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Repeated-sprint training in heat and hypoxia: Acute responses to manipulating exercise-to-rest ratio

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ABSTRACT

The aim of this study was to investigate acute performance and physiological responses to the manipulation of exercise-to-rest ratio (E:R) during repeated-sprint hypoxic training (RSH) in hot conditions. Twelve male team-sport players completed two experimental sessions at a simulated altitude of ~3000 m (F_{iO_2} 0.144), air temperature of 40°C and relative humidity of 50%. Exercise involved either $3 \times 5 \times 10$ -s (E:R_{1:2}) or $3 \times 10 \times 5$ -s (E:R_{1:4}) maximal cycling sprints interspersed with active recoveries at 120W (20-s between sprints, 2.5 and 5-min between sets for E:R_{1:2} and E:R_{1:4} respectively). Sessions were matched for overall sprint and total session duration (47.5-min). Peak and mean power output, and total work were greater in E:R_{1:4} than E:R_{1:2} ($p < 0.05$). Peak core temperature was significantly higher in E:R_{1:4} than E:R_{1:2} (38.44 ± 0.33 vs. $38.20 \pm 0.35^\circ\text{C}$, $p = 0.028$). Muscle deoxygenation magnitude during sprints was greater in E:R_{1:2} (28.2 ± 1.6 vs. $22.4 \pm 4.6\%$, $p < 0.001$), while muscle reoxygenation did not differ between conditions ($p > 0.05$). These results indicate E:R_{1:4} increased mechanical power output and core temperature compared to E:R_{1:2}. Both protocols had different effects on measures of muscle oxygenation, with E:R_{1:2} generating greater muscle oxygen extraction and E:R_{1:4} producing more muscle oxygenation flux, which are both important signals for peripheral adaptation. We conclude that the E:R manipulation during RSH in the heat might be used to target different physiological and performance outcomes, with these findings forming a strong base for future mechanistic investigation.

KEYWORDS

Environmental physiology; performance; team sport; training; physiology

Highlights

- During a typical repeated-sprint training session conducted in hot and hypoxic conditions, an exercise-to-rest ratio of 1:4 during sprint efforts displayed an increased mechanical power output compared to an exercise-to-rest ratio of 1:2. This represents a potentially useful increase in training stimulus.
- An exercise-to-rest ratio of 1:2 generated greater muscle oxygen extraction, while an exercise-to-rest ratio of 1:4 resulted in more muscle oxygenation flux and a higher core temperature, indicating key markers of environment-related physiological strain were varied between conditions.
- Exercise-to-rest ratio manipulation may be used to target different physiological and performance outcomes when prescribing repeated-sprint training in hot and hypoxic conditions.

Introduction

Repeated-sprint training in hypoxia (RSH) is an increasingly popular method of training in elite athlete cohorts (Girard, Brocherie, Goods, & Millet, 2020), and has been shown to improve repeated-sprint ability in normoxia by 1-5% compared to matched training at sea-level (Brocherie, Girard, Faiss, & Millet, 2017). The concurrent use of heat with RSH has been recently

investigated as a method of potentially enhancing in-session sprint performance (Dennis, Goods, Binnie, Girard, Wallman, Dawson, Billaut, et al., 2021; Dennis, Goods, Binnie, Girard, Wallman, Dawson, & Peeling, 2021; Yamaguchi et al., 2020). Reportedly, ambient air temperatures of up to 40°C do not negatively affect repeated-sprint cycling performance (Dennis, Goods, Binnie, Girard, Wallman, Dawson, Billaut, et al., 2021) as

long as a deleterious core temperature (T_c) is not reached (Girard, Brocherie, & Bishop, 2015). Studies in normoxia have shown that adding heat to a repeated-sprint session can augment overall mechanical power output (Girard, Bishop, & Racinais, 2013), due to mechanisms such as a faster rate of phosphocreatine utilisation (Gray, De Vito, Nimmo, Farina, & Ferguson, 2006) and decreased muscle and joint viscous resistance at higher temperatures (Bishop, 2003).

Current guidelines for the implementation of RSH with athletic cohorts include scope for great variation around in-session interval duration (ranging from 4 to 15-s), exercise-to-rest ratio (E:R; 1:2–1:5), and inter-set rest (3–5-min) (Brocherie et al., 2017). Manipulating these parameters during an RSH session can acutely alter a range of performance, physiological, and perceptual variables, such as power production, T_c , muscle oxygenation or rating of perceived exertion (RPE), which in turn may increase the effectiveness of the RSH session. The realisation of potential physiological and performance benefits as a result of normoxic and temperate training has been found to be influenced by E:R and sprint duration (Fiorenza et al., 2019; Iaia et al., 2017). More specifically, the structure of the protocol performed during RSH at hot temperatures may also influence training outcomes. For example, Yamaguchi, Kasai, Hayashi, Yatsutani, and Goto (2019) observed higher peak power production during the first set of 3×10 -s cycle sprints in hypoxia with added heat (35°C, 50% relative humidity [RH]) compared to cooler conditions (20°C, 50% RH), employing an E:R of 1:4 (Yamaguchi et al., 2020). In contrast, research from our laboratory found no differences in mean or peak power output during the completion of 3 sets of 5×10 -s sprints when comparing exercise in conditions identical to those employed by Yamaguchi et al., except with an E:R of 1:2 (Dennis, Goods, Binnie, Girard, Wallman, Dawson, & Peeling, 2021). This comparison highlights the capability of manipulating E:R when performing RSH in hot ambient conditions to affect training outcomes and indicates the need for further research investigating the effect of E:R on performance, T_c , and other physiological variables during this type of session.

