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Work Rate Adjustments During High-Intensity Interval Training in the Heat With Active and Passive Recovery

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Abstract

To test the hypotheses that maintaining target rating of perceived exertion (RPE) with active (ACT) versus passive (PASS) recovery during high-intensity interval training (HIIT) in the heat results in 1) greater thermal and cardiovascular strain, 2) larger work rate adjustments during work intervals, and 3) larger decreases in maximal aerobic capacity ($\dot{V}O_{2max}$). Eight adults completed 4 cycling trials in 35°C, consisting of an 8-min warm-up at RPE 12 followed by 1 (15_{ACT}, 15_{PASS}) or 5 (43_{ACT}, 43_{PASS}) rounds of HIIT (1 round = 4 min at RPE 17 and 3 min recovery at RPE 12 or rest) totaling 15 or 43 min of exercise. Each trial was immediately followed by measurement of $\dot{V}O_{2max}$. Rectal temperature was not different between conditions (43_{ACT}=38.3°C, 43_{PASS}=38.2°C, p = 0.003 for interaction, post-hoc both p > 0.09) but increased over time (both p < 0.001). HR during work intervals was comparable between conditions (p = 0.50) with a 15 b·min^{-1} increase between from interval 1 to 5 (p = 0.001). Average HR during recovery was 14 b·min^{-1} higher in the fifth versus first interval across conditions (p = 0.003) and + 24 b·min^{-1} during ACT versus PASS (p = 0.002). Percent change in work rate from first to fifth interval was larger during 43_{ACT} (−14%±24%) versus 43_{PASS} (−1%±16%; p = 0.04). $\dot{V}O_{2max}$ was 0.4 L•min^{-1} (11.5%) lower following 43-min compared to 15-min of HIIT (p = 0.008) but was not different between recovery mode (p = 0.69). Maintaining target RPE with ACT and PASS resulted in similar physiological strain but averaged 21 W lower power output by the final work interval during ACT. Five rounds of HIIT in a hot environment resulted in small–moderate reductions in $\dot{V}O_{2max}$.

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 consist 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 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. Indeed, with active recovery during HIIT in the heat, power output had to be lowered by 30 ± 28 W (Yoder et al. 2025). 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 heart rate (HR_{max}) and $\dot{V}O_{2max}$ in a temperate environment. For the 4 remaining experimental trials in the heat, participants were randomly assigned to a counterbalanced treatment order. These trials began with an 8-min warm-up at a rating of perceived exertion (RPE) (Borg and Noble 1974) of 12 followed by 1 or 5 rounds of HIIT (4 min of work at a RPE of 17 and 3 min of either active recovery [ACT] at a RPE of 12 or passive recovery [PASS] 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 ≥ 30 rev·min^{-1}. 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 1 depicts a general overview of the experimental trials.

15-min high-intensity interval training trial

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 difference between the change score in power output from the first to the final work interval in 43_{PASS} versus 43_{ACT}, assuming α = 0.05 and power ≈ 0.80 (Faul et al. 2009; Faul et al. 2007). 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 (2022). 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. Informed consent was obtained from all participants before the study procedures began. The study was approved by the Institutional Review Board of The University of Alabama (protocol #21-02-4259, May 12, 2021).

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. Pre-testing directions have been used by our laboratory previously and adherence was confirmed using a 24-h history questionnaire (Yoder et al. 2023; Mulholland et al. 2023). 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 then 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 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 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 $\dot{V}O$ 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 (128 ± 54 Watts) 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. $\dot{V}O_2$ and other metabolic responses were 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 $\dot{V}O_{2max}$ ≥ 20th percentile for cycle ergometer-based testing for their sex and age (American College of Sports Medicine 2022).

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}). Oxygen uptake was recorded continuously by the metabolic cart and total energy expenditure was calculated by the metabolic cart using the Weir equation (Weir 1949). 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 via venipuncture or intravenous catheter from a superficial forearm vein at the time points shown in Fig. 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 an 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 maintain 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.

