal sensation increased from 6.0 to 7.0 from the end of the first work interval to the final work interval across both 43-min trials ($p = 0.006$ for main effect of time). Likewise, session RPE was similar between 43_{HR} (8 ± 1) and 43_{RPE} (9 ± 1) ($p = 0.44$).

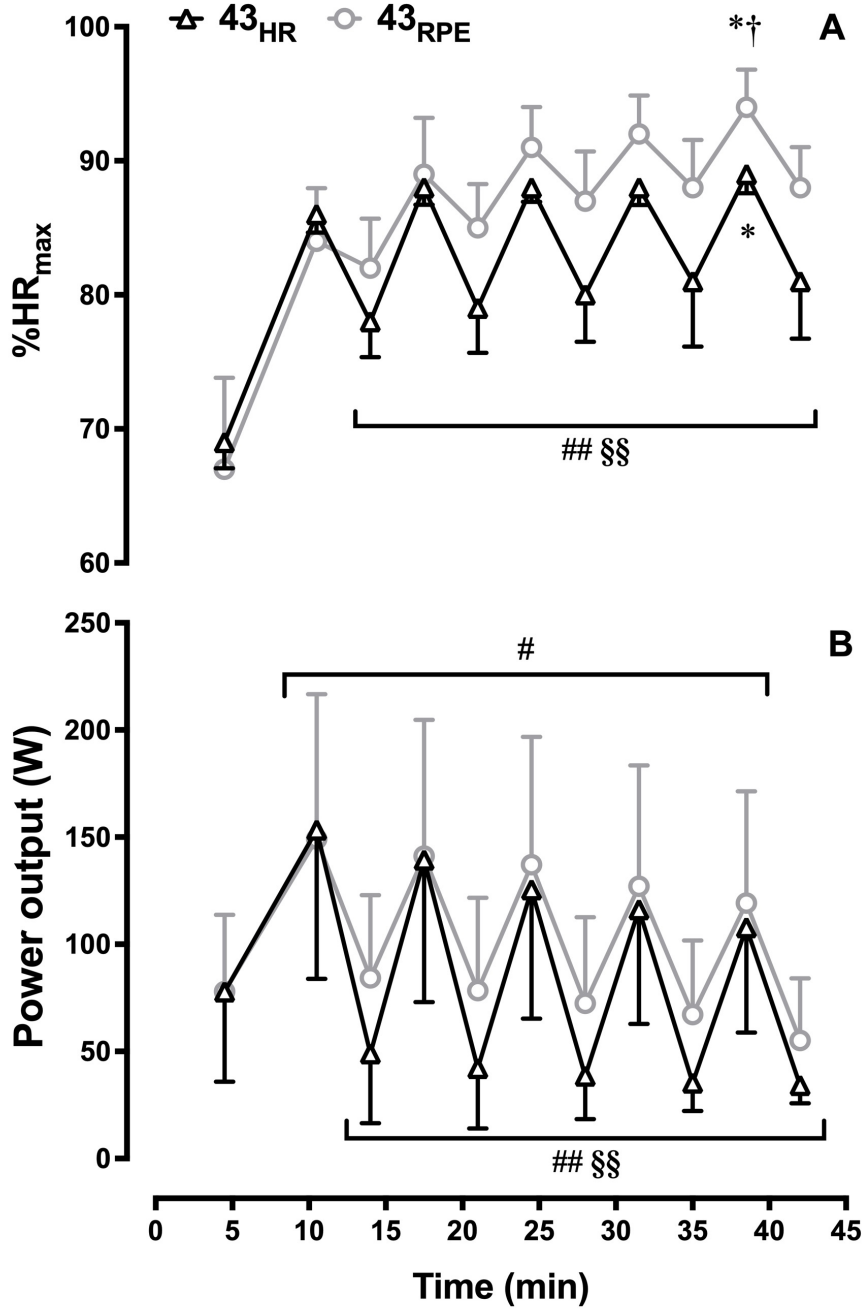


Figure 3.2. Percent of maximal heart rate (%HR_{max}; Panel A) and power output (Panel B) averaged over each interval during the 43-min trials. 43_{HR} = 43-min trial based on target heart rate; 43_{RPE} = 43-min trial based on target rating of perceived exertion. Data are summarized as mean ± SD. $^{\#}p < 0.05$ main effect of time during work intervals; $^{\#\#}p < 0.05$ main effect of time during recovery intervals; $^{\S\S}p < 0.05$ main effect of condition during recovery intervals; $^{*}p < 0.05$ compared with work interval 1 of the given condition; $^{\dagger}p < 0.05$ compared with HR-based trial during the same work interval.

Table 3.1. Responses during the first (1) and fifth (5) work and recovery intervals.

	43_{HR}		43_{RPE}	
Interval number	**1**	**5**	**1**	**5**
Work intervals				
Δ Power output (%)	—	–30 ± 10	—	–18 ± 18
Average HR	159 ± 6	165 ± 8*	156 ± 8	173 ± 4*†
Final min HR (b·min^{-1})	168 ± 6	167 ± 7	164 ± 6	176 ± 5*†
Blood lactate (mmol·L^{-1}) ‡	3.2 ± 1.0	2.5 ± 1.5	2.8 ± 1.2	3.2 ± 2.0
RPE	16 ± 2	16 ± 1	—	—
$M - W$ (W) ‡	622 ± 290	603 ± 287	620 ± 300	673 ± 312
$\dot{V}O_2$ (L·min^{-1})	2.3 ± 1.1	2.0 ± 0.8	2.2 ± 1.0	2.2 ± 0.9
Recovery intervals				
Δ Power output (%)	—	–18 ± 24	—	–33 ± 14
Average HR (b·min^{-1}) $^{\#\S}$	144 ± 10	150 ± 12	152 ± 8	163 ± 5
Final min HR (b·min^{-1}) $^{\#\S}$	133 ± 11	147 ± 9	144 ± 15	157 ± 6
RPE	11 ± 2	12 ± 2	—	—
$\overline{T}_{sk}$ (°C) $^{\#}$	36.3 ± 0.4	36.7 ± 0.6	36.4 ± 0.3	36.9 ± 0.5
T_{re} - $\overline{T}_{sk}$ (°C) $^{\#}$	1.0 ± 0.4	1.3 ± 0.4	0.9 ± 0.3	1.3 ± 0.3
$\overline{T}_b$ (°C)	37.1 ± 0.3	37.8 ± 0.4*	37.1 ± 0.3	37.9 ± 0.4*

ΔPower output = change in power output from the first to final work interval; Average HR = heart rate averaged across the entire interval; Final min HR = heart rate during the final minute of the interval; $\dot{V}O_2$ = oxygen uptake; RPE = rating of perceived exertion; T_{re} = rectal temperature; $\overline{T}_{sk}$ = mean skin temperature; $M - W$ = rate of metabolic heat production. $^{\ddagger}p < 0.05$ for interaction; $^{\#}p < 0.05$ for main effect of time; $^{\S}p < 0.05$ main effect of condition; $^{*}p < 0.05$ compared with interval 1 within the same condition; $^{\dagger}p < 0.05$ compared with HR-based trial during the same interval.

Thermoregulatory responses to HIIT exercise

Baseline T_{re} was not different among the 4 experimental trials (15_{HR} = 37.3 ± 0.3 °C, 15_{RPE} = 37.2 ± 0.3 °C, 43_{HR} = 37.1 ± 0.3 °C, 43_{RPE} = 37.1 ± 0.3 °C, $p = 0.19$). ($M - W$) increased over time in 43_{RPE} and decreased over time in 43_{HR} ($p = 0.04$ for interaction), but nonetheless T_{re} increased by a comparable amount between experimental conditions (0.7 °C for 43_{HR}, $p < 0.001$ and 0.8 °C for 43_{RPE}, $p < 0.001$; $p = 0.04$ for interaction) so that T_{re} at the end of the final

recovery interval was not different between conditions ($\alpha' = 0.0125$, $p = 0.03$; ES = 0.36; 95% CI for mean difference = –0.05, 0.46, Figure 3.3). T_{re} was also not different between conditions at the end of the first recovery interval ($p = 0.91$). Across both environmental conditions, skin temperature increased from the end of the first to final recovery interval by 0.4 °C ($p = 0.02$ for main effect of time; Table 3.1). The core-to-skin thermal gradient followed a similar pattern and increased by 0.3 °C in both trials ($p = 0.04$ for main effect of time). $\bar{T}_b$ increased by 0.6 °C in 43_{HR} ($p < 0.001$) and by 0.8 °C in 43_{RPE} ($p < 0.001$; $p = 0.03$ for interaction) from the end of the first to final recovery interval.

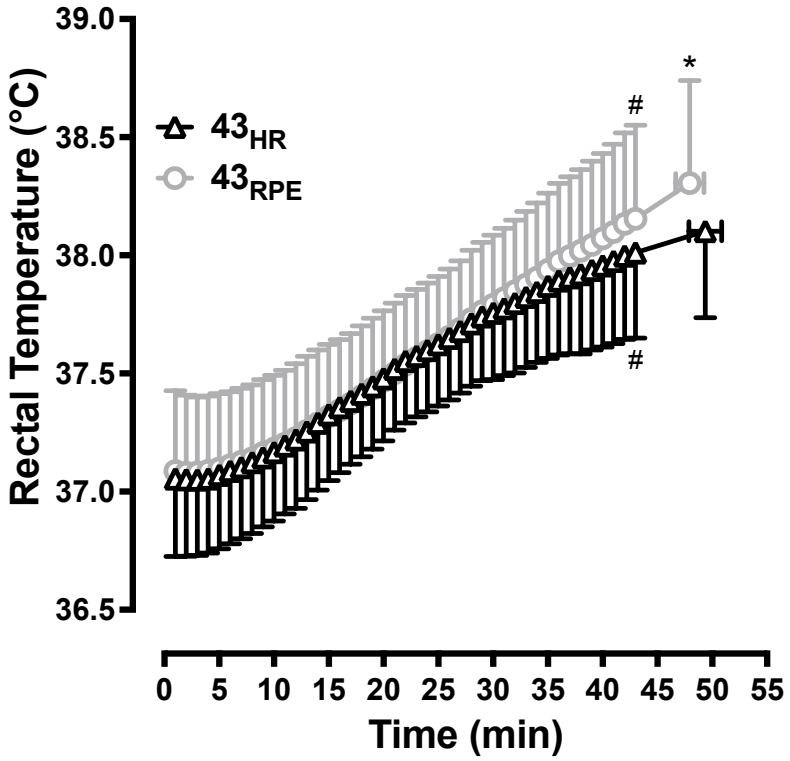


Figure 3.3. Rectal temperature (mean ± SD) from the start of exercise to the end of the graded exercise test. 43_{HR} = 43-min trial based on target heart rate; 43_{RPE} = 43-min trial based on target rating of perceived exertion. $^{\#}p < 0.05$ compared to min 15 of the same condition; $^{*}p < 0.05$ compared to 43_{HR} at max.