Repeated fluctuations in muscle oxygen utilisation can augment muscle oxidative capacity following high-intensity exercise (Daussin et al., 2008), making muscle oxygenation an important contributor to the efficacy of RSH. Varying sprint lengths and E:R during RSH affects sprint energetics (Balsom, Seger, Sjödín, & Ekblom, 1992), and subsequently influence muscle oxygenation. However, it is unknown how different exercise

protocols (specifically different E:R) may affect muscle oxygenation during RSH under hot conditions. For example, Yamaguchi et al. (2021) found that *vastus lateralis* muscle oxygenation measured during repeated 6-s sprints via Near-Infrared Spectroscopy (NIRS) was no different in hot and hypoxic (35°C, 50% relative humidity (RH), 0.145 $F_{I}O_2$) compared to cool and hypoxic (23°C, 50% RH, 0.145 $F_{I}O_2$) conditions (Yamaguchi et al., 2021). In contrast, prior research from our laboratory (Dennis, Goods, Binnie, Girard, Wallman, Dawson, Billaut, et al., 2021) indicated that muscle deoxygenation during 10-s sprints was up to 8% greater in hot and hypoxic (35°C and 40°C, both 50% RH, 0.144 $F_{I}O_2$) compared to cool and hypoxic conditions (20°C, 50% RH, 0.144 $F_{I}O_2$). During both studies, no differences in cycling performance were observed between any conditions. However, Yamaguchi et al. (2021) employed a RSH protocol based on an E:R of 1:6 during sets, whereas our protocol (Dennis, Goods, Binnie, Girard, Wallman, Dawson, Billaut, et al., 2021) utilised an E:R of 1:2. Considering these results, it is likely that the intricacies of the protocol design (specifically E:R), rather than the environmental conditions, may be responsible for the differences in variation of muscle oxygenation between the similar conditions across studies that were observed.

Accordingly, the aim of the current study was to investigate the acute effect of manipulating E:R during RSH in hot ambient conditions on performance and physiological responses. We hypothesised that a smaller E:R (i.e. more rest per unit of exercise, 1:4) with shorter sprints would result in an augmented mechanical power output and T_c compared to a greater E:R (1:2) with longer duration sprints. We further hypothesised that a decrease in muscle oxygenation would be exacerbated with a greater E:R.

Materials and methods

Participants

Twelve trained (McKay et al., 2022) male athletes from a variety of team sports were recruited (age: 19.8 ± 2.8 y; height: 183.6 ± 6.1 cm; body mass: 82.7 ± 8.0 kg; BMI: 24.5 ± 2.38). Participants were living and training near sea-level and had no exposure to altitude (>600 m) in the month prior to testing. Trials were conducted between November and March in Perth, Western Australia; as such, some seasonal heat acclimatisation may have been present. Written informed consent was obtained from participants prior to commencement. For participants under 18 years of age, parental consent was also obtained. Ethics approval was received from the host institution's human research ethics

committee (XXXXXXXXXX), conforming to the Declaration of Helsinki.

Design

This research utilised a randomised and repeated-measures design.

Experimental overview

Participants attended one familiarisation and two experimental trials. Sessions were separated by between 3 and 14 days. Trials took place in a 213 m³ environmental chamber (The Altitude Centre, London, UK) situated near sea-level. During all sessions, the chamber was set to a simulated altitude of ~3000 m (F₁O₂ 0.144), air temperature of 40°C, and RH of 50%. There was no active airflow on participants in the environmental chamber.

A different exercise protocol was performed at the two experimental trials, where the E:R was manipulated. These were conducted in a randomised order and were matched for both sprint and overall duration. The tasks were:

- (a) 3 × 10 × 5-s “all-out” sprints, with 20-s active recoveries (~120W) between sprints, and an inter-set active recovery period of 2.5-min (E:R_{1:4}).
- (b) 3 × 5 × 10-s “all-out” sprints, with 20-s active recoveries (~120W) between sprints, and an inter-set active recovery period of 5-min (E:R_{1:2}).

Participants kept a food diary for the 24-h prior to each session. During this time, they were asked to maintain a normal diet, but to avoid the consumption of alcohol. During experimental sessions, participants drank water *ad libitum*. Water bottles were weighed pre- and post-session to measure fluid intake (to 1 mL).

Experimental procedures

Familiarisation trial

Participants initially attended the laboratory to familiarise themselves with the climate chamber (in temperate conditions), exercise tasks, and the equipment that would be used during the experimental trials. During the familiarisation exercise, participants were instructed to sprint maximally when required, and not employ any pacing strategies. This familiarisation task included a standardised warm-up, one set of exercise from each experimental session (i.e. E:R_{1:4} and E:R_{1:2}), and a standardised cool-down.