The plateau verification procedure for $\dot{V}O_{2max}$ that was completed during the control trials was not completed in the experimental trials. However, the $\dot{V}O_2$ values measured after the 4 experimental trials were still referred to as $\dot{V}O_{2max}$ (instead of $\dot{V}O_{2peak}$). Expressing the values as $\dot{V}O_{2max}$ signified the observed changes were temporary changes in $\dot{V}O_{2max}$, which is consistent with naming conventions in other studies involving cardiovascular drift and $\dot{V}O_{2max}$ (Lafrenz et al. 2008; Yoder et al. 2025).

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 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).

Next, 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 (T_{sk}) was calculated using the following equation (Ramanathan 1964):

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

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 (T_b) was calculated using a weighted average of T_{re} and T_{sk} according to the following equation (Stolwijk and Hardy 1966):

$$T_b = 0.8(T_{re}) + 0.2(T_{sk}).$$

The core-to-skin thermal gradient was calculated as the difference between rectal temperature and mean skin temperature ($T_{re} - T_{sk}$). $\dot{V}O_2$ 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):

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

where $\dot{V}O_2$ is the rate of oxygen uptake in L•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 $\dot{M}$ and the external work rate ($\dot{W}$) on the cycle ergometer was calculated as the rate of metabolic heat production ($\dot{M} - \dot{W}$ and expressed in watts (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. A paired samples t-test was used to assess change in power from first to fifth recovery interval during 43_{PASS}. Paired samples t-tests were also used to evaluate differences in maximal values for T_{re}, T_{sk}, T_{re} – T_{sk}, and T_b between 43_{ACT} and 43_{PASS}.

Baseline data for control and experimental trials were analyzed using a 1-way repeated measures ANOVA. Planned contrasts were performed to compare $\dot{V}O_{2max}$ 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}, T_{sk}, power output during work intervals, 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):

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

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 $(\dot{M} - \dot{W})$, the average over the entire interval was used for data analysis; for T_{re} and 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

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•min^{-1}.

Hydration

Participants began trials adequately hydrated based on U_{SG} (mean ± SD, 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 was similar between conditions (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, p = 0.13 interaction). Similarly, percent change in plasma volume from pre- to post-exercise was not different between conditions (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). T_{sk} increased over time for the 43-min trials (p = 0.005), but the type of recovery was not influential (p = 0.16 for main effect of condition; Fig. 2a). T_{re} increased by 0.9°C 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 (Fig. 2a) (both p < 0.001). Since T_{re} increased proportionally more than 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 1). 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 differentially affected by conditions but increased from the first to final interval by 14 b·min^{-1} (p = 0.001 main effect of time; Table 1). HR and $\%HR_{max}$ averaged over each work interval followed a similar pattern (Fig. 3).

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 1). During recovery, final minute HR of the intervals increased over time during 43_{PASS} by 18 b·min^{-1} ($p < 0.001$), but the increase was not statistically significant during 43_{ACT} (10 b·min^{-1}, $\alpha' = 0.0125$, $p = 0.02$). 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).