Maximal responses

Maximal responses are shown in Table 3.2 and Figure 3.3. Planned comparisons between control $\dot{V}O_{2max}$ (3.2 ± 1.2 L·min^{-1}) and $\dot{V}O_{2max}$ after each experimental trial did not reveal any differences [($\alpha' = 0.0125$) $p = 0.58$ for 15_{HR}; $p = 0.52$ for 15_{RPE}; $p = 0.014$ for 43_{HR}; $p = 0.014$ for 43_{RPE}]. $\dot{V}O_{2max}$ decreased 15.6% (ES = 0.41) between 15_{RPE} and 43_{RPE} ($p = 0.005$, 95% CI for mean difference = 0.08, 0.87; Figure 3.4), but it was not different over time during the HR-based trials [6.5%, ES = 0.16, ($\alpha' = 0.0125$) $p = 0.03$, 95% CI for mean difference = –0.05, 0.47). Furthermore, the change score (15-min value minus 43-min value) in $\dot{V}O_{2max}$ for RPE-based trials was greater than HR-based trials (mean difference = 0.3 ± 0.3 L·min^{-1}, 95% CI for mean difference = 0.01, 0.52, ES = 1.13, $p = 0.04$ for t-test comparing change scores). However, $\dot{V}O_{2max}$ was not different between conditions at 43 min ($p = 0.11$; ES = 0.18). During 43_{HR}, because $\dot{V}O_{2max}$ and absolute $\dot{V}O_2$ during HIIT exercise did not change over time, relative intensity ($\dot{V}O_2$ expressed as a percentage of $\dot{V}O_{2max}$ at the specified time point) was maintained (72% ± 5% $\dot{V}O_{2max}$ at work interval 1 and 69% ± 8% $\dot{V}O_{2max}$ at work interval 5, $p = 0.17$). However, since $\dot{V}O_{2max}$ decreased during 43_{RPE} and absolute $\dot{V}O_2$ did not change over time, the relative intensity increased by 11 percentage units (from 70% ± 10% $\dot{V}O_{2max}$ at work interval 1 to 81% ± 11% $\dot{V}O_{2max}$ at work interval 5, $p = 0.006$; $p = 0.002$ for interaction effect), and it was 11.5 percentage units higher on average during work interval 5 in 43_{RPE} vs. 43_{HR} ($p = 0.01$).

For both 43-min trials, the maximal power output achieved during the GXT was lower compared to the respective 15-min trial (43_{HR} = 22 W, p = 0.006; 43_{RPE} = 56 W, p = 0.002). Additionally, the maximal power output during the GXT was 31 W lower in the 43_{RPE} trial versus the 43_{HR} trial (p = 0.01). T_{re} and $\overline{T}_b$ were both 0.2 °C higher on average (T_{re}, p = 0.03, ES = 0.47; $\overline{T}_b$, p = 0.04, ES = 0.47) upon completion of the GXT following 43_{RPE} compared to 43_{HR}; however, $\overline{T}_{sk}$ (p = 0.24, ES = 0.47) and the core-to-skin thermal gradient (p = 0.96) were not different between conditions at maximum in the 43-min trials.

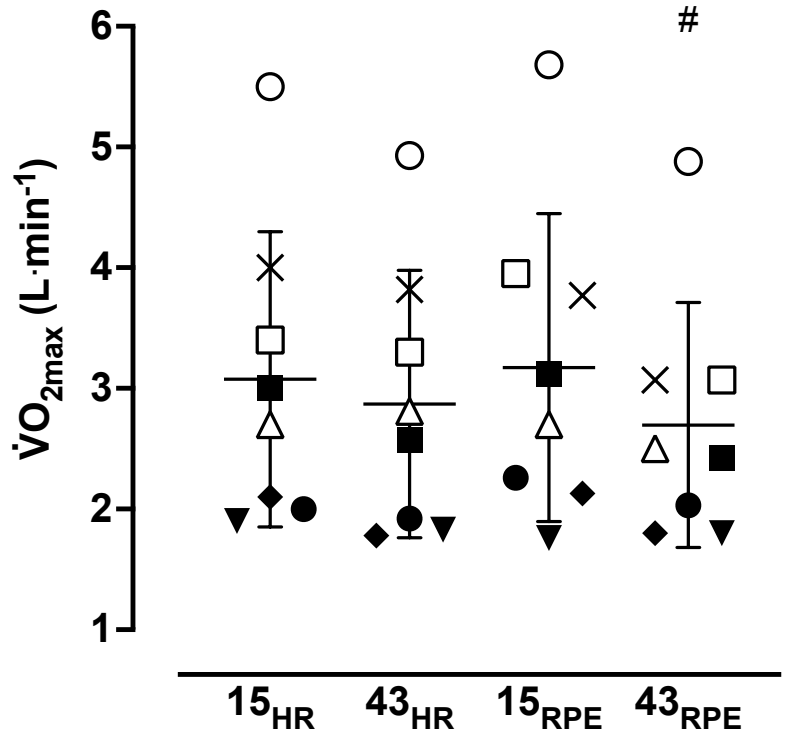


Figure 3.4. Vertical scattergram of maximal oxygen uptake ($\dot{V}O_{2max}$) during the experimental trials. Symbols represent data from individual participants and horizontal bars and accompanying error bars represent mean ± SD. $^{\#}p < 0.05$ compared to 15_{RPE}.

Table 3.2. Maximal responses during a graded exercise test following 15 and 43 min of high-intensity interval training in a hot environment using target heart rate (HR) or target rating of perceived exertion (RPE).

	Condition			
Variable	15_{HR}	43_{HR}	15_{RPE}	43_{RPE}
$\dot{V}_E$ (STPD, L·min^{-1}) [#]	84.9 ± 24.4	80.0 ± 21.6	88.6 ± 24.9	74.1 ± 20.1
$\dot{V}O_2$ (mL·kg^{-1}·min^{-1})	41.0 ± 13.7	38.0 ± 12.2	41.9 ± 13.7	35.9 ± 11.2[†]
RER	1.03 ± 0.04	1.02 ± 0.05	1.06 ± 0.06	0.95 ± 0.07[†]
RPE	20 ± 1	20 ± 1	20 ± 1	20 ± 0
HR (b·min^{-1})	187 ± 7	185 ± 5	187 ± 8	187 ± 6
Blood lactate (mmol·L^{-1}) [#]	4.9 ± 1.4	3.7 ± 1.1	5.4 ± 1.4	3.5 ± 1.5
$\overline{T}_{sk}$ (°C)	—	36.6 ± 0.6	—	36.9 ± 0.6
$\overline{T}_b$ (°C)	—	37.8 ± 0.4	—	38.0 ± 0.4[‡]
$T_{re} - \overline{T}_{sk}$ (°C)	—	1.5 ± 0.5	—	1.4 ± 0.5
Test duration (min) [#§]	7.8 ± 1.4	6.4 ± 1.5	8.8 ± 1.3	5.0 ± 1.3
Power output (W)	194 ± 73	172 ± 69[†]	197 ± 78	141 ± 72[†‡]

$\dot{V}_E$ = minute ventilation; $\dot{V}O_2$ = oxygen uptake; RER = respiratory exchange ratio; RPE = rating of perceived exertion; HR = heart rate; T_{re} = rectal temperature; $\overline{T}_{sk}$ = mean skin temperature; $\overline{T}_b$ = mean body temperature; $T_{re} - \overline{T}_{sk}$ = core-to-skin thermal gradient.
[#] $p < 0.05$ main effect of time; [§] $p < 0.05$ main effect of condition; [†] $p < 0.05$ compared with 15-min trial of same condition; [‡] $p < 0.05$ compared with 43_{HR}.

DISCUSSION

The purpose of this study was to evaluate work rate adjustments and thermal and cardiovascular strain using 2 simple methods of exercise prescription, THR and target RPE, to prescribe HIIT in the heat. A secondary purpose was to evaluate changes in aerobic capacity before (following 15 min and 1 round of HIIT) and after (following 5 rounds totaling 43 min of HIIT) cardiovascular drift is known to occur. The primary outcome was that work rate decreased from the first to the fifth work interval in both conditions, but by a non-significant, yet moderately larger amount during 43_{HR} (46 W) compared to 43_{RPE} (30 W). The moderately smaller reduction in work rate during 43_{RPE} did not result in differences in T_{re} over time between the two 43-min trials. However, as hypothesized, participants experienced increased

cardiovascular strain during the 43_{RPE} trial; HR was 9 b·min^{-1} higher during the final work interval and 13 b·min^{-1} higher during the first and final recovery intervals. Furthermore, a greater reduction in maximal aerobic capacity was observed following 43_{RPE} compared to 43_{HR}.

The range of decreases in work rate from the first to final work interval (43_{RPE} = 18% and 43_{HR} = 30%) were comparable to what others have observed during HIIT in a temperate environment using THR (21%) (Morales-Palomo et al. 2017), during 45 min of continuous exercise in the heat using THR (37%) (Wingo and Cureton 2006b), and during 30 min of continuous exercise in the heat using target RPE (≈27%) (Tucker et al. 2006). While work rate was not statistically different during the work intervals, the 16 W (ES = 0.53) greater reduction in work rate during 43_{HR} may be practically meaningful. However, unlike our results where work rate was lower during recovery intervals of 43_{HR}, no differences in running speed were observed during work or recovery intervals when using RPE compared to THR to prescribe exercise intensity during a 20-min treadmill walking/running HIIT session in a temperate environment (Ciolac et al. 2015). Still others have found lower intensities using RPE compared to THR during interval training (Aamot et al. 2014) and during continuous exercise (Shea et al. 2022) in cardiac rehabilitation patients in temperate environments. Taken together, it appears that findings related to work rate adjustments during HIIT based on THR and target RPE are equivocal. It is likely the heat stress in the current study contributed to the variability of findings when compared to the literature. Differences could also be attributed to variations in exercise protocol, mode and duration or participant characteristics.

We predicted work rate and thermal strain would be greater during 43_{RPE} versus 43_{HR}, but statistically higher work rates were only observed during the recovery intervals of 43_{RPE} and did not result in increased thermal strain. During the HIIT sessions, T_{re}, $\overline{T}_{sk}$, and the core-to-skin

thermal gradient were similar between conditions. The increase in ($M - W$) in 43_{RPE} apparently was not large enough to result in differences in T_{re} between conditions. Maxwell et al. (2008) observed a higher T_{re} during a sprint interval exercise session (twenty 5-s sprints interspersed with 110-s recovery) in the heat with higher versus lower recovery intensities. Differences in the ratio of work to recovery intervals, as well as the intensities used, could explain the differences between the results of the present study and those of Maxwell et al. (2008).

Although the elevated work rate during the recovery intervals of the RPE-based trial did not result in increased thermal strain, cardiovascular strain was greater during 43_{RPE} as indicated by an ≈ 5% higher HR averaged over the 5th work interval and in the final min of the 5th work interval. An increase in HR over time during interval training was observed in temperate environments when work rate was held constant (Thomas et al. 2020), when work rate was self-selected during intervals of 4 or 8 min (Fennell and Hopker 2021), and in temperate and hot environments when maximal sprint intervals were performed (Maxwell et al. 2008). Fennell and Hopker (2021) manipulated recovery intensity during a similar HIIT protocol (6 × 4 min with 2 min recovery in a temperate environment) where participants recovered at 80% or 110% of the power output corresponding to their lactate threshold. Unlike our results, the different recovery intensities did not affect HR during the work intervals, but during the recovery intervals HR was 7 b·min^{-1} higher during the 110% compared to 80% power output of their lactate threshold. Similarly, the difference in HR between conditions was unlike the findings of Ciolac et al. (2015) and Johnson et al. (2017) who observed similar HR when using THR and RPE to prescribe running intensity in temperate indoor and outdoor environments at varying exercise intensities. It appears heat stress may alter the relationship between HR and RPE that is observed in temperate environments.