Experimental trials

On each testing day, participants first provided a mid-stream urine sample for the measurement of urine-specific gravity (U_{SG}) via refractometer (URC-NE, 1.000-1.050, Atago, Tokyo, Japan); no participants were hypohydrated (U_{SG} > 1.030). Body mass was then measured using a digital platform scale (FG-150K, A&D Weighing, Adelaide, Australia). Next, participants were left in private to self-insert a rectal probe (~10 cm beyond the anal sphincter) for the measurement of T_c (RET-1, Physitemp Instruments Inc, Clifton, NJ, USA). Following this, a NIRS monitor (Moxy Monitors, Fortiori Design, Hutchinson, MN, USA) was attached to the muscle belly of the right *vastus lateralis*, approximately 15 cm above the proximal border of the patella, as per the methods of Billaut and Buchheit (2013). The NIRS monitor was fitted into a custom-built polyurethane light shield and attached using non-woven adhesive fabric tape, which was then covered by a dark bandage to reduce the intrusion of extraneous light. The unit (sampling rate 0.5 Hz) calculated the percentage of haemoglobin containing oxygen (S_mO₂) present beneath the device. Further information regarding the NIRS unit can be found in Crum et al (Crum, O'Connor, Van Loo, Valckx, & Stannard, 2017).

After entering the environmental chamber participants sat on a front-access air-braked cycle ergometer (Model EX-10, Repco, Melbourne, Australia) while they were instrumented with a chest-strap heart rate monitor (H1, Polar Electro, Kempele, Finland) and wrist-watch receiver (RS300X, Polar Electro, Kempele, Finland), as well as a wrist-worn, finger-tip pulse oximeter (S_pO₂, WristOx2 Model 3150, Nonin Medical, Inc, Plymouth, MN, USA). Skin thermistors (SST-1, Physitemp Instruments Inc, Clifton, NJ, USA) were taped to the sternum, right forearm, right mid-thigh, and right mid-calf, for the measurement of skin temperature (T_{sk}). This procedure took no longer than 5-min per session. Both T_{sk} and T_c were continuously monitored throughout the exercise protocol using DASYLab Light software (Version 11, National Instruments, Ireland Resources Ltd., Dublin, Ireland) at a sampling frequency of 0.1 Hz. Mean T_{sk} was calculated using the formula described by Ramanathan (1964).

Before exercise commencement, a capillary blood sample was taken from the earlobe and analysed for blood lactate concentration (BLa; Lactate Plus Meter, Nova Biomedical, Waltham, MA, USA). Subsequently, a 20-min standardised cycling warm-up at 120W was completed, where participants performed a series of 5-s progressive sprint efforts every 2-min during this period. For

each submaximal sprint, they were instructed to work at a subjective “sense of effort” of 5, 6, 7, 7, 8, 8, 9, 9, 10 and 10 on the modified Borg CR10 “sense of effort scale” (Christian, Bishop, Girard, & Billaut, 2014). Two minutes following the warm-up, participants commenced one of either the E:R_{1:4} or E:R_{1:2} exercise tasks. Following either protocol, BL_a was collected 60-s after the cessation of exercise, and a 10-min standardised cool-down (120W cycling) was enforced.

Repeated-sprint ability was analysed by calculating peak power output (PPO) and mean power output (MPO) for each sprint (Cyclemax version 6.3, School of Human Sciences, UWA), averaged across each set. Total work during each set was also calculated. From MPO values, a percentage decrement score for each set was calculated as per Spencer et al. (Spencer, Dawson, Goodman, Dascombe, & Bishop, 2008). Heart rate (HR), thermal sensation and S_{pO_2} were assessed at baseline immediately prior to the beginning of the warm-up, after the warm-up, after each set of exercise, and at the conclusion of the cool-down. Thermal sensation was reported at the aforementioned time points using a 9-point Likert scale (Young, Sawka, Epstein, DeCristofano, & Pandolf, 1987), and RPE was also recorded at each of these time points (except for baseline) using the Borg 6–20 scale (Borg, 1982).

Regarding NIRS analysis, absolute maximal and minimal S_{mO_2} values ($S_{mO_{2max}}$ and $S_{mO_{2min}}$) were extracted for subsequent analysis from each sprint during each exercise set, and each post-exercise recovery interval, respectively. We further calculated the deoxygenation amplitude during each set of exercise ($\Delta S_{mO_{2exercise}}$) as the average difference between $S_{mO_{2max}}$ and $S_{mO_{2min}}$ values for each sprint within the set. Similarly, reoxygenation amplitude during the post-exercise recovery ($\Delta S_{mO_{2recovery}}$) was determined as the average difference between $S_{mO_{2min}}$ for each sprint within a set and $S_{mO_{2max}}$ at the end of the subsequent recovery period. These values were normalised by calculating them as a percentage of the Δ from the lowest point recorded during the session to the highest point, averaged across 12-s (six data collection periods) to smooth outlying data. Similar methods have been used and described previously (Paquette, Bieuzen, & Billaut, 2020).