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

Work	**ACT**		**PASS**		**Interaction**
	Interval 1	**Interval 5**	**Interval 1**	**Interval 5**	***P***
ΔPower output (W)	—	−28 ± 38	—	−6 ± 19	—
ΔPower output (%)	—	−14 ± 24	—	−1 ± 6§	—
Average HR (b•min^{-1})*	155 ± 7	173 ± 7	156 ± 15	176 ± 9	0.06
Final min HR (b•min^{-1})*	164 ± 7	178 ± 6	162 ± 15	176 ± 5	0.97
Final min %HR_{max}	87 ± 6	95 ± 3	87 ± 8	94 ± 4	0.91
$\dot{V}O_2$ (L·min^{-1})	2.4 ± 0.9	2.5 ± 0.9	2.5 ± 1.0	2.5 ± 1.0	0.58
Blood lactate (mmol·L^{-1})*	3.2 ± 1.5	3.9 ± 2.1	3.0 ± 1.1	4.2 ± 1.7	0.27
$\dot{M} - \dot{W}$ (W)	711 ± 264	762 ± 281	706 ± 276	703 ± 302	0.008
Recovery					
Power output (W)	90 ± 40	66 ± 35‡	—	—	—
Average HR (b•min^{-1})*†	152 ± 10	163 ± 8	125 ± 20	141 ± 14	0.13
Final min HR (b•min^{-1})	147 ± 11	157 ± 10	112 ± 21†	130 ± 16‡§	0.02
Final min %HR_{max}	78 ± 5	84 ± 3	60 ± 11†	70 ± 8‡§	0.02
Thermal sensation*	6.0 ± 1.0	7.0 ± 0.5	6.0 ± 0.5	7.0 ± 0.5	0.69
$T_{re} - T_{sk}$ (°C)*	0.9 ± 0.5	1.4 ± 0.6	1.1 ± 0.5	1.4 ± 0.5	0.25
T_b (°C)	37.2 ± 0.3	38.0 ± 0.4‡	37.3 ± 0.2	37.9 ± 0.3‡	0.005

ACT = 43-min trial with active recovery; PASS = 43-min trial with passive recovery; Δ Power output = change in power output from the first to fifth interval; Average HR = heart rate averaged over the entire interval; Final min HR = heart rate of the final minute of the interval; %HR_{max} = percent of maximal HR

averaged over the interval; HR = heart rate; $\dot{V}O_2$ = oxygen uptake; $M - W$ = rate of metabolic heat production; $T_{re} - T_{sk}$ = core-to-skin thermal gradient; T_b = mean body temperature.

* $p < 0.05$ 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 ACT during the same interval

Power output

The decrease in power output from the first to final work interval was not statistically different between conditions ($p = 0.07$ for the t test comparing change scores), 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). A 2-way ANOVA used to compare power output at each time point for each condition revealed no main effect of time ($p = 0.11$), condition ($p = 0.31$) or interaction ($p = 0.07$). Despite the lack of an interaction the magnitude of difference in power output seemed to be differentially affected by the recovery mode, as evidenced by a medium effect (22 W, ES = 0.42) for the magnitude of difference in power output between the first and fifth work interval in 43_{ACT} but a small effect (8 W; ES = 0.08) in 43_{PASS}. Furthermore, the difference between change scores between ACT and PASS for absolute power output was medium–large (ES = 0.73; 95% CI for mean difference in change scores = − 45, 2).

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 target RPE of 17. Additionally, the same 2 participants and 1 additional participant increased power output from the first to fifth work interval during 43_{PASS}.

Total energy expenditure over 43 min was greater in the 43_{ACT} (450 ± 161 kcal) trials compared to the 43_{PASS} (384 ± 142 kcal; $p = 0.003$; ES = 0.97; 95% CI for mean difference in = 38, 94).

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 after the fifth work interval ($p = 0.005$). Session RPE was comparable between conditions (43_{ACT} = 9 ± 1, 43_{PASS} = 8 ± 1; $p = 0.35$).

Maximal responses

Maximal responses are shown in Table 2 and Fig. 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 (α' = 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 on decrements in $\dot{V}O_{2max}$ ($p = 0.38$ for interaction; $p = 0.69$ for main effect of condition).

Maximal power output achieved during the 43-min trials was 12.7% lower than that during the 15-min trials ($p = 0.04$ for main effect of time). Lastly, Fig. 2b shows that T_{sk} was similar at maximum, but T_{re} was 0.2°C higher ($p = 0.047$) in 43_{ACT} compared to 43_{PASS}.

Table 2
Maximal responses during a graded exercise test following 15 and 43 min of HIIT exercise in a hot environment with active or passive recovery.