The progressive increase in HR (and accompanying decrease in stroke volume) during continuous exercise in the heat has been shown to be associated with decreased maximal aerobic capacity (Wingo and Cureton 2006b; Lafrenz et al. 2008). As such, the ~2.5 times greater decrease in $\dot{V}O_{2max}$ in 43_{RPE} compared to 43_{HR} is consistent with our hypothesis. The 16% reduction in $\dot{V}O_{2max}$ between 15_{RPE} and 43_{RPE} is similar to reductions observed following 45 min of continuous exercise in the heat during cycling (13%) and running (15%) (Wingo et al. 2020). The magnitude of change following the RPE trial is also comparable to the decline observed during a repeated time trial performance (4 × 16.5 min with 5 min active recovery) in the heat; $\dot{V}O_{2max}$ was 97% of the control $\dot{V}O_{2max}$ at the end of the first time trial and decreased to 85% at the end of the final time trial (Périard and Racinais 2015). However, in this same study, during each time trial, participants maintained the same relative intensity (%$\dot{V}O_{2max}$; based on the $\dot{V}O_{2max}$ at that moment) despite the decreasing maximal aerobic capacity (Périard and Racinais 2015). In the present study, relative intensity was maintained during 43_{HR} (~71% $\dot{V}O_{2max}$), but during 43_{RPE} it increased by 11 percentage units to 81% $\dot{V}O_{2max}$ from the first to last work interval. The non-significant 6.5% (ES = 0.16) decline between 15_{HR} and 43_{HR} is comparable to the 7.5% reduction Wingo and Cureton (2006b) observed following 45 min of continuous exercise in the heat using THR to prescribe exercise intensity. These results indicate that when HR is allowed to drift upwards during the work intervals of HIIT exercise, cardiovascular drift accumulates and is accompanied by declines in maximal aerobic capacity.

Although work rate was not statistically different during the work intervals, the method of exercise prescription had a moderate to large effect on work rate during the work and recovery intervals, respectively, which explains why HR was elevated in the final work interval of 43_{RPE}. The higher intensities sustained in 43_{RPE} drove ($M - W$) upward. Since heat strain results in

tachycardia from increased sympathetic nervous system activity (Gorman and Proppe 1984) and catecholamine release (Kim et al. 1979), as well as direct effects of heat on sinoatrial node firing (Bolter and Atkinson 1988), the increase in ($M - W$) could explain exacerbated cardiovascular strain over the course of the 43_{RPE} HIIT session.

As mentioned, aerobic capacity decreased over twice as much following the RPE trial. We speculate the greater ($M - W$) in this trial resulted in a larger peripheral displacement of blood volume to the skin for heat dissipation. This peripheral displacement of blood volume, combined with the higher HR at the end of the final round of HIIT, could have corresponded to a lower SV (Coyle and Gonzalez-Alonso 2001; Turkevich et al. 1988; Rowell et al. 1966; Rowell et al. 1969; Wingo et al. 2012). If this lower SV persisted during maximal exercise, it could explain the decrease in $\dot{V}O_{2max}$.

Although both conditions resulted in large declines in work rate, participants were able to complete the entire HIIT protocol followed by a GXT in the heat. Exploring other methods or strategies for intensity prescription of HIIT in the heat may be beneficial for maintaining work interval intensity. Manipulating recovery intensity (such as using passive recovery instead of active recovery) may be one way to preserve work rate during the work intervals based on RPE.

Limitations

A limitation of using RPE to prescribe exercise intensity is the different interpretations of the scale. The RPE scale was explained to participants using standardized instructions and they were told to complete the work intervals at an RPE of 17, which meant adjusting the resistance up or down to elicit the prescribed RPE. Nonetheless, it appears 1 participant paced themselves and increased power output from the first to final work interval in the 43_{RPE} trial. This participant preserved their $\dot{V}O_{2max}$ compared to the 15-min trial while the remaining 7 participants

experienced an 18% decline (0.6 $L \cdot min^{-1}$) in $\dot{V}O_{2max}$ on average between the 15-min and 43-min trials. The participant who started off slower may have employed teleoanticipation whereby exercise intensity is regulated based on the anticipated endpoint of the exercise session (Ulmer 1996), although it is unclear why only 1 person may have adopted this strategy. RPE is easy to use for exercise prescription and it can be practical for prescribing HIIT (Buchheit and Laursen 2013), but the range of work rate adjustments during the work intervals of 43_{RPE} (–69 W to +19 W) presents a challenge to using RPE.

Another challenge with using RPE to prescribe intensity during exercise in the heat is the disassociation between the prescribed intensity and the HR response observed during recovery intervals. For instance, participants were instructed to cycle at an RPE of 12 during recovery, which is considered a moderate intensity corresponding to 64% – 74% HR_{max} (Garber et al. 2011). However, based on $\%HR_{max}$, participants exercised at a vigorous intensity (85% HR_{max}) during the final recovery interval (Garber et al. 2011). Even during the first interval of recovery, based on HR, participants were at a vigorous intensity (79% HR_{max}). Heart rate was elevated to such an extent that the $\%HR_{max}$ during the last 3 recovery intervals of 43_{RPE} were about the same as the work intervals of the 43_{HR} trial (Figure 3.2B). Furthermore, 2 participants achieved or surpassed the HR_{max} observed during the control trial during the submaximal HIIT portion of 43_{RPE}. Using THR in the heat also proved problematic for prescribing intensity during recovery intervals because most participants were unable to achieve the THR and instead cycled at 30 W.

Besides the aforementioned limitations, participants could have also become heat acclimated over the course of the study. However, this was unlikely because the trials took place on non-consecutive days and 2 of the trials were short in duration (< 25 min). Even though short term heat acclimation can take place in ≤ 7 days of consecutive heat stress (Pryor et al. 2018),

full heat acclimation typically requires 8-14 days of consecutive heat stress. Indeed, exercise performance in the heat remained impaired following 5 consecutive days of HIIT in the heat (Reeve et al. 2019). So, the amount of heat stress needed to have induced heat acclimation far exceeds the heat stress the participants in this study experienced.

It is also possible that participants improved their fitness throughout the study; however, more training sessions over a longer period would likely have been required for participants to measurably increase fitness. For example, in moderately trained men, HIIT performed 2–4 times per week over 4 weeks did not lead to a significant improvement in $\dot{V}O_{2max}$ (Keith et al. 1992), which surpasses the amount of training participants in the present study completed. Lastly, any systematic effects a change in acclimatization, acclimation, or training status may have had should have been avoided since experimental trials were completed in a counterbalanced order.

HIIT can be prescribed many different ways by altering the duration, intensity, and mode of exercise. This study implemented submaximal, longer duration work intervals (4 min) on a cycle ergometer; therefore, the results are not likely applicable to all types of HIIT where shorter durations and/or higher intensities are used with different modes of exercise. Additionally, all of the participants were young and healthy individuals free of disease and it is unclear if similar magnitudes of work rate adjustments and cardiovascular strain would have been observed in different populations. Despite the limitations of using RPE and THR to prescribe intensity in the present study, both resulted in the exercise intensity being attainable across the varying fitness levels of the participants.

CONCLUSION

Using target RPE and THR to prescribe HIIT in the heat resulted in considerable declines in work rate during the work intervals, and lower work rates were needed to maintain THR

compared to target RPE during the recovery intervals. The higher power outputs sustained in 43_{RPE} recovery intervals corresponded to elevated cardiovascular strain during both work and recovery intervals, as well as a greater decline in $\dot{V}O_{2max}$ over time. The non-significant, but moderately smaller reduction in work rate from the first to fifth work interval of 43_{RPE} may have also contributed to the increase cardiovascular strain observed. Although with both methods of exercise prescription reductions in work rate were necessary to maintain the target intensity, all participants (regardless of varying fitness levels; $\dot{V}O_{2max}$ range: 28.1 $mL \cdot kg^{-1} \cdot min^{-1}$ to 68.2 $mL \cdot kg^{-1} \cdot min^{-1}$) were able to complete the exercise protocol, which as our pilot testing indicated would not have been possible if work rate adjustments had not been made. If total energy expenditure is the goal of the exercise session and magnitude of cardiovascular strain is not important, using RPE to prescribe intensity during HIIT may be preferable. Using THR may be preferable if cardiovascular strain is a concern; however, this may limit the training stimulus since larger declines in work rate are necessary.

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CHAPTER 4

WORK RATE ADJUSTMENTS DURING HIGH-INTENSITY INTERVAL TRAINING IN THE HEAT WITH ACTIVE AND PASSIVE RECOVERY

INTRODUCTION

High-intensity interval training (HIIT) is a popular (Thompson 2021) and effective form of exercise for improving health and fitness (Esfarjani and Laursen 2007; Tjønna et al. 2008; Helgerud et al. 2007). Typical HIIT workouts are comprised of work intervals performed at high intensities and recovery intervals performed either actively (exercise continues at a lower intensity) or passively (exercise ceases). Recommendations regarding active and passive recovery are generally based on the duration and intensity of the work intervals and duration of the rest intervals (Schoenmakers et al. 2019). For instance, for HIIT with work intervals ≥ 1 min, passive recovery is recommended when recovery intervals are < 2–3 min and active recovery is recommended when recovery intervals are > 3–4 min (Buchheit and Laursen 2013).

The studies upon which these recommendations were based occurred in temperate environments (Buchheit and Laursen 2013), so findings may not be applicable to hot environments. Exercise limitations associated with hot ambient temperatures could impact the prescription of recovery intervals and subsequent work intervals. For example, during a HIIT workout involving 4-s sprints in hot conditions, active recovery resulted in no difference in power output during work intervals—but higher core body temperature—compared to passive recovery, likely because of a higher rate of metabolic heat production sustained in the active protocol (Bishop et al. 2007). It is unclear whether similar outcomes would result from HIIT

with longer work bouts (≥ 4 min at intensities ~90% maximal heart rate) with active versus passive recovery in the heat. Greater thermal strain would be expected with active recovery because the collective rate of metabolic heat production from the work and recovery intervals would be higher compared to the same workout with passive recovery. However, the extent to which the method of recovery differentially affects thermal strain during HIIT with longer intervals in a hot environment remains unknown.

Besides greater thermal strain, greater cardiovascular strain [manifested as cardiovascular drift, a progressive increase in heart rate (HR) and decrease in stroke volume (SV) despite no change in work rate] would also be expected during HIIT with longer work intervals and active recovery in the heat compared to HIIT with longer work intervals and passive recovery. Elevated thermal strain and accompanying cardiovascular drift are associated with momentary declines in maximal oxygen uptake ($\dot{V}O_{2max}$) during continuous exercise (Wingo et al. 2005; Nybo et al. 2001; Cheuvront et al. 2010; Wingo et al. 2012), which amplifies the perception of exercise intensity. As such, after the first or second cycle of work and active recovery intervals, power output would be expected to decrease during subsequent work and recovery intervals in order to maintain the target intensity, which could compromise the training stimulus. Passive recovery may lessen the severity of thermal and cardiovascular strain, and accompanying declines in $\dot{V}O_{2max}$, which would enable better maintenance of target intensities during work intervals, but this has not been tested.

Accordingly, the purpose of this study was to test the hypotheses that 1) active recovery will increase thermal and cardiovascular strain compared to passive recovery during HIIT in the heat, 2) work rate must be lowered to a greater extent to maintain target intensity during HIIT utilizing active recovery compared to passive recovery in a hot environment and 3) $\dot{V}O_{2max}$ will

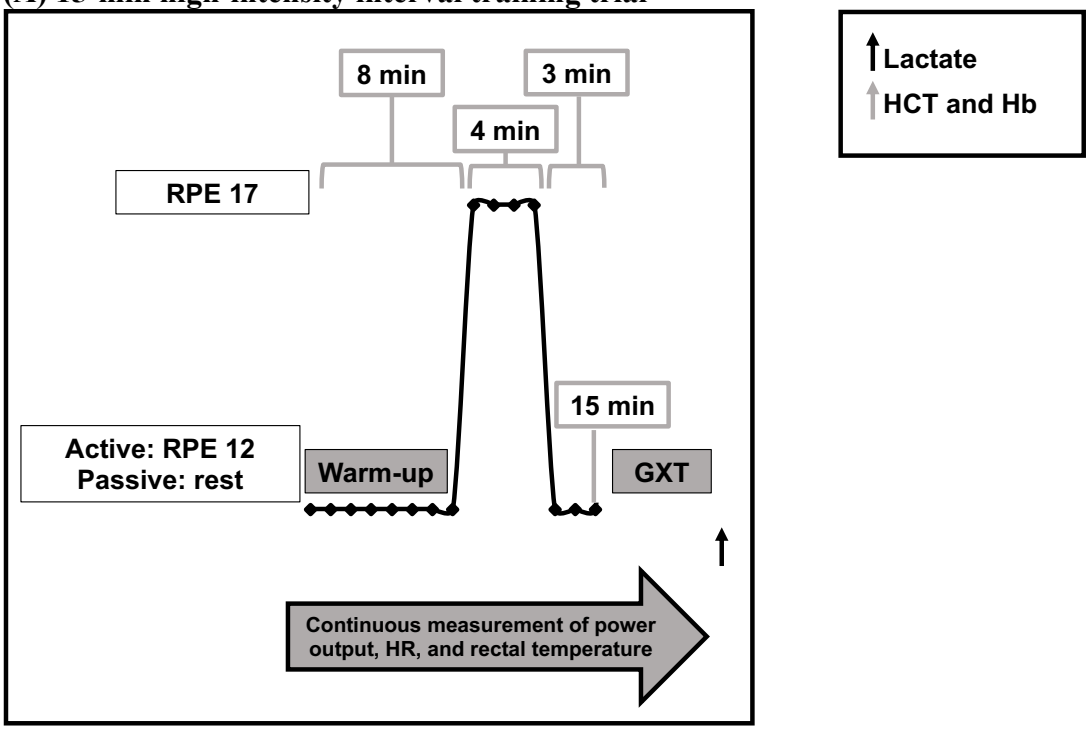
decrease to a greater extent after HIIT in the heat with active recovery compared to passive recovery.