At the conclusion of the cool-down, participants exited the environmental chamber and were seated in the lab with the right lower limb parallel to the floor. A manual blood pressure cuff (Heine Gamma XXL Thigh 20 × 86 cm, Heine Optotechnik, Herrsching, Germany) was then fitted to the right thigh proximal to the NIRS monitor and inflated to 220 mmHg to elicit full arterial

occlusion for 5-min, as per prior recommendations (Brown et al., 2018). This period was used to aid in normalising the NIRS values, with the associated occlusion and reperfusion providing anchor points for whole session minimal and maximal muscle oxygenation. Subsequently, the NIRS monitor was removed, and a post-exercise dry and semi-nude (underpants on) body mass was recorded. Whole-body sweat production was then calculated as per Shapiro et al (Shapiro, Pandolf, & Goldman, 1982).

Statistical analysis

A General Linear Mixed Model using the R (R Core Team, 2020) package lme4 was used to analyse data. A random intercept was included to adjust for baseline levels and inter-individual homogeneity. All models were estimated using Restricted Maximum Likelihood. *P*-values were obtained using Type II Wald F tests with Kenward–Roger degrees of freedom as implemented in the R package car. Post-hoc pairwise-comparisons using the Tukey HSD test were performed using the R package emmeans if a significant main effect for condition or time was observed. The null hypothesis was rejected at $p < 0.05$. All data are expressed as mean ± standard deviation.

Results

A main condition effect was observed for MPO ($p = 0.002$), PPO ($p = 0.007$) and total work ($p = 0.002$). Total work and MPO were both 5.2% higher and PPO 4.4% higher during the E:R_{1:4} protocol as compared to E:R_{1:2} across all sets (Figure 1). A main effect for time (all $p < 0.001$) demonstrated that MPO, PPO and total work were higher during set 1 as compared to sets 2 and 3 within E:R_{1:4}, and higher during set 1 as compared to set 3 within E:R_{1:2} (all $p < 0.001$).

Significant condition effects were observed for both $\Delta S_{mO_{2exercise}}$ ($p < 0.001$) and $S_{mO_{2min}}$ ($p = 0.034$, Figure 2). A greater $\Delta S_{mO_{2exercise}}$ was found for E:R_{1:2} compared to E:R_{1:4} across all sets ($p < 0.05$). This also resulted in an overall lower $S_{mO_{2min}}$ in E:R_{1:2} ($p < 0.05$). No condition effects were observed for $\Delta S_{mO_{2recovery}}$ and $S_{mO_{2max}}$. Significant time effects ($p < 0.05$) were observed for all S_{mO_2} variables except $S_{mO_{2min}}$. Both $\Delta S_{mO_{2exercise}}$ and $\Delta S_{mO_{2recovery}}$ values during sets 2 and 3 were lower than set 1 within E:R_{1:4} (all $p < 0.023$). However, no post-hoc effects ($p > 0.05$) were observed for $S_{mO_{2max}}$.

A significant condition effect was observed for T_c , showing that T_c during E:R_{1:4} was significantly higher than E:R_{1:2} (38.10 ± 0.41 vs. $37.89 \pm 0.39^\circ\text{C}$, $p < 0.001$).