Condition					**Interaction**
Variable	**15_{ACT}**	**43_{ACT}**	**15_{PASS}**	**43_{PASS}**	***P***
$\dot{V}_E$ (STPD, L·min^{-1}) *	94.0 ± 23.5	81.5 ± 21.0	93.8 ± 20.9	86.0 ± 25.7	0.17
$\dot{V}O_2$ (mL·kg^{-1}·min^{-1}) *	45.0 ± 14.0	39.5 ± 13.1	45.1 ± 12.7	41.0 ± 12.0	0.48
RER *	1.06 ± 0.07	0.97 ± 0.06	1.03 ± 0.09	0.98 ± 0.08	0.46
RPE †	19 ± 1	20 ± 1	19 ± 1	19 ± 1	0.79
HR (b·min^{-1}) †	188 ± 9	188 ± 10	187 ± 9	186 ± 7	0.76
Blood lactate (mmol·L^{-1}) *†	5.4 ± 1.3	4.3 ± 1.5	7.2 ± 1.8	4.6 ± 1.8	0.16
T_b (°C)	—	38.2 ± 0.4	—	38.0 ± 0.3‡	—
$T_{re} - T_{sk}$ (°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	0.09
Power output (W) *	209 ± 82	178 ± 56	209 ± 74	188 ± 73	0.48

$\dot{V}_E$ = minute ventilation; $\dot{V}O_2$ = oxygen uptake; RER = respiratory exchange ratio; RPE = rating of perceived exertion; HR = heart rate; T_b = mean body temperature; T_{re} = rectal temperature; 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 decrease 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 fifth 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 fifth 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 that of 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, 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 ($\dot{M} - \dot{W}$) was expected, which would in turn result in lower T_{re}. Despite the different recoveries, T_{re} increased by the same magnitude during the passive recovery condition as during the active recovery condition. The significant interaction for ($\dot{M} - \dot{W}$) suggests it increased more over time in 43_{ACT} vs. 43_{PASS}, but post hoc comparisons revealed no individual differences, and the ES comparing ($\dot{M} - \dot{W}$) at the fifth work interval was small (0.19). This suggests the difference in ($\dot{M} - \dot{W}$) between conditions was insufficient to result in differences in heat balance. Furthermore, even if the ~ 59 W lower ($\dot{M} - \dot{W}$) 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 ($\dot{M} - \dot{W}$) with active recovery. The authors concluded that core temperature was similar between the conditions because of reduced dry and evaporative heat loss during passive recovery. In the current study, perhaps reductions in dry and evaporative heat loss during the passive recovery intervals—combined with modestly (~ 8%) lower ($\dot{M} - \dot{W}$) during 43_{PASS} work intervals—partially explains the comparable core temperatures between conditions.

Not surprisingly, active recovery resulted in greater overall cardiovascular strain during recovery intervals compared to passive recovery as evidenced by 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 recorded during the entire time-to-exhaustion of repeated 15-s bouts 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, participants cycled 125% longer before reaching exhaustion when completing the interval workout 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). Similarly, in a running study evaluating active and passive recovery, participants ran faster during the sprint intervals with passive recovery (Germano et al. 2022). 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 decrease 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 change 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 43-min of interval exercise in the heat using RPE [−15.6% decline in $\dot{V}O_{2max}$] (Yoder et al. 2025)] and one using target HR (and accompanying decrements in work rate) to mitigate cardiovascular strain [non-statistically significant − 6.5% decline in $\dot{V}O_{2max}$ (Yoder et al. 2023)]. It was also similar to the 13% and 16% decline in $\dot{V}O_{2max}$ observed following 45 min of continuous exercise in the heat during follicular and luteal phase of the menstrual cycle, respectively (Stone et al. 2021). The magnitude of change is also comparable to the decline observed by others during a repeated time trial performance (4 × 16.5 min with 5 min of 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% of control $\dot{V}O_{2max}$ at the end of the final time trial (Périard and Racinais 2015). 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 recovery conditions 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 T_{sk} over time, but the effect on the core-to-skin thermal gradient later in exercise was small and comparable between conditions (Table 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, especially considering that experimental trials were separated by at least 48 h. 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 the participants who appeared to pace 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 dissociation 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 maintaining high intensity training stimulus. However, active recovery resulted in greater total energy expenditure by 66 ± 42 kcal 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 on average lower power output by the final work interval 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 a proportional overall training stimulus compared to protocols utilizing active recovery. Furthermore, if maximizing total energy expenditure is the goal of the session and cardiovascular strain is inconsequential, then active recovery may be more beneficial.