METHODS

Experimental design

A repeated measures crossover design was used in which participants completed 5 trials (1 control trial, two 15-min experimental trials, and two 43-min experimental trials) on a cycle ergometer (LC6 Novo, Monark Exercise, Vansbro, Sweden). The control trial consisted of a graded exercise test (GXT) to determine maximal HR (HR_{max}) and $\dot{V}O_{2max}$. For the 4 remaining experimental trials, participants were randomly assigned to a counterbalanced treatment order. These trials began with an 8-min warm-up at an RPE of 12 followed by 1 or 5 rounds of HIIT (4 min of work at an RPE of 17 and 3 min of either active recovery at an RPE of 12 or passive recovery involving sitting on the cycle ergometer) as follows: 15_{ACT} = warm-up and 1 round of HIIT with active recovery, 15_{PASS} = warm-up and 1 round of HIIT with passive recovery, 43_{ACT} = warm-up and 5 rounds of HIIT with active recovery, 43_{PASS} = warm-up and 5 rounds of HIIT with passive recovery. Participants who were unable to achieve a minimum RPE of 12 during the active recovery intervals due to increased thermal and cardiovascular strain cycled at 30 W with a cadence $\geq$ 30 rev·min^{-1} to ensure active recovery was measurably different compared to passive recovery. At the end of each experimental trial, participants immediately began a GXT to measure $\dot{V}O_{2max}$. The purpose of the separate 15- and 43-min trials was to evaluate $\dot{V}O_{2max}$ before (15-min trials) and after (43-min trials) work rate adjustments resulting from thermal and cardiovascular strain had been made. Figure 4.1 depicts a general overview of the experimental trials.

(A) 15-min high-intensity interval training trial



(B) 43-min high-intensity interval training trial

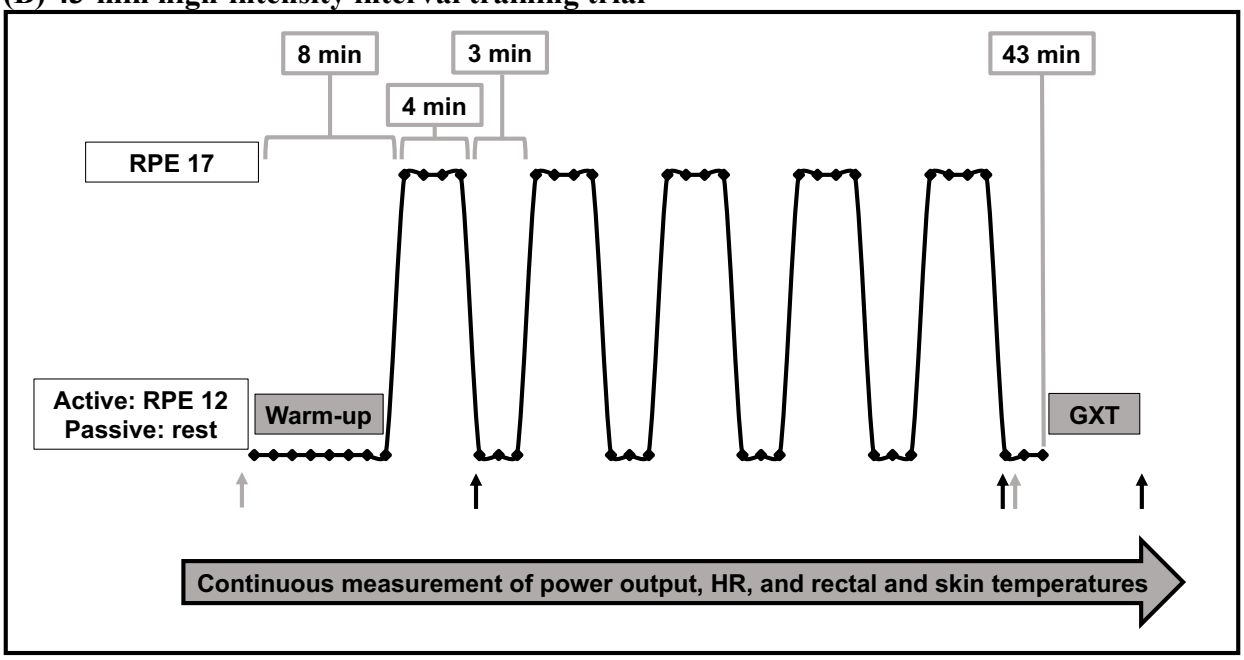


Figure 4.1. General exercise protocols for the (A) 15- and (B) 43-min experimental trials. All trials included an 8-min warm-up at an RPE of 12. GXT, graded exercise test; $\dot{V}O_2$, oxygen uptake; HR, heart rate; RPE, rating of perceived exertion; Hb, hemoglobin; HCT, hematocrit.

Participants

An a priori power analysis (G*power v.3.1.9.6) revealed a sample size of 7 would be sufficient to detect a 25-W (effect size = 1.4) difference between the change score in power output from the first to the final work interval in 43_{PASS} versus 43_{ACT}, assuming $\alpha = 0.05$ and power ≈ 0.80 (Faul et al. 2009; Faul et al. 2007). Ten healthy adults consented to participated and 8 completed all study procedures. One participant withdrew from the study after completing 1 visit and another after 2 visits. Participants included 2 women and 6 men; both women and 4 of the men were recreationally active (participating in ≥ 30 min of aerobic exercise per day, ≥ 3 times per week for the past ≥ 3 months) as defined by the American College of Sports Medicine (2021). The other 2 men were competitive endurance athletes. Physical activity levels and health history were confirmed through questionnaires; all participants reported engaging in physical activity outdoors. Physical characteristics of participants were (mean ± SD) age = 24 ± 7 y, body mass = 75.4 ± 7.5 kg, height = 181 ± 10 cm, percent body fat = 17.9% ± 7.1%, HR_{max} = 187 ± 10 $b \cdot min^{-1}$.

Female participants had a menstrual cycle lasting 21–35 days (Elliott-Sale et al. 2021). Women were asked to self-report the first and last day of previous menses so that all experimental trials could be performed within the same phase of their menstrual cycle, although the specific phase was not expected to affect study outcomes (Stone et al. 2021). Both women completed all experimental trials in the luteal phase.

Procedures

All trials

For each of the 5 trials, participants were instructed to report to the laboratory after a 2-h fast, well rested, euhydrated, and having refrained from ingesting non-prescription drugs and

caffeine on the day of testing. Additionally, they were instructed to refrain from consuming alcohol or participating in strenuous exercise during the 24 h prior to testing. Adherence to pre-test instructions was confirmed using a 24-h history questionnaire. Upon arrival, participants provided a urine sample that was analyzed for urine specific gravity (U_{SG}) using a digital refractometer (ATAGO PAL-10S digital refractometer, Tokyo, Japan). U_{SG} had to be ≤ 1.020 for a participant to be considered adequately hydrated (Sawka et al. 2007). Participants whose U_{SG} values were > 1.020 were given fluids to ingest for 20–30 min and reevaluated. Then participants donned padded cycling shorts, a mesh tank top, and a chest strap HR monitor (H10, Polar Electro, Kempele, Finland). Prior to beginning exercise, the Borg 6–20 RPE scale was explained using standardized instructions (Borg and Noble 1974). Approximately 3–5 min after the end of the each GXT a 2-mL blood sample was drawn from a superficial forearm vein into a Vacutainer tube containing EDTA (BD Vacutainer, Becton, Dickinson and Co., Franklin Lakes, NJ, USA) for the measurement of blood lactate (YSI 2300 STAT Plus, Yellow Spring Instruments, OH, USA). All trials were completed at a similar time of day (± 2 h) to control for fluctuations in core body temperature associated with circadian rhythm (Moore-Ede et al. 1983). A minimum of 24 h separated control trials from subsequent experimental trials and at least 48 h separated experimental trials from one another. All trials for a given participant took place over ≤ 8 weeks.

Control $\dot{V}O_{2max}$ trial

At the first visit, participants completed a questionnaire about their readiness to participate in exercise, a general health history form, and provided written informed consent. Next, height and body mass were measured using a stadiometer (SECA 213, Seca Ltd., Hamburg, Germany) and digital scale (Tanita WB-800S plus, Tanita Corp., Tokyo Japan),

respectively. Body fat percentage was calculated from the sum of 3 skinfolds (Jackson and Pollock 1985).

Next, participants entered an environmental chamber maintained at 22.6 ± 0.6 °C, 35% ± 7% relative humidity (RH). Participants mounted a cycle ergometer and then warmed up for 5–10 min at a self-selected moderate-intensity in a temperate environment. Then they began the GXT at a moderate intensity based on RPE and HR observed during the warm-up. Every 2 min the power output on the cycle ergometer was increased 25 W until volitional exhaustion or pedal cadence fell below 30 rev·min^{-1}. HR was measured continuously using a smartphone application (Polar Beat, version 3.5.0, Polar Electro, Kempele, Finland) integrated with the chest strap. $\dot{V}O_2$ was measured continuously using open circuit spirometry (Parvo Medics Metabolic Measurement System, model TrueOne 2400, Salt Lake City, UT, USA).

Twenty min after the test, participants remounted the cycle ergometer and completed a $\dot{V}O_{2max}$ plateau verification protocol. Those who completed < 1 min of the final stage of the initial GXT performed the verification protocol at the final power output achieved during the initial GXT; those who completed ≥ 1 min of the final stage of the initial GXT performed the verification protocol at a power output 25 W higher than that achieved during the initial GXT (Wingo et al. 2005). To remain in the study all participants had to exhibit a $\dot{V}O_{2max}$ value ≥ 20th percentile for cycle ergometer-based testing for their sex and age (American College of Sports Medicine 2021).

Experimental Trials

All experimental trials

Experimental trials were performed in an environmental chamber maintained at 35.1 ± 0.3 °C, 40.2% ± 3.3% RH. In addition to the aforementioned initial procedures, for the

experimental trials, participants measured nude body mass and inserted a flexible rectal thermistor (MEAS 401, Measurement Specialties, Andover, MN, USA) 10 cm beyond the anal sphincter. The thermistor was integrated with wireless amplifiers (BioNomadix Wireless SKT Transmitter, Biopac Systems, Inc., Goleta, CA, USA) set to a sampling frequency of 1000 Hz and used to measure and record rectal temperature (T_{re}). T_{re} and ambient temperature were recorded continuously using a data acquisition system (MP150, Biopac Systems, Inc., Goleta, CA, USA) powered by data analysis software (AcqKnowledge 5.0, Biopac Systems, Inc., Goleta, CA, USA). Blood samples were drawn from a superficial forearm vein at the time points shown in Figure 4.1. During exercise, participants were responsible for adjusting the intensity to match the given RPE; power output and HR were not visible on the ergometer display, but the RPE scale was continuously visible to participants during exercise.