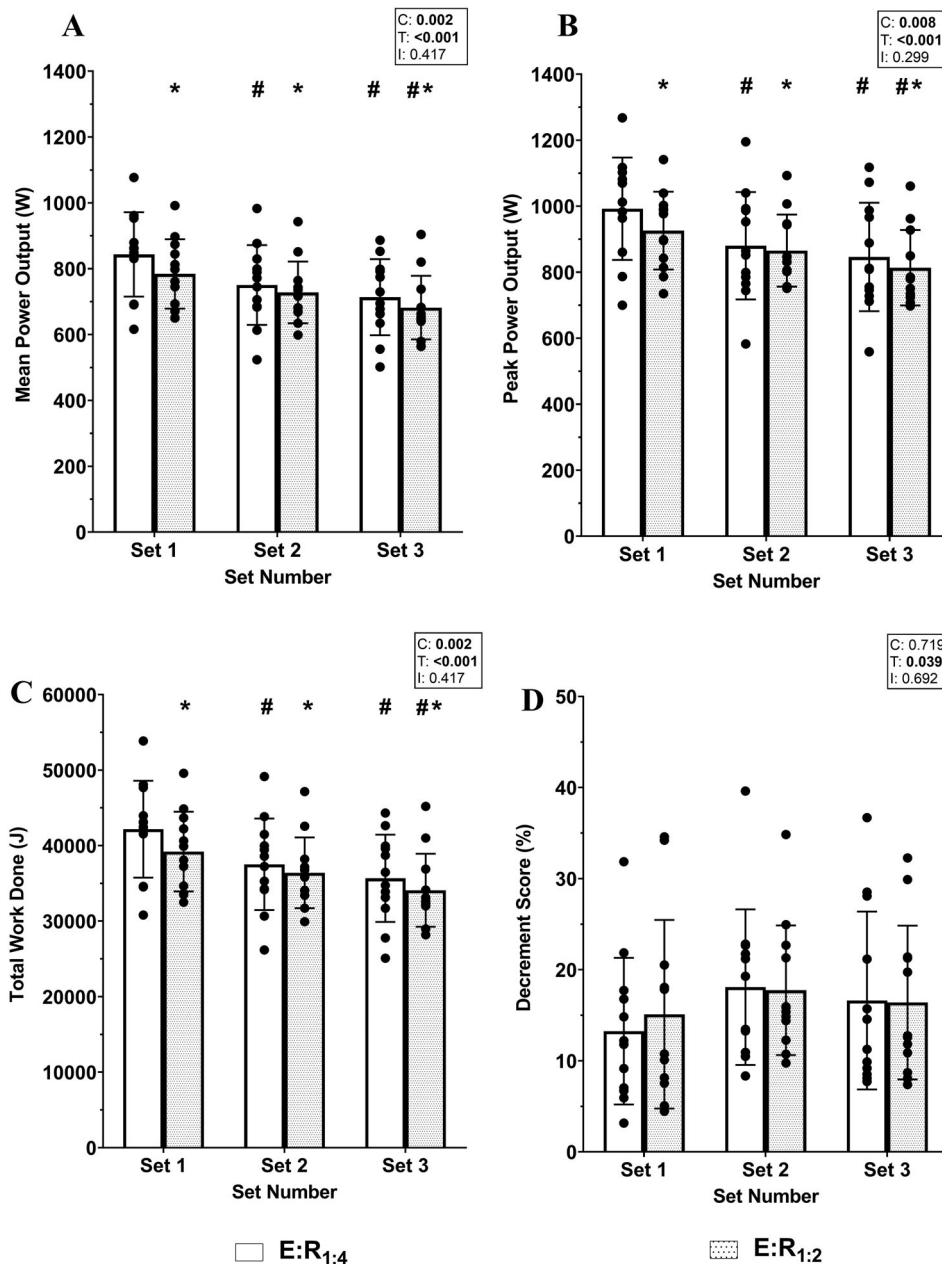


Figure 1. (A) Mean power output, (B) peak power output, (C) total work, and (D) sprint decrement score for each of the three sets in E:R_{1:4} and E:R_{1:2} ($n = 12$). Values are means \pm SD, $n = 12$. Main effects for condition (C), time (T), and interaction (I) are presented. E:R_{1:4}: Exercise-to-rest ratio of 1:4; E:R_{1:2}: Exercise-to-rest ratio of 1:2. # Significant within group difference compared to Set 1; * Significant between group difference compared to E:R_{1:4}.

Significant time effects showed that T_c and T_{sk} (both $p < 0.001$) increased across sets, independent of conditions ($p < 0.05$; Table 1). Higher peak T_c was reached with E:R_{1:4} ($38.44 \pm 0.33^\circ\text{C}$) compared to E:R_{1:2} ($38.20 \pm 0.35^\circ\text{C}$, $p = 0.028$).

Significant time effects were evident for S_{pO_2} , HR, thermal sensation and RPE (all $p < 0.001$; Table 2). Additionally, RPE readings were higher for E:R_{1:2} than E:R_{1:4} throughout the protocol ($p = 0.019$). There was a significant condition \times time interaction for BLA ($p = 0.027$) as the increase in BLA from baseline to end-

exercise was higher for E:R_{1:2} compared to E:R_{1:4} ($p = 0.017$). No interaction effects were evident for any other collected variables ($p > 0.05$).

Sweat loss (1065 ± 209 vs. 1052 ± 357 mL; $p = 0.909$) and water intake (662 ± 236 vs. 824 ± 301 mL; $p = 0.079$) were not different between conditions.

Discussion

The aim of the present study was to investigate the acute effect of manipulating E:R during an