Abbreviations

15_{ACT}
15-minute active recovery trial
15_{PASS}
15-minute passive recovery trial
43_{ACT}
43-minute active recovery trial
43_{PASS}
43-minute passive recovery trial
α'
Alpha prime
ANOVA
Analysis of variance
CI
Confidence Interval
ES
Effect size
GXT

Graded exercise test

HIIT

High intensity interval training

HR

Heart rate

M

Metabolic rate

$\dot{M} - \dot{W}$

Rate of metabolic heat production

RH

Relative humidity

RPE

Rating of perceived exertion

SD

Standard deviation

SV

Stroke volume

T_b

Mean body temperature

T_{calf}

Skin temperature of the calf

T_{chest}

Skin temperature of the chest

T_{delt}

Skin temperature of the deltoid

T_{re}

Rectal temperature

T_{sk}

Mean skin temperature

T_{thigh}

Skin temperature of the thigh

U_{SG}

Urine specific gravity

$\dot{V}_E$

Minute ventilation

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

Maximal oxygen uptake

$\dot{V}O_2$

Oxygen uptake

Declarations

Competing interest information: The authors declare no competing interests.

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Author contribution

All authors contributed to study design and reviewed and edited the manuscript. HAY and AMM completed data collection. HAY completed data entry. HAY and JEW completed data analysis and writing.

Data Availability

Data generated or analyzed during this study are available from the corresponding author upon reasonable request.

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Figures

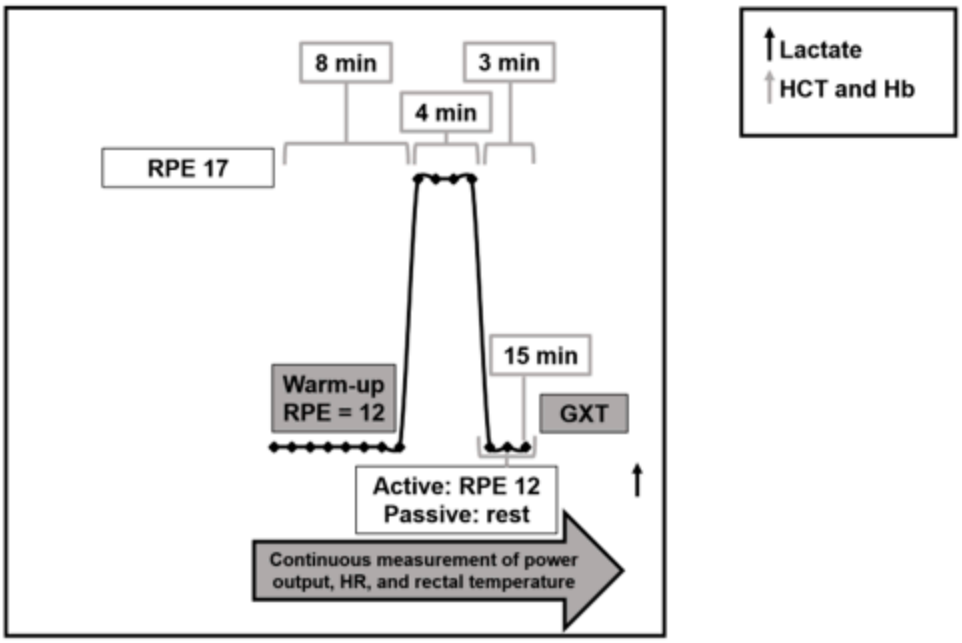

Figure 1

General exercise protocols for the 15- and 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