Participants began the HIIT session with an 8-min warm-up at an RPE of 12. For the 15-min trials, following the warm-up, participants completed 1 round of HIIT (4 min at an RPE = 17 and 3 min at RPE = 12). For the 43-min trials, participants completed 5 rounds of HIIT. During all experimental trials a member of the research team provided frequent reminders to adjust the work rate as needed to stay at the prescribed RPE. All experimental trials were followed by a GXT to determine $\dot{V}O_{2max}$ immediately after the final recovery bout; power output was increased to approximately half of the maximal power output observed during the control trial, with 25-W increases every 2 min thereafter until volitional exhaustion.

15_{ACT} and 15_{PASS}

Participants entered an environmental chamber and mounted the cycle ergometer. Next, instrumentation was connected and baseline measurements were taken (~15 min). After

collecting baseline measurements, participants completed 1 of the 15-min trials that consisted of a warm-up and 1 round of HIIT immediately followed by a GXT to measure $\dot{V}O_{2max}$.

43_{ACT} and 43_{PASS}

For the 43_{ACT} and 43_{PASS} trials, a flexible catheter was placed into a forearm vein for 2-mL blood sample collection before, during exercise at time points corresponding to the end of the first and final high intensity bouts (min 12 and 40), and after the GXT as shown in Figure 4.1. Blood lactate concentrations were measured at each time point and hematocrit and hemoglobin concentration were measured prior to exercise and at min 40. Hematocrit microcapillary tubes were centrifuged (Autocrit Ultra 3 Microhematocrit Centrifuge, model 420575, Becton, Dickinson and Co., Franklin Lakes, NJ, USA) and then assessed in triplicate using a microcapillary reader (Model 3201, International Equipment Co., Boston, MA, USA) and hemoglobin was measured in duplicate with a hemoglobin analyzer (HemoPoint H2, EKF Diagnostics, Inc., Boerne, TX, USA); together, hematocrit and hemoglobin were used to calculate plasma volume change (Dill and Costill 1974).

After the indwelling catheter placement, four iButtons (model no. DS1921H, Embedded Data Systems, KY, USA) were secured using therapeutic elastic tape to each participant's right side on their upper chest, lateral deltoid, anterior thigh, and lateral calf to measure skin temperature. Mean skin temperature ($\overline{T}_{sk}$) was calculated using the following equation (Ramanathan 1964):

$$\overline{T}_{sk} = 0.3(T_{chest} + T_{delt}) + 0.2(T_{thigh} + T_{calf}),$$

where T_{chest}, T_{delt}, T_{thigh}, and T_{calf} are the skin temperatures at the chest, deltoid, thigh, and calf, respectively. Mean body temperature ($\overline{T}_b$) was calculated using a weighted average of T_{re} and $\overline{T}_{sk}$ using the following equation (Stolwijk and Hardy 1966):

$$\overline{T}_b = 0.8(T_{re}) + 0.2(\overline{T}_{sk}).$$

The core-to-skin thermal gradient was calculated as the difference between rectal temperature and mean skin temperature (T_{re} - $\overline{T}_{sk}$). $\dot{V}O_2$ was measured during the first and final work interval and during the GXT. Metabolic rate was calculated for the first and final work intervals using the following equation (Kenny and Jay 2013):

$$M = (\dot{V}O_2[(((RER - 0.7)0.3^{-1})e_c) + (((1 - RER)0.3^{-1})e_f)]60^{-1},$$

where $\dot{V}O_2$ is the rate of oxygen uptake in $L \cdot min^{-1}$, e_c = 21,130 J (caloric equivalent per liter of oxygen for carbohydrate oxidation), e_f = 19,630 J (caloric equivalent per liter of oxygen for fat oxidation), and RER is respiratory exchange ratio. The difference between *M* and the external work rate on the cycle ergometer was calculated as the rate of metabolic heat production (*M – W)* and expressed in W (Kenny and Jay 2013).

Following the placement of the iButtons, participants entered an environmental chamber and mounted the cycle ergometer. Final instrumentation was connected, a 2-mL baseline blood sample was obtained, and other baseline measurements were taken (~15 min), then participants completed one of the 43-min trials (warm-up, 5 rounds of HIIT, and GXT). At min 12 and 40, participants also indicated their thermal sensation using a numerical scale (Young et al. 1987). Approximately 20 min following exercise, participants were asked to rate the exercise session using session RPE (Foster et al. 2001).

Data analysis

All statistical analyses were performed using SPSS for Mac v.28.0.0.0 (IBM Corporation, Somers, NY). Mean data were generated on the indicated outcome measures. To test the significance of mean differences in power output, a 2-way [condition × time (work intervals 1 and 5)] repeated measures analysis of variance (ANOVA) was used. Power output was also

assessed by comparing the change in power output from the first to final work interval between 43_{ACT} and 43_{PASS} using a paired samples t-test was used to compare. Paired samples t-tests were also used to evaluate differences in maximal values for T_{re}, $\overline{T}_{sk}$, $T_{re} - \overline{T}_{sk}$, and $\overline{T}_{b}$ between conditions.

Baseline data for control and experimental trials were analyzed using a 1-way repeated measures analysis of variance (ANOVA). Planned contrasts were performed to compare $\dot{V}O_{2max}$ of the experimental trials to the control trial with a Bonferroni correction to control for family-wise α. To evaluate if $\dot{V}O_{2max}$ changed differently between experimental trials over time, a 2-way repeated measures ANOVA [condition × time (from after 15 min to after 43 min)] was conducted.

For hematological variables, 2-way (condition × time) repeated measures ANOVAs were conducted. For other variables, such as HR, T_{re}, $\overline{T}_{sk}$, power output, and $\dot{V}O_2$, 2-way repeated measures ANOVAs [condition × time (work intervals 1 and 5) and/or condition × time (recovery intervals 1 and 5)] were conducted. If the assumption of sphericity was violated for relevant repeated measures ANOVAs, then the Greenhouse-Geisser correction was used.

In the event of a significant omnibus test, post hoc pairwise comparisons with Bonferroni α correction (α′) were performed accordingly. Effect sizes (ES) for paired samples t-tests were calculated using the following formula (Lakens 2013) for Cohen's d_{av} (Cohen 1988) adjusted for positive bias using Hedges's correction (g_{av}):

$$\text{ES} = \frac{Mean\ difference}{\frac{SD_1 + SD_2}{2}} \times \left(1 - \frac{3}{4(n \times 2) - 9}\right),$$

where, SD_1 and SD_2 are the standard deviations of the respective time points or conditions and n is the number of pairs. ES were interpreted as: 0.20 = small, 0.5 = medium, and 0.80 = large (Caldwell and Cheuvront 2019; Fritz et al. 2012).

For select variables, a 95% confidence interval (CI) was calculated for the mean difference using a critical t (adjusted for multiple comparisons, if applicable) with the following formula (Weir and Vincent 2020):

$$CI = \text{Mean difference} \pm t_{cv}(SE_d),$$

where t_{cv} is the critical t value and SE_d is the standard error of the differences.

For power output, $\dot{V}O_2$, and $(M - W)$, the average over the entire interval was used for data analysis; for T_{re} and $\bar{T}_{sk}$, the average of the final min of the interval was used for data analysis; for HR, both the average over the entire interval and the average of the final min were analyzed. Data are presented as mean ± SD, and all statistical tests used an α level of 0.05.

RESULTS

Hydration

Participants began trials adequately hydrated based on U_{SG} (control = 1.005 ± 0.002, 15_{ACT} = 1.008 ± 0.005, 15_{PASS} = 1.004 ± 0.004, 43_{ACT} = 1.005 ± 0.002, 43_{PASS} = 1.008 ± 0.006, p = 0.82). Additionally, pre-exercise body mass was comparable among trials (control = 75.4 ± 7.5 kg, 15_{ACT} = 75.9 ± 8.1 kg, 15_{PASS} = 75.6 ± 7.8 kg, 43_{ACT} = 75.4 ± 7.7 kg, 43_{PASS} = 75.5 ± 7.8 kg, p = 0.61). Percent change in body mass from before to after exercise was larger for the 43-min trials compared to the 15-min trials, but time points across conditions were not different (15_{ACT} = –0.7% ± 0.4%, 15_{PASS} = –0.7% ± 0.3%, 43_{ACT} = –1.5% ± 0.8%, 43_{PASS} = –1.4% ± 0.7%; $p < 0.001$ for main effect of time). Similarly, percent change in plasma volume was not different between trials pre- to post-exercise (43_{ACT} = –11.7% ± 4.1%, 43_{PASS} = –12.0% ± 3.9%; p = 0.71, ES = 0.07).

Thermoregulatory responses to HIIT exercise

Baseline T_{re} (15_{ACT} = 37.3 ± 0.3 °C, 43_{ACT} = 37.2 ± 0.4 °C, 15_{PASS} = 37.3 ± 0.3 °C, 43_{PASS} = 37.2 ± 0.3 °C) was not different among the 4 experimental trials (p = 0.39). $\overline{T}_{sk}$ increased over the same time for the 43-min trials, but the type of recovery was not influential (p = 0.16 for main effect of condition; Figure 4.2A). T_{re} increased by 0.9 °C (p < 0.001) from the end of the first (15 min) to final (43 min) recovery interval with active recovery and by 0.7 °C with passive recovery (p < 0.001; Figure 4.2A). Since T_{re} increased proportionally more than $\overline{T}_{sk}$, the gradient was elevated 0.4 °C at the end of the last recovery interval compared to the first (p = 0.009 for main effect of time; Table 4.1). $\overline{T}_{b}$ was 0.7 °C higher during the final recovery interval compared to the first (p < 0.001 for main effect of time).

Cardiovascular, power output, and perceptual responses during HIIT exercise

Heart rate responses

HR during the final minute of the work intervals was similar between conditions but increased from the first to final interval by 14 b·min^{-1} (p = 0.001; Table 4.1). HR and $\%HR_{max}$ averaged over each work interval followed a similar pattern (Figure 4.3A).

As hypothesized, during 43_{PASS} HR was 35 b·min^{-1} lower during the final min of the first recovery interval (p < 0.001) and 27 b·min^{-1} lower during the final recovery interval (p < 0.002) compared to 43_{ACT} (Table 4.1). During recovery, final minute HR of the intervals increased over time during 43_{PASS} by 18 b·min^{-1} (p < 0.001), but it did not increase by a statistically significant amount during 43_{ACT} (10 b·min^{-1}, α' = 0.0125, p = 0.02). Average HR during recovery intervals was 14 b·min^{-1} higher in the fifth versus first interval across both conditions (p = 0.003 for main effect of time) and 24 b·min^{-1} higher with active compared to passive recovery during the recovery intervals (p = 0.002 for main effect of condition).