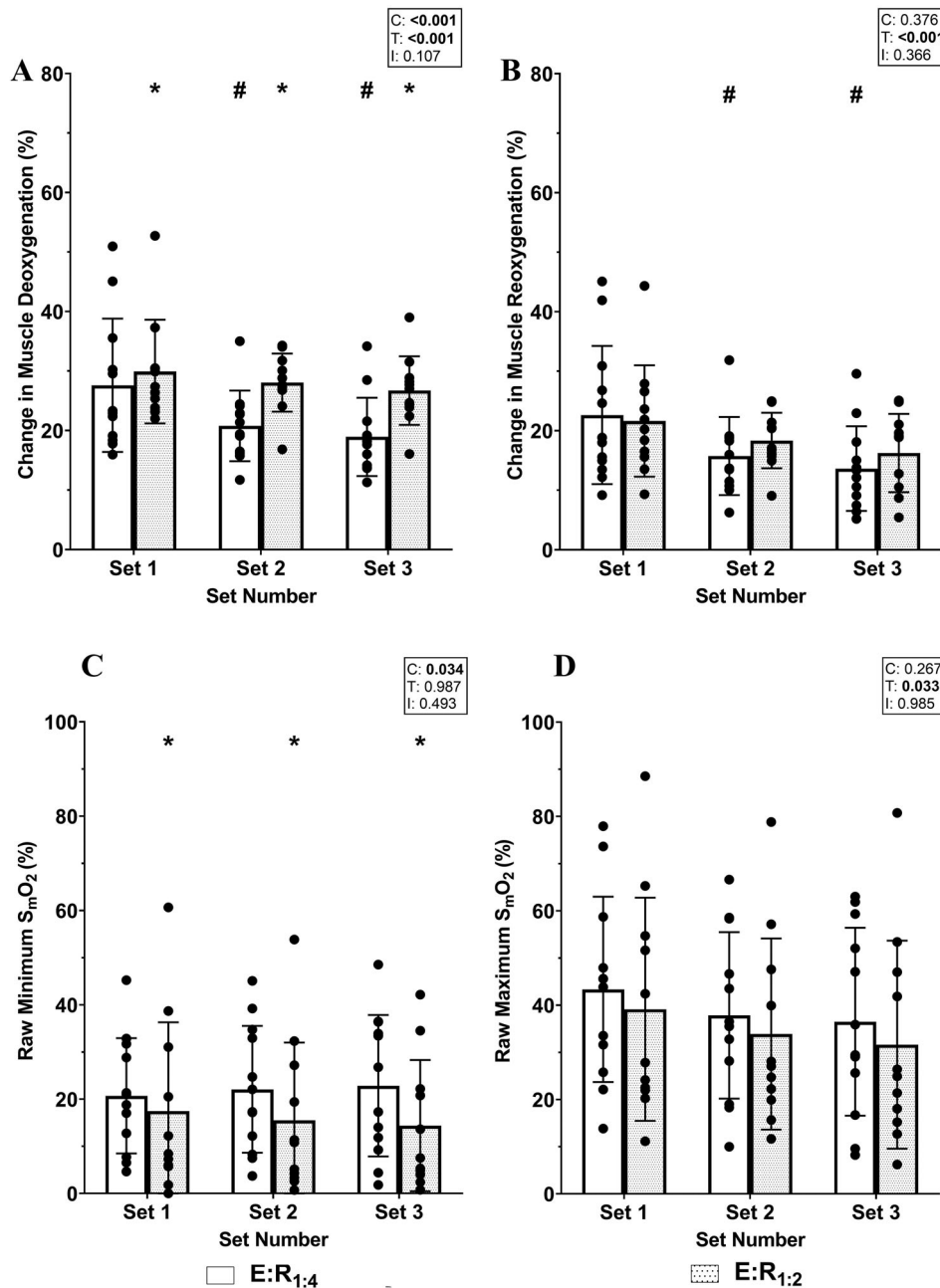


Figure 2. Mean change in S_{mO_2} during (A) exercise, and (B) between-sprint recovery, as well as raw minimum S_{mO_2} during exercise (C) and raw maximum S_{mO_2} during between-sprint recovery (D) for each of the three sets in E:R_{1:4} and E:R_{1:2} ($n = 12$). Values are means \pm SD, $n = 12$. Main effects for condition (C), time (T), and interaction (I) are presented. E:R_{1:4}: Exercise-to-rest ratio of 1:4; E:R_{1:2}: Exercise-to-rest ratio of 1:2. # Significant within group difference compared to Set 1; * Significant between group difference compared to E:R_{1:4}. Note: All figures were created using GraphPad Prism Version 9.3.1.

RSH session performed at an ambient air temperature of 40°C and 50% RH. We observed that cycling performance was augmented for E:R_{1:4} compared to E:R_{1:2}, which was in line with our first hypothesis. Additionally, the magnitude of $\Delta S_{mO_2\text{exercise}}$ was greater, and overall $S_{mO_2\text{min}}$ was lower during E:R_{1:2} compared to E:R_{1:4}. This supports our second hypothesis, that a greater decrease in muscle oxygenation would occur during E:R_{1:2} compared to E:R_{1:4}. Furthermore, T_c peaked

significantly higher during E:R_{1:4} than E:R_{1:2}, which aligns with our hypothesis that T_c would be greater at the end of E:R_{1:4} compared to E:R_{1:2}. Broadly, these results indicate that E:R_{1:4} increased mechanical power output compared to E:R_{1:2}, while both protocols had different effects on measures of internal physiological response.

Previous literature aligns with the results of the present study, showing that a smaller E:R (i.e. more

Table 1. Temperature responses during the repeat sprint cycling protocol ($n = 12$).