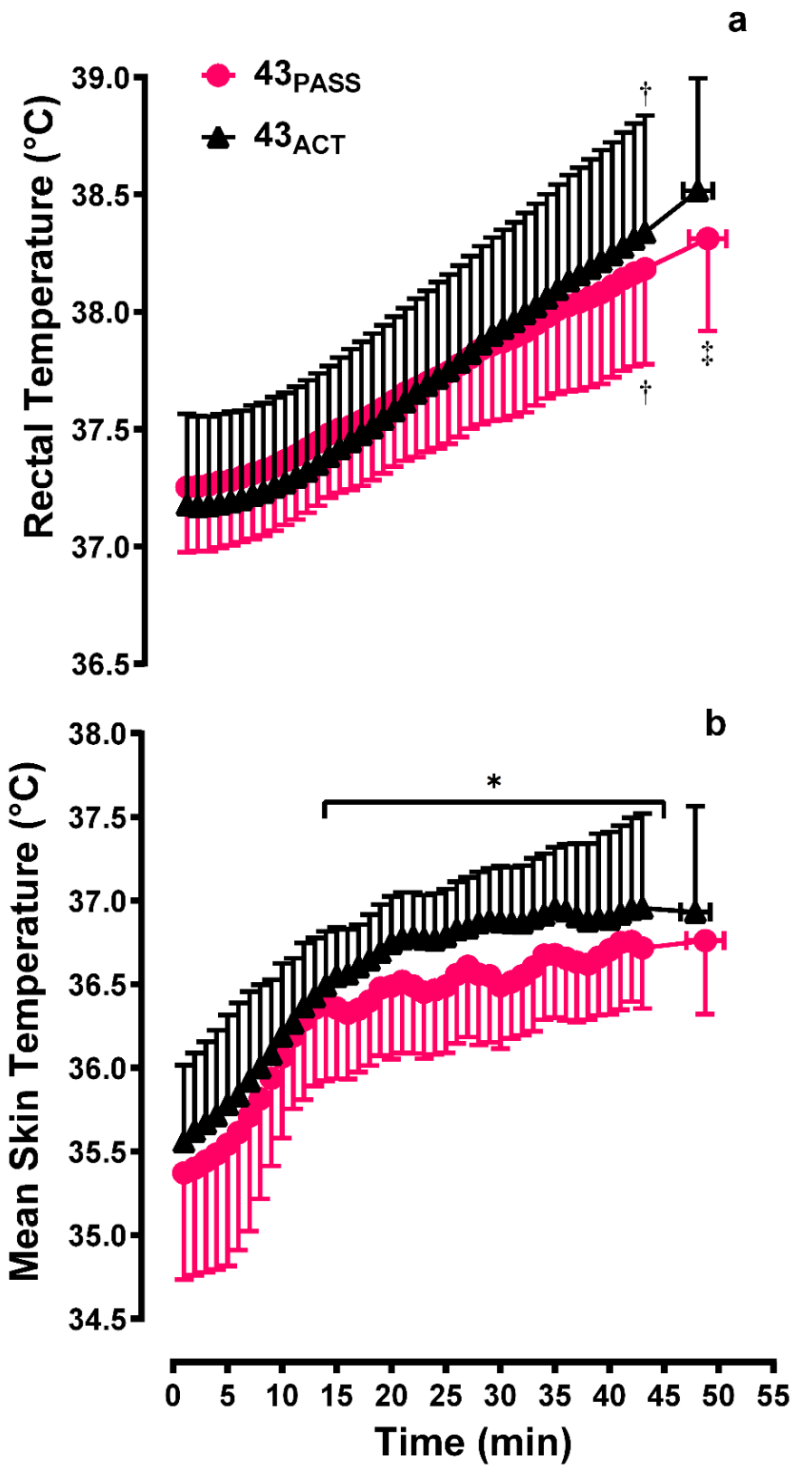

Figure 2

Mean ± SD rectal temperature (panel a) and mean skin temperature (panel b) during the 43-min trials to the end of the graded exercise test. 43_{PASS} = 43-min trial with passive recovery; 43_{ACT} = 43-min trial with active recovery. $^{*}p < 0.05$ for main effect of time from 15 to 43 min; $^{\dagger}p < 0.05$ compared to min 15 of the

respective trial; ‡ $p < 0.05$ compared to 43_{ACT} at end of graded exercise test. ‡ $p < 0.05$ for compared to 43_{ACT} at end of graded exercise test.