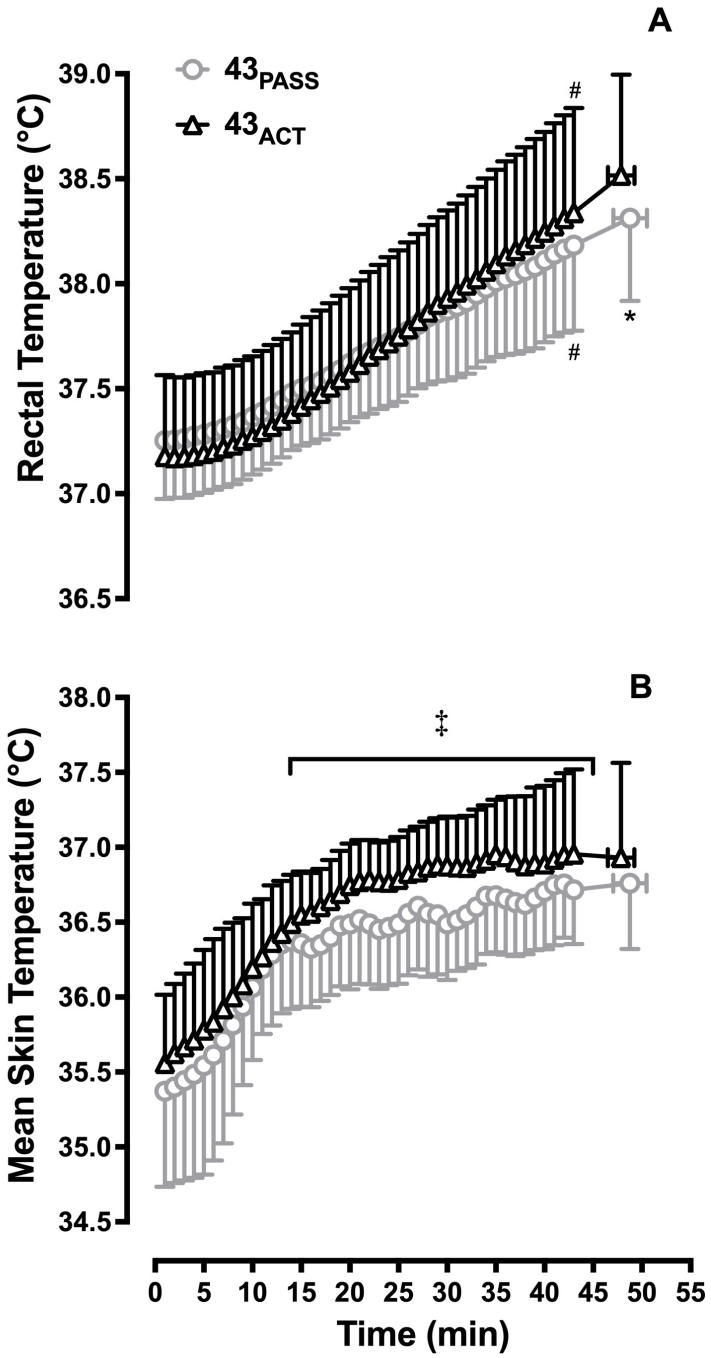


Figure 4.2. Rectal temperature (panel A) and mean skin temperature (panel B) during the 43-min trials to the end of the graded exercise test. 43_{ACT} = 43-min trial with active recovery; 43_{PASS} = 43-min trial with passive recovery. Data are summarized as mean ± SD.‡ $p < 0.05$ for main effect of time from 15 to 43 min. $^{\#}$ $p < 0.05$ compared to min 15 of the respective trial; * $p < 0.05$ compared to 43_{ACT} at end of graded exercise test.

Table 4.1. Responses during the first (1) and fifth (5) work and recovery intervals.

	43_{ACT}		**43_{PASS}**	
Interval number	**1**	**5**	**1**	**5**
Work interval				
Δ Power output (W)		–28 ± 38	—	–6 ± 19
Average HR ($b \cdot min^{-1}$) $^{\#}$	155 ± 7	173 ± 7	156 ± 15	167 ± 11
Final min HR ($b \cdot min^{-1}$) $^{\#}$	164 ± 7	178 ± 6	162 ± 15	176 ±9
Final min $\%HR_{max}$	87 ± 6	95 ± 3	87 ± 8	94 ± 4
$\dot{V}O_2$ ($L \cdot min^{-1}$)	2.4 ± 0.9	2.5 ± 0.9	2.5 ± 1.0	2.5 ± 1.0
Blood lactate ($mmol \cdot L^{-1}$) $^{\#}$	3.2 ± 1.5	3.9 ± 2.1	3.0 ± 1.1	4.2 ± 1.7
$M - W$ (W)‡	711 ± 264	762 ± 281	706 ± 276	703 ± 302
Recovery interval	**1**	**5**	**1**	**5**
Power output (W)	90 ± 40	66 ± 35*	—	—
Average HR ($b \cdot min^{-1}$) $^{\#\S}$	152 ± 10	163 ± 8	125 ± 20	141 ± 14
Final min HR ($b \cdot min^{-1}$)	147 ± 11	157 ± 10	112 ± 21†	130 ± 16*†
Final min $\%HR_{max}$	78 ± 5	84 ± 3	60 ± 11†	70 ± 8*†
$T_{re} - \overline{T}_{sk}$ (°C) $^{\#}$	0.9 ± 0.5	1.4 ± 0.6	1.1 ± 0.5	1.5 ± 0.5

43_{ACT} = 43-min trial with active recovery; 43_{PASS} = 43-min trial with passive recovery; Δ Power output = change in power output from the first to fifth interval; HR = heart rate; $\dot{V}O_2$ = oxygen uptake; $M - W$ = rate of metabolic heat production; $T_{re} - \overline{T}_{sk}$ = core-to-skin thermal gradient. ‡ $p < 0.05$ for interaction; $^{\#}$ $p < 0.05$ for main effect of time; § $p < 0.05$ for main effect of condition; * $p < 0.05$ compared with interval 1 within the same condition; † $p < 0.05$ compared with active during the same interval.

Power output

The decrease in power output from the first to final work interval was not statistically different between conditions (Table 4.1; $p = 0.07$ for the t test comparing change scores; $p = 0.07$ for interaction from 2-way ANOVA), but the percent change in work rate was larger during 43_{ACT} (–14% ± 24%) versus 43_{PASS} (–1% ± 16%; $p = 0.04$; 95% CI for mean difference = –24, –1; ES = 0.61). Furthermore, despite the lack of statistical significance, the magnitude of difference between change scores was medium–large (22 W; 95% CI for mean difference in change scores = –45, 2; ES = 0.73). The 2-way ANOVA revealed that power output was not statistically different between the first and final work interval across conditions ($p = 0.11$ for

main effect of time) or between conditions across time ($p = 0.31$ for main effect of condition). However, the magnitude of difference in power output between the first and final work interval was medium (ES = 0.42) for 43_{ACT} but small (ES = 0.08) for 43_{PASS}. For the active recovery intervals, work rate decreased by 27% (24 W) from the first to final interval ($p = 0.02$).

During 43_{ACT}, 2 participants increased work rate over the course of the exercise session, while the other 6 decreased the work rate to maintain the 17 target RPE. Additionally, the same 2 participants and 1 additional participant increased power output from the first to final work interval during 43_{PASS}.

Perceptual responses

In both 43-min trials, thermal sensation increased from 6.0 ± 0.5 at the completion of the first work interval to 7.0 ± 0.5 at the final work interval ($p = 0.005$). Session RPE was similar between trials (43_{ACT} = 9 ± 1, 43_{PASS} = 8 ± 1; $p = 0.35$).

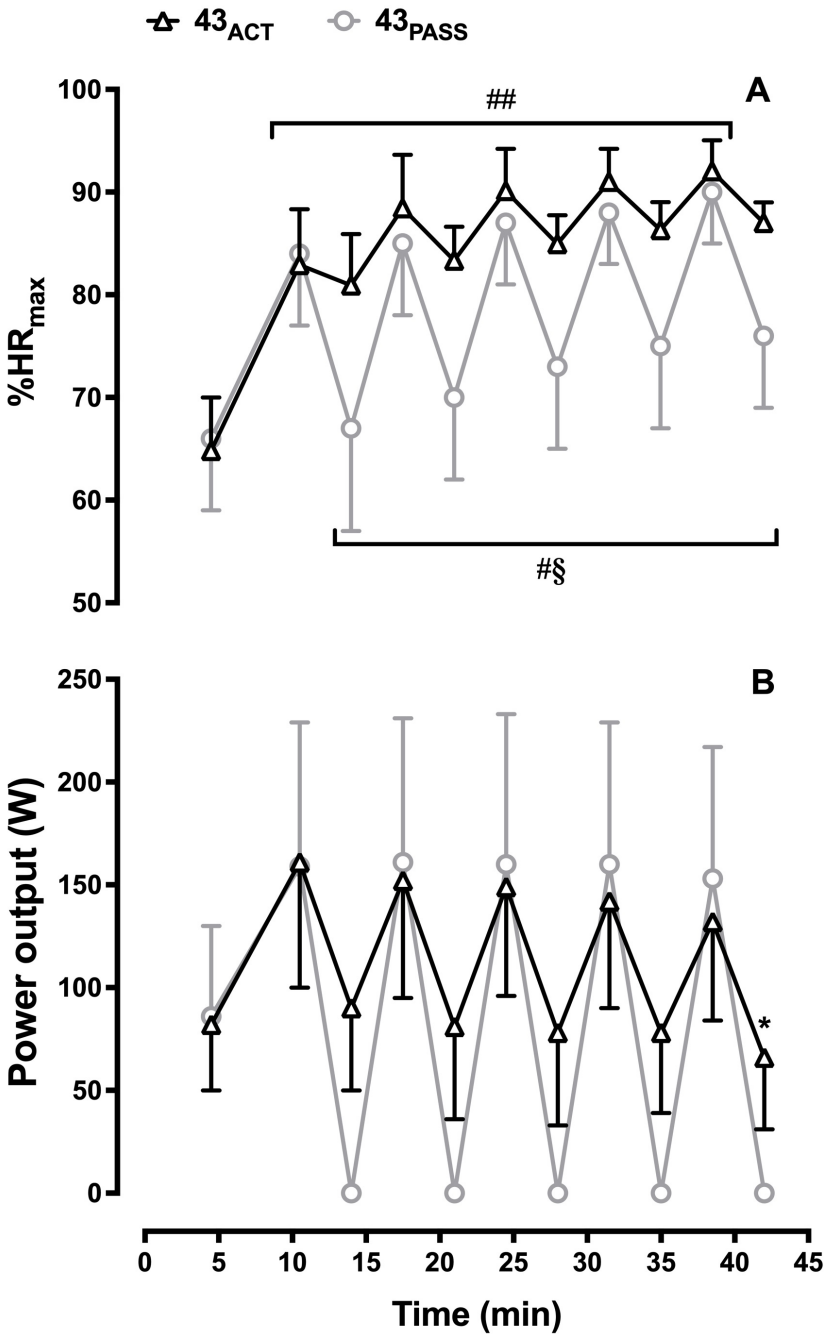


Figure 4.3. Mean ± SD percent maximal HR (%HR_{max}; panel A) and power output (panel B) of each interval during the 43-min trials. Peaks represent work intervals and nadirs represent recovery intervals. Time points (x coordinates) represent the middle of the interval and values (y coordinates) were averaged over the entire interval. Data are summarized as mean ± SD. 43_{ACT} = 43-min trial with active recovery; 43_{PASS} = 43-min trial with active recovery. $^{\#\#}p < 0.05$ for main effect of time for work interval; $^{\#}p < 0.05$ for main effect of time for recovery interval; $^{\S}p < 0.05$ for main effect of condition for recovery intervals; $^{*}p < 0.05$ compared to first interval of same condition.

Maximal responses

Maximal responses are shown in Table 4.2 and Figure 4.4. Planned comparisons of control $\dot{V}O_{2max}$ ($\dot{V}O_{2max}$ = 3.5 ± 1.2 L·min^{-1}) with $\dot{V}O_{2max}$ after each experimental trial revealed only 43_{ACT} was different ($\alpha' = 0.0125$; $p = 0.99$ for 15_{ACT}; $p = 0.77$ for 15_{PASS}; $p = 0.02$ for 43_{PASS}; $p = 0.007$ for 43_{ACT}). $\dot{V}O_{2max}$ was 0.4 L·min^{-1} (11.5%; ES = 0.34) lower following the 43-min trials compared to 15-min trials ($p = 0.008$), but recovery mode did not have an effect ($p = 0.38$ for interaction; $p = 0.69$ for main effect of condition). The change score (15-min value minus 43-min value) in $\dot{V}O_{2max}$ was no different between conditions = 0.1 ± 0.5 L·min^{-1}, 95% CI for mean difference = –0.4, 0.5, ES = 0.17, $p = 0.74$ for t-test comparing change scores).