Condition	Baseline	Post-WU	Set 1			Set 2			Set 3			General Linear Mixed Model		
			Time	Condition	Interaction	Time	Condition	Interaction	Time	Condition	Interaction	Time	Condition	Interaction
Core temperature (°C)														
E:R _{1:4}	37.29 ± 0.27 (36.74–37.82)	37.66 ± 0.22 ^a (37.23–38)	37.74 ± 0.29 ^a (37.18–38.15)	38.12 ± 0.26 ^{abc} (37.71–38.64)	38.42 ± 0.36 ^{abcd} (37.87–39.08)	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.105
E:R _{1:2}	37.25 ± 0.27* (36.72–37.72)	37.57 ± 0.29 ^a (37.15–38.02)	37.65 ± 0.29 ^a (37.2–38.05)	37.89 ± 0.33 ^{abc} (37.42–38.44)	38.22 ± 0.35 ^{abcd} (37.75–38.78)									
Skin temperature (°C)														
E:R _{1:4}	35.62 ± 0.33 (35.20–36.26)	36.71 ± 0.11 ^a (36.55–36.83)	36.62 ± 0.26 ^a (36.19–37.03)	36.89 ± 0.51 ^a (35.92–37.45)	37.07 ± 0.53 ^{abc} (36.18–37.64)	0.780	0.780	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.999
E:R _{1:2}	35.6 ± 0.25 (35.27–36.10)	36.72 ± 0.19 ^a (36.36–37.01)	36.56 ± 0.26 ^a (36.07–37.00)	36.84 ± 0.26 ^a (36.41–37.29)	37.01 ± 0.34 ^{abc} (36.20–37.47)									

Note: Data are expressed as mean ± SD (range). E:R_{1:4}: Exercise-to-rest ratio of 1:4; E:R_{1:2}: Exercise-to-rest ratio of 1:2.

^aSignificant within group difference compared to baseline.

^bSignificant within group difference compared to post warm-up.

^cSignificant within group difference compared to Set 1.

^dSignificant within group difference compared to Set 2.

*Significant between group difference.

rest per unit of exercise) can increase mechanical power output during an RSH session, albeit performed in temperate conditions (Tong, Tao, Chow, Baker, & Jiao, 2021). Tong et al. (2021) performed three sets of 5 × 5-s “all-out” treadmill sprints at 3500 m simulated altitude, where E:Rs of 1:3 and 1:4 significantly reduced peak and mean running velocity to a similar extent to an E:R of 1:5 (Tong et al., 2021). Of note, the present study manipulated sprint duration as well as E:R, whereas Tong et al. kept sprint duration identical. The greater mechanical power output for smaller E:R seen in both Tong et al. (2021) and the present study indicates that it is the influence of E:R, not sprint duration, that induces a greater effect on power output. An increase in relative recovery time for each sprint allows for greater recovery of the anaerobic energy systems, which in turn, leads to a greater recruitment of fast twitch muscle fibres, augmenting mechanical power output (Bogdanis, Nevill, Lakomy, Graham, & Louis, 1996). Of note, exposure to a hypoxic stimulus promotes compensatory vasodilatation due to increased nitric oxide (NO) production (Casey & Joyner, 2012). Vasodilation induced by NO is exercise intensity dependent (Casey & Joyner, 2012), meaning that the “all-out” nature of RSH enhances this phenomenon compared to lower intensity exercise. Thus, the use of shorter sprints and an E:R that allows for greater recovery means that NO-induced vasodilation may be more effective in assisting responsiveness of the vascular bed and improving muscular blood perfusion (Casey & Joyner, 2012). In turn, it is likely that these responses could aid in promoting the peripheral adaptations to RSH (Faiss, Girard, & Millet, 2013). However, further research is required on the specific effects of E:R manipulation on vasodilation.

Despite lower mechanical power output, ΔS_{mO_2} exercise was significantly greater, and $S_{mO_{2min}}$ significantly lower in E:R_{1:2} compared to E:R_{1:4}. These results suggest that the relative contribution of aerobic energy metabolism compared to anaerobic glycolysis was greater in E:R_{1:2}, which in turn may explain why sprint power outputs were lower. Despite the lower mechanical power output, the decrease in the partial pressure of oxygen created by a reduction in muscle oxygenation is an important factor for inducing peripheral adaptation (Hoppeler, Vogt, Weibel, & Flück, 2003). Interestingly, by virtue of utilising double the number of sprints as E:R_{1:2}, E:R_{1:4} induced an increase in overall muscle oxygenation flux, which is also an important contributor to the effectiveness of RSH (Casey & Joyner, 2012; Daussin et al., 2008). As such, each protocol may have merit depending on if the target muscle oxygenation response relates to the reduction of partial

Table 2. Physiological and perceptual responses during the repeat sprint cycling protocol ($n = 12$).