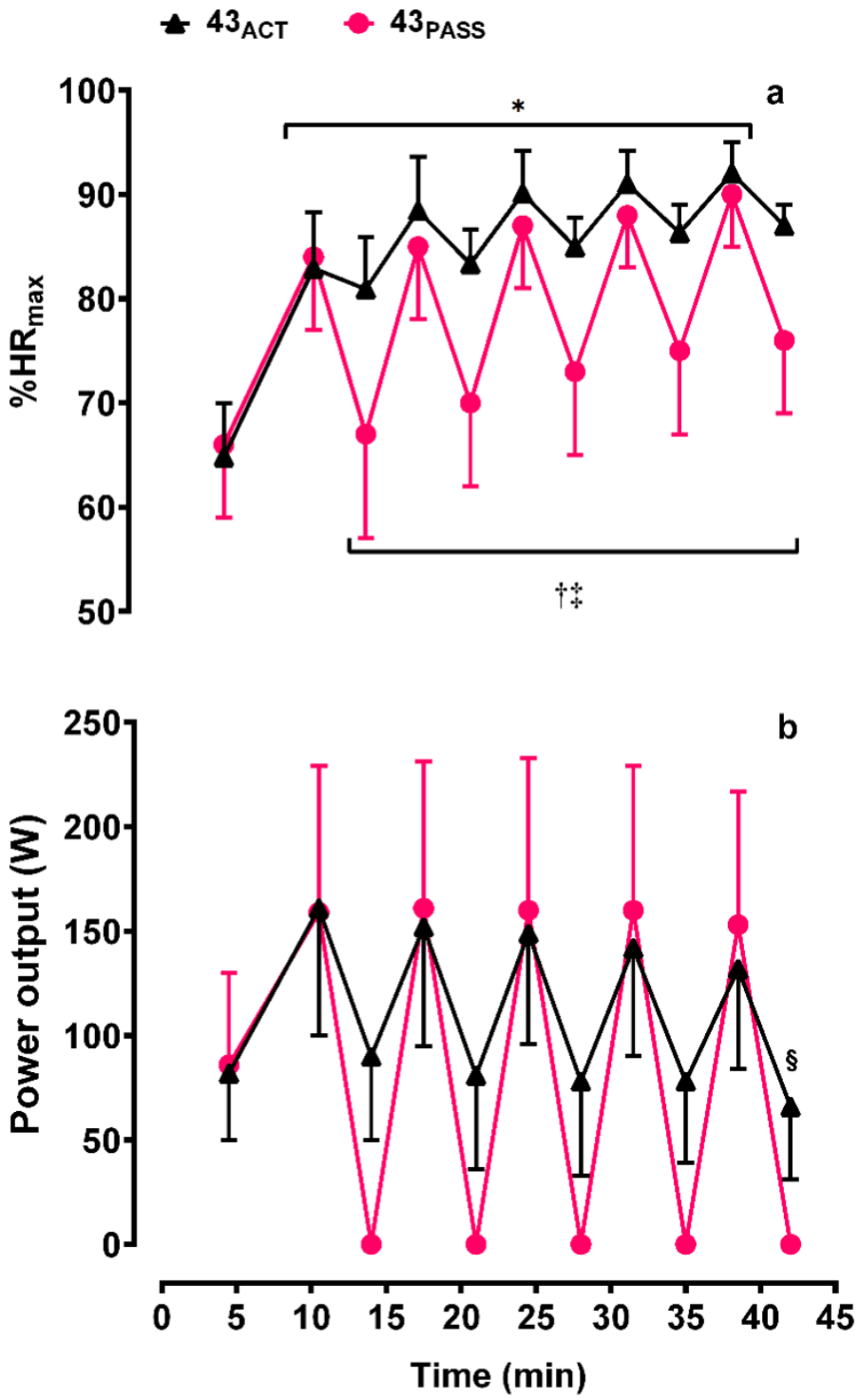

Figure 3

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. 43_{ACT} = 43-min trial with active recovery; 43_{PASS} = 43-min trial with passive recovery; * p < 0.05 for main effect of time for work intervals; † p < 0.05 for main