Maximal power output achieved during the 43-min trials was 12.7% lower than that during the 15-min trials ($p = 0.04$). Lastly, Figure 4.2 shows that $\overline{T}_{sk}$ was similar at maximum, but T_{re} was 0.2 °C higher ($p = 0.047$) in 43_{ACT} compared to 43_{PASS}.

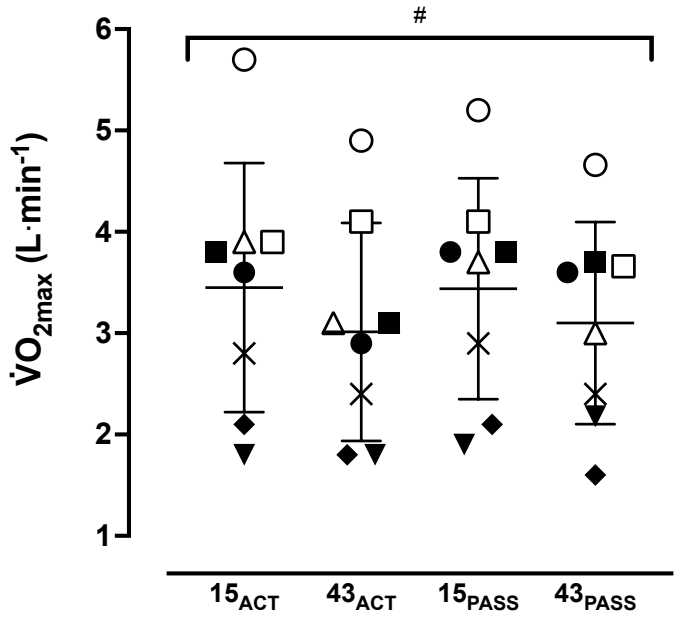


Figure 4.4. Vertical scattergram of maximal oxygen uptake ($\dot{V}O_{2max}$) measured during the experimental trials. Symbols represent data from individual participants and horizontal bars and accompanying error bars represent mean ± SD. $^{\#}p < 0.05$ for main effect of time.

Table 4.2. Maximal responses during a graded exercise test following 15 and 43 min of high-intensity interval training in a hot environment with active or passive recovery.

	Condition			
Variable	15_{ACT}	43_{ACT}	15_{PASS}	43_{PASS}
$\dot{V}_E$ (STPD, $L \cdot min^{-1}$) #	94.0 ± 23.5	81.5 ± 21.0	93.8 ± 20.9	86.0 ± 25.7
$\dot{V}O_2$ ($mL \cdot kg^{-1} \cdot min^{-1}$) #	45.0 ± 14.0	39.5 ± 13.1	45.1 ± 12.7	41.0 ± 12.0
RER #	1.06 ± 0.07	0.97 ± 0.06	1.03 ± 0.09	0.98 ± 0.08
RPE †	19 ± 1	20 ± 1	19 ± 1	19 ± 1
HR ($b \cdot min^{-1}$) †	188 ± 9	188 ± 10	187 ± 9	186 ± 7
Blood lactate ($mmol \cdot L^{-1}$) #†	5.4 ± 1.3	4.3 ± 1.5	7.2 ± 1.8	4.6 ± 1.8
$\overline{T}_b$ (°C)	—	38.2 ± 0.4	—	38.0 ± 0.3‡
$T_{re} - \overline{T}_{sk}$ (°C)	—	1.7 ± 0.8	—	1.6 ± 0.5
Test duration (min) #	8.2 ± 2.0	4.9 ± 1.4	8.1 ± 1.6	5.8 ± 1.7
Power output (W) #	209 ± 82	178 ± 56	209 ± 74	188 ± 73

$\dot{V}_E$ = minute ventilation; $\dot{V}O_2$ = oxygen uptake; RER = respiratory exchange ratio; RPE = rating of perceived exertion; HR = heart rate; $\overline{T}_b$ = mean body temperature; T_{re} = rectal temperature; $\overline{T}_{sk}$ = mean skin temperature. # $p < 0.05$ for main effect of time; † $p < 0.05$ for main effect of condition; ‡ $p < 0.05$ compared to 43_{ACT} at the same time.

DISCUSSION

The primary purpose of this investigation was to evaluate if active recovery during HIIT exercise in the heat resulted in greater thermal and cardiovascular strain compared to passive recovery, and if so, whether this necessitated greater reductions in work rate to maintain target RPE during the work intervals. A secondary purpose was to determine if $\dot{V}O_{2max}$ decreased to a greater extent following HIIT with active recovery than HIIT with passive recovery. The main finding was that thermal strain was comparable between experimental conditions, and higher HR during recovery intervals was accompanied by a larger percent change in work rate during 43_{ACT} compared to 43_{PASS}. $\dot{V}O_{2max}$ decreased between 15 min (after the warm-up and first round of HIIT) and 43 min (after the final round of HIIT), but the decrease was not affected by recovery mode.

Despite no exercise during passive recovery intervals, T_{re} nonetheless increased by the same extent over time (i.e., between the first and final recovery interval) in both experimental conditions, although T_{re} was 0.3 °C higher during 43_{ACT} upon completion of the GXT. This finding is in contrast to others who have observed higher core temperatures during active versus passive recovery in the heat with sprint interval training (4-6-s sprints with ~ 2 min of recovery repeated 16-20 times) (Bishop et al. 2007; Maxwell et al. 2008). The difference in results could be explained by differences in the prescriptions of the HIIT protocols, such as the intensity, duration, and number of repetitions. In our study, apparently HIIT exercise duration contributed more to the magnitude of thermal strain than the type of recovery.

Because no work was completed during passive recovery intervals, a lower ($M - W$) was expected, which would in turn result in lower T_{re}. Why then did T_{re} increase the same during the passive recovery condition as during the active recovery condition? The significant interaction for ($M - W$) suggests it increased more over time in 43_{ACT} vs. 43_{PASS}, but post hoc comparisons revealed no individual differences, and the ES comparing ($M - W$) at the fifth work interval was small (0.19). This suggests the difference in ($M - W$) between conditions was insufficient to result in differences in heat balance. Furthermore, even if the ~59 W lower ($M - W$) during the fifth work interval of the passive recovery trial was considered meaningful, it may have been offset by lower heat dissipation. For instance, during seated recovery after exercise, skin blood flow and sweating have been shown to return to baseline levels despite core temperature remaining elevated above baseline values (Journeay et al. 2006; Kenny et al. 2007). Furthermore, Jay et al. (2008) evaluated heat balance during 30 min of active (cycling against no resistance) or passive recovery following 15 min of cycling at 75% $\dot{V}O_{2max}$ in a temperate environment; core temperature was similar between recovery modes despite higher ($M - W$) with active recovery.

The authors concluded that core temperature was similar between the conditions because lower rates of dry and evaporative heat loss and sweating occurred shortly after (2–10 min) the cessation of exercise. In the current study, perhaps reductions in dry and evaporative heat loss during the passive recovery intervals—combined with only a modestly smaller ($M - W$) during 43_{PASS} work intervals—partially explains the similar core temperatures between conditions.

Not surprisingly, active recovery resulted in greater overall cardiovascular strain during recovery intervals compared to passive recovery as evidenced by a 24 $b \cdot min^{-1}$ (ES = 1.97) higher HR on average. During HIIT protocols in temperate and hot environments, others have also observed elevated HR with active recovery compared to passive either during the entire exercise session or during the recovery intervals depending on the protocol (Bishop et al. 2007; Ahmaidi et al. 1996; Fennell and Hopker 2021; Stanley and Buchheit 2014). Peak HR during 15 s of cycling at 120% $\dot{V}O_{2max}$ with 15-s active or passive recovery until exhaustion in a temperate environment was not different between recovery modes; however, time to exhaustion completing the interval workout was 125% greater with passive recovery (Dupont et al. 2004). In the current study, HR was not different between conditions during work intervals because participants lowered their work rate over time during 43_{ACT} to maintain the target RPE. Had work rate adjustments not been made, some participants probably would have reached exhaustion (or at least maximal HR/cardiovascular capacity) prior to the final interval in 43_{ACT}.

A recent systematic review on passive versus active recovery highlighted the mixed results regarding mechanical performance (i.e., mean power, peak power, or maximal velocity depending on exercise mode and study design) during interval exercise with some studies finding no differences, others finding better performance with active recovery, and still others finding better performance with passive recovery (Perrier-Melo et al. 2021). Differences in the HIIT

paradigm (duration and intensities of work and recovery intervals) and environmental conditions may explain the discrepant results in work rate adjustments. For example, Bishop et al. (2007) found no differences between conditions in mean peak power output during work intervals involving 4-s sprints in a hot environment (active = 1257 ± 64 W, passive = 1245 ± 47 W). In an investigation that implemented a HIIT protocol similar to ours (self-paced cycling in a temperate environment using 6 × 4 min work intervals with 2-min recovery intervals), investigators found work rates were higher during the work intervals with passive recovery (Fennell and Hopker 2021). However, unlike in our study where the perceived intensity was held constant, participants were instructed to cycle at the maximal power output that could be sustained for the interval and RPE drifted upwards to near maximal levels from the first to sixth work interval. Others have observed this upward drift in RPE during continuous exercise in the heat as well (Galloway and Maughan 1997; Wingo and Cureton 2006). Because RPE drifts upward during continuous and interval exercise—especially that in the heat—to maintain a given RPE, we hypothesized work rate would need to be lowered. Despite the lack of statistical significance for change in power output, we found work rate was lowered by 22 W more in 43_{ACT} than in 43_{PASS} and the percent change in work rate was significantly greater in 43_{ACT}. Similar to our results, to maintain an RPE of 16 during 30 min of constant cycling in the heat, Tucker et al. (2006) observed work rate had to be lowered by ≈ 27%. The target RPE of 17 in the present study was achieved at a lower work rate during the latter stages of HIIT involving active recovery intervals for 6 of 8 participants.

Even though work rate had to be lowered over time by most participants in the 43_{ACT} trial, HR still increased over time and maximal aerobic capacity declined in both 43-min trials. The magnitude of decrease in $\dot{V}O_{2max}$ following both 43-min trials (11.5%; ES = 0.34) is

consistent with other studies from our laboratory that assessed $\dot{V}O_{2max}$ immediately following 45-min of continuous exercise in the heat, falling between a protocol using unmitigated cardiovascular strain (–18.2%) (Wingo et al. 2004) and one using target HR (and accompanying decrements in work rate) to mitigate cardiovascular strain (–7.5%) (Wingo and Cureton 2006). The magnitude of change is also comparable to the decline observed during a repeated time trial performance (4 × 16.5 min with 5 min active recovery) in the heat; $\dot{V}O_{2max}$ was 97% of the control $\dot{V}O_{2max}$ at the end of the first time trial and decreased to 85% at the end of the final time trial (Périard and Racinais 2015). Six min of active or passive recovery following a swimming HIIT protocol (eight 25-m sprints) did not affect the time to complete a 50-m sprint although the 50-min sprint time was not compared to an earlier or control time point and it is not clear if the HIIT workout impaired maximal performance (Toubekis et al. 2005). The present results demonstrate maximal aerobic performance is impaired over time following a HIIT exercise session using target RPE in the heat with active or passive recovery.

The lack of interaction effect for $\dot{V}O_{2max}$ between min 15 (after the warm-up and first round of HIIT) and min 43 (after the final round of HIIT) for active and passive experimental trials is contrary to our hypothesis. Elevated core temperatures can impair aerobic performance (Ely et al. 2007; Galloway and Maughan 1997; Lafrenz et al. 2008), and since core temperatures were similar between conditions, this may partially explain why the reduction in $\dot{V}O_{2max}$ was not different between 43_{ACT} and 43_{PASS}. T_{re} increased more proportionately than $\overline{T}_{sk}$ over time, but the effect on the core-to-skin thermal gradient later in exercise was small and comparable between conditions (Table 4.1). Elevated body temperature likely resulted in peripheral displacement of blood volume to the skin (Rowell et al. 1966; Rowell et al. 1969) and if this

persisted during maximal exercise, maximal SV would have been diminished, which would have lowered $\dot{V}O_{2max}$.

A limitation of this study is that participants could have improved their fitness throughout the duration of the study; however, given the training status of participants, more training sessions over a longer period would likely have been required for participants to measurably increase their fitness. For example, in moderately trained men, HIIT performed 2–4 times per week over 4 weeks did not lead to a significant improvement in $\dot{V}O_{2max}$ (Keith et al. 1992), suggesting the 4 HIIT sessions spread over 3–8 weeks in the present study was not a sufficient training stimulus to increase $\dot{V}O_{2max}$. Similarly, participants could have become heat acclimated during the 4 trials in the heat. The magnitude of heat stress needed for full heat acclimation generally requires 8–14 consecutive days (Pryor et al. 2018), far exceeding the heat stress the participants in this study experienced. Furthermore, the counterbalanced treatment orders should have prevented any systematic effects a change in training, acclimatization, or acclimation status could have had.

RPE was used to gauge intensity because it is easy to use for exercise prescription and can be practical for prescribing HIIT (Buchheit and Laursen 2013). However, another limitation of the study was that using RPE to assess physiological consequences of HIIT meant that participants were free to continuously self-select work rate. As a result, 2 participants likely paced themselves (i.e., intentionally selected lower work rates at the start and higher work rates at the end) instead of focusing exclusively on target RPE in a given work interval. Johnson et al. (2017) found that using target RPE compared to target HR at different running intensities in untrained men resulted in a larger variation in the intensity for the RPE training condition, which agrees with the varying responses to the prescribed RPE we observed. The 2 participants who

probably paced themselves increased work rate by 25 W between the first and final interval while the others declined by 46 W. The increase in work rate over time also resulted in a larger increase in HR over time (26 $b \cdot min^{-1}$ versus 10 $b \cdot min^{-1}$). Compared to the 2 participants suspected of pacing who essentially maintained $\dot{V}O_{2max}$ over time, the remaining 6 participants averaged a 17% decline in $\dot{V}O_{2max}$ after the final round of HIIT (43 min) compared to after the first round of HIIT (15 min) in the active recovery condition. If they had selected work rate based on the target RPE, work rate would have been expected to remain the same throughout the HIIT bout or perhaps decrease over time in conjunction with exacerbated physiological strain. The participants who started off at lower power outputs were likely implementing teleoanticipation whereby exercise intensity is regulated based on the anticipated endpoint of the exercise session (Ulmer 1996). The preservation of $\dot{V}O_{2max}$ in the participants who paced themselves during the 43_{ACT} trial could partially explain why negative pacing strategies (an increase in speed over time) (Abbiss and Laursen 2008) can improve performance during cycling time trials (Mattern et al. 2001), although caution is warranted in generalizing from our small sample.

Besides pacing, this study demonstrates another challenge of using RPE to prescribe intensity during HIIT in the heat given the disassociation between the prescribed intensity and the HR response during recovery intervals. Participants were instructed to cycle at an RPE of 12 during active recovery, a moderate intensity that should have corresponded to ~64% – 74% HR_{max} (Garber et al. 2011). However, based on HR, participants actually exercised at a vigorous intensity (77% – 95% HR_{max}) (Garber et al. 2011) during active recovery intervals. Even during the first active recovery interval, before cardiovascular drift would have been expected to occur, participants cycled at a vigorous intensity (78% HR_{max}). Additionally, during the work intervals,

3 participants achieved or exceeded the HR reached at maximum during the control GXT, even though work intervals were intended to be performed at a submaximal intensity. Despite the increased cardiovascular strain observed during the work intervals, all participants were able to complete the exercise protocol, but larger declines in work rate during active recovery were necessary to maintain target RPE. A possible way to overcome challenges like pacing and reaching maximal HR is to use multiple indicators of intensity, such as RPE combined with HR, power output, or both, although this would reduce the simplicity of exercise prescription based on RPE alone.

As the goal of HIIT is typically to spend more time at higher intensities, during HIIT in the heat using a protocol like that in the present study, passive recovery may be beneficial for the high intensity training stimulus. However, active recovery likely resulted in greater total energy expenditure, and despite increased cardiovascular strain, did not exacerbate thermal strain compared to passive recovery. A lower intensity of active recovery may balance lower cardiovascular strain during the recovery intervals with maintenance of a greater overall net energy expenditure (and metabolic training stimulus) from the workout as a whole. Since this study only evaluated acute responses, it is unclear how implementing active or passive recovery exclusively would alter chronic training adaptations.

CONCLUSIONS

This study shows that thermal strain was comparable between HIIT sessions using active and passive recovery. As expected, greater cardiovascular strain was evident during active recovery intervals. Furthermore, active recovery during HIIT in the heat necessitated 21 W lower power output, on average, by the final work interval in order to achieve the same target RPE as

the passive recovery condition. Five rounds of HIIT in a hot environment, regardless of recovery mode, resulted in small–moderate reductions in $\dot{V}O_{2max}$ over time.

Despite the high intensities (even during active recovery), all participants were able to complete the full HIIT session in a hot environment. Without work rate adjustments during the work intervals in the 43_{ACT} trial this may not have been possible. These results support the notion that in hot conditions, HIIT protocols utilizing passive recovery allow maintenance of higher work rates during the work intervals and lower cardiovascular strain during recovery intervals. However, lack of any work during passive recovery intervals may offset these benefits and result in no better overall training stimulus than protocols utilizing active recovery. Furthermore, if maximizing total energy expenditure is the goal of the session and cardiovascular strain is not of importance, then active recovery may be more beneficial.

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CHAPTER 5

CONCLUSION

High-intensity interval training (HIIT) is a popular mode of exercise that is performed in varying environmental conditions, but little is known about how hot conditions interact with different methods of exercise prescription to affect work rate and cardiovascular and thermal strain while performing a HIIT workout. This dissertation used target heart rate (THR) and rating of perceived exertion (RPE)—2 common and practical methods of exercise prescription—which allows the results to be translated into practical considerations for completing HIIT workouts (8-min warm-up, 5 rounds of 4-min work × 3-min recovery) in the heat.

The primary objective of the first study was to evaluate the magnitude of thermal and cardiovascular strain and work rate adjustments during work intervals when THR is used to prescribe exercise intensity in a temperate compared to hot environment. The main finding in support of the first hypothesis was that to maintain THR, power output had to be reduced over time by 18% in a temperate environment and 33% in a hot environment. In agreement with our second hypothesis, core temperature increased more in the hot environment, and even though THR was used during work intervals, increased cardiovascular strain was observed during the recovery intervals in the heat. Lastly, in contrast to the third hypothesis, $\dot{V}O_{2max}$ decreased marginally following HIIT in both conditions. The large work rate adjustments likely mitigated thermal and cardiovascular strain but were insufficient to completely preserve $\dot{V}O_{2max}$.

Because of the large decrements in work rate needed to maintain THR during HIIT in the heat, the second study investigated whether RPE would result in maintenance of higher work

rates compared to THR. The main finding was that work rate decreased over time during the work intervals in both conditions, but by 16 W more during the HR-based trial which partially agrees with the first hypothesis. Additionally, work rate was higher during the recovery intervals of the RPE-based trial compared to the HR-based trial. The second hypothesis was partially supported as the difference in work rates during recovery did not result in increased thermal strain but using RPE to gauge exercise intensity resulted in greater cardiovascular strain. In support of the third hypothesis, an ≈ 2.5 times larger reduction in $\dot{V}O_{2max}$ was observed following RPE-based HIIT.

The primary purpose of the third investigation was to evaluate if active recovery during HIIT exercise in the heat resulted in greater thermal and cardiovascular strain compared to passive recovery, and if so, whether this necessitated greater reductions in work rate to maintain target RPE during the work intervals. The main finding was that thermal strain was comparable between experimental conditions and increased cardiovascular strain in the active recovery intervals was accompanied by a larger percent decrease in work rate during work intervals which partially supports the first and second hypotheses. Although the work rate adjustments and cardiovascular strain were different between recovery modes, unlike predicted, $\dot{V}O_{2max}$ declined similarly.

The findings of this dissertation showed that work rate adjustments were necessary to maintain target intensity because of cardiovascular drift (higher HR for a given work rate) during HIIT, especially in the heat. If work rate had not been adjusted, HR likely would have reached maximum, ending the exercise sessions prematurely. But work rate was adjusted, which permitted maintenance of the prescribed intensities and mitigated the drift in heart rate over time, allowing participants of varying fitness levels to complete all workouts. Depending on the goal

of the exercise session, completing a similar HIIT session in a cooler environment if it is an option, or using RPE and passive recovery if a cooler environment is not an option, may be the best choice for maintaining higher work rates during the work intervals. When prescribing HIIT and using THR or RPE in a temperate or hot environment, consideration should be given to the magnitude of expected work rate adjustments and the accompanying decreases in the training stimulus.

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APPENDIX A

STUDIES 1 AND 2 INSTITUTIONAL REVIEW BOARD CERTIFICATION

THE UNIVERSITY OF ALABAMA | Research & Economic Development
Office for Research Compliance

May 12, 2021

Hillary Yoder
Department of Kinesiology
College of Education
The University of Alabama
Box 870312

Re: IRB Protocol # 21-02-4259 "Heart rate-based and RPE-based exercise intensity during HIIT in the heat"

Ms. Yoder:

The University of Alabama Medical Institutional Review Board has granted approval for your proposed research. Your application has been given full board approval according to 45 CFR part 46.

The approval for your application will lapse on May 5, 2022. If your research will continue beyond this date, please submit a continuing review to the IRB as required by University policy before the lapse. Please note, any modifications made in research design, methodology, or procedures must be submitted to and approved by the IRB before implementation. Please submit a final report form when the study is complete.

Please use reproductions of the IRB approved informed consent form to obtain consent from your participants.

Good luck with your research.

Jessup Building | Box 870127| Tuscaloosa, AL 35487-0127 | 205-348-8461
Fax 205-348-7189| Toll Free 1-877-820-3066 | rscompliance@research.ua.edu

APPENDIX B

STUDY 3 INSTITUTIONAL REVIEW BOARD CERTIFICATION

THE UNIVERSITY OF ALABAMA | Office of the Vice President for Research & Economic Development
Office for Research Compliance

July 19, 2021

Hillary Yoder
Department of Kinesiology
College of Education
The University of Alabama
Box 870312

Re: IRB Protocol # 21-03-4419 "Work Rate Adjustments During High-Intensity Interval Training in the Heat with Active and Passive Recovery"

Ms. Yoder:

The University of Alabama Medical Institutional Review Board has granted approval for your proposed research. Your application has been given full board approval according to 45 CFR part 46.

The approval for your application will lapse on June 2, 2022. If your research will continue beyond this date, please submit a continuing review to the IRB as required by University policy before the lapse. Please note, any modifications made in research design, methodology, or procedures must be submitted to and approved by the IRB before implementation. Please submit a final report form when the study is complete.

Please use reproductions of the IRB approved informed consent form to obtain consent from your participants.

Good luck with your research.

Jessup Building | Box 870127 | Tuscaloosa, AL 35487-0127
205-348-8461 | Fax 205-348-7189 | Toll Free 1-877-820-3066