effect of time for recovery intervals; ‡ p < 0.05 for main effect of condition for recovery intervals, § p < 0.05 compared to first interval of same condition (t-test).

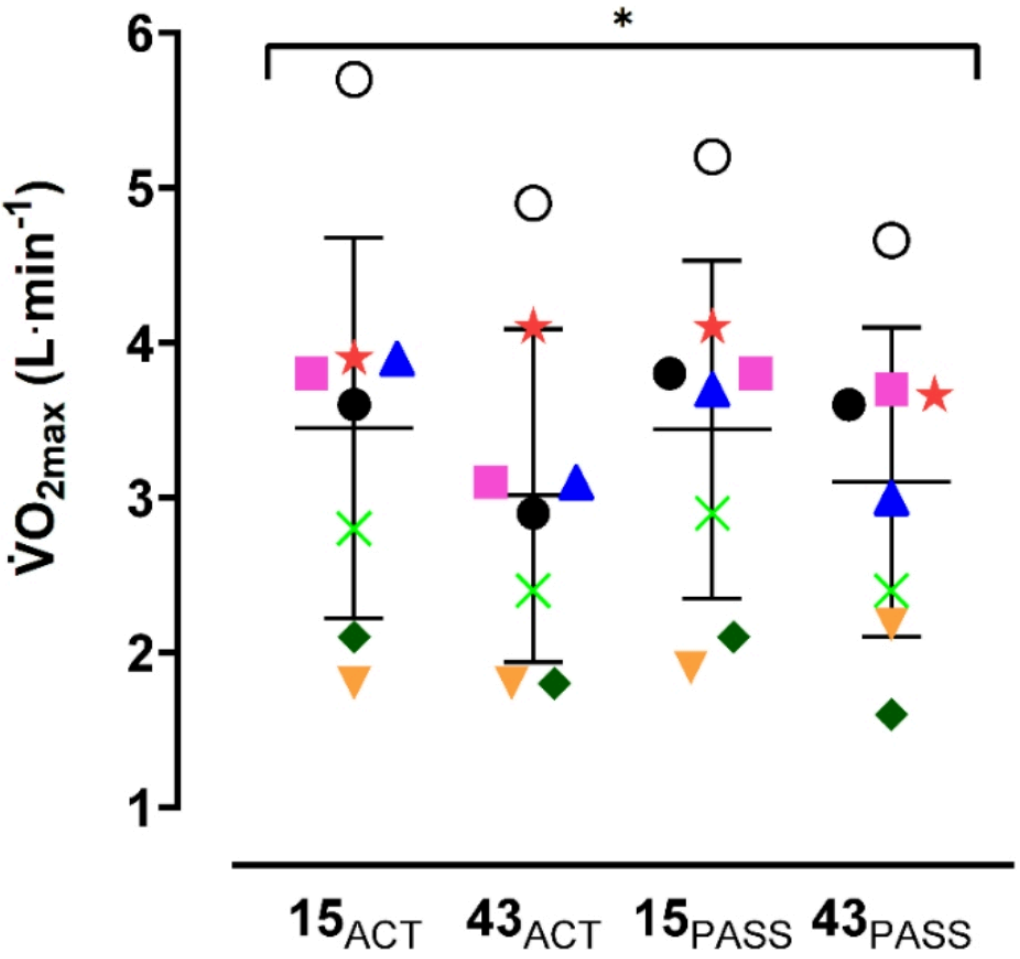

Figure 4

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.