Condition	Baseline	Post-WU	Set 1	Set 2	Set 3	General Linear Mixed Model		
						Condition	Time	Interaction
Pulse oxygen saturation (%)								
E:R _{1:4}	89 ± 3 (82–91)	89 ± 2 (86–92)	87 ± 3 (82–92)	87 ± 2 (83–90)	87 ± 1 (85–90)	0.931	<0.001	0.604
E:R _{1:2}	90 ± 2 (86–93)	88 ± 2 (85–90)	87 ± 4 (80–92)	87 ± 3 (82–93)	87 ± 2 ^a (84–90)			
Heart rate (bpm)								
E:R _{1:4}	92 ± 12 (72–116)	131 ± 13 ^a (110–149)	180 ± 6 ^{ab} (168–190)	183 ± 5 ^{ab} (172–193)	185 ± 8 ^{ab} (172–198)	0.581	<0.001	0.973
E:R _{1:2}	91 ± 13 (63–108)	134 ± 14 ^a (111–156)	180 ± 7 ^{ab} (166–189)	184 ± 5 ^{ab} (175–193)	186 ± 7 ^{ab} (175–197)			
Thermal sensation (0–8)								
E:R _{1:4}	5.5 ± 0.8 (4.5–6.5)	6.1 ± 0.9 ^a (4.5–7.0)	6.8 ± 0.8 ^a (5.0–8.0)	7.3 ± 0.6 ^{ab} (6.0–8.0)	7.6 ± 0.6 ^{abc} (6.0–8.0)	0.231	<0.001	0.085
E:R _{1:2}	5.8 ± 0.8 (4.0–7.0)	6.4 ± 0.7 ^a (5.0–7.0)	6.9 ± 0.7 ^a (5.5–8.0)	7.2 ± 0.8 ^{ab} (5.0–8.0)	7.4 ± 0.8 ^{ab} (5.0–8.0)			
Rating of perceived exertion (6–20)								
E:R _{1:4}	–	12 ± 3 (7–15)	16 ± 2 ^b (13–19)	18 ± 2 ^{bc} (15–20)	19 ± 1 ^{bc} (15–20)	0.019	<0.001	0.504
E:R _{1:2}	–	13 ± 2* (11–16)	17 ± 2 ^{ab} (15–20)	19 ± 2 ^{ab} (15–20)	19 ± 1 ^{abc} (15–20)			
Blood Lactate Concentration (mmol/L)								
E:R _{1:4}	1.0 ± 0.3 (0.6–1.7)	–	–	–	11.8 ± 3 ^a (6.1–15.6)	0.022	<0.001	0.027
E:R _{1:2}	1.2 ± 0.5 (0.5–1.9)	–	–	–	14.5 ± 3.2 ^{a†} (7.7–18)			

Note: Data are expressed as mean ± SD (range). E:R_{1:4}: Exercise-to-rest ratio of 1:4; E:R_{1:2}: Exercise-to-rest ratio of 1:2.

^aSignificant *within group* difference compared to baseline.

^bSignificant *within group* difference compared to post warm-up.

^cSignificant *within group* difference compared to Set 1.

^dSignificant *within group* difference compared to Set 2.

*Significant *between group* difference.

[†]Significant interaction compared to baseline and E:R_{1:4}.

pressure in the muscle or an increase in muscle oxygenation flux, each of which may lead to divergent physiological adaptations. Measures of muscle reoxygenation showed no differences between conditions for these variables ($\Delta S_mO_{2\text{recovery}}$ and $S_mO_{2\text{max}}$), potentially due to the similar inter-sprint recovery times employed in both protocols.

The present study has found different outcomes for different E:R protocols regarding muscle oxygenation. As such, it is difficult to interpret the results of this study towards demarcating appropriate comprehensive guidelines for the future use of combined heat and RSH sessions. In fact, this avenue has been explored recently with different training stimuli (high-intensity interval vs sprint interval training) imposed over 4 weeks in trained athletes, and physiological adaptations have also been difficult to disentangle and correlate to performance gains (Paquette, Bieuzen, & Billaut, 2021). It may be that if value is placed on the preservation of mechanical power output and muscle oxygenation flux, then E:R_{1:4} may be more suitable. However, if a greater level of deoxygenation across a session is sought, then E:R_{1:2} may be preferred. It is worth remarking that the use of NIRS in the heat for the measurement of S_mO_2 has attracted some criticism (Tew, Ruddock, & Saxton, 2010). Of note, Tew et al. (2010) found that *at rest*, NIRS derived measures of S_mO_2 were increased (from $69 \pm 8\%$ to $71 \pm 7\%$ and $73 \pm 6\%$) when investigating the difference between no heating and two localised skin-heating conditions (to 37 and 42°C). However, this research also found that there were no between group differences in S_mO_2 following the exercise. This suggests that increases in skin blood flow brought on by localised heating have negligible influence on NIRS measurement of S_mO_2 , when exercise-induced muscle blood flow is also increased.

The use of heat up to 40°C during RSH is not detrimental to mechanical power output (Dennis, Goods, Binnie, Girard, Wallman, Dawson, Billaut, et al., 2021), while in some cases, even potentially mitigating the performance impairment in acute hypoxia (Yamaguchi et al., 2019). In the current study, E:R_{1:4} generated a significantly higher peak T_c than E:R_{1:2}. The larger increases in T_c during E:R_{1:4} may be related to the increased metabolic heat production caused by a greater mechanical power output. Previous work from our laboratory found that there were no differences in T_c between three ambient temperature conditions (20, 35 and 40°C) in participants that completed a protocol identical to E:R_{1:2}, in hypoxic conditions (Dennis, Goods, Binnie, Girard, Wallman, Dawson, Billaut, et al., 2021). Given the current study found a significant difference in T_c between E:R_{1:2} and E:R_{1:4}, it appears that the nuances

of the protocol can play a more substantial role than air temperature in determining T_c during a typical RSH session. Of note, in neither condition did peak T_c reach the temperature that is recommended in literature as an acceptable dose for heat acclimation ($\sim 38.5^\circ$) (Racinais et al., 2015). Therefore, the prospect of adding heat to RSH requires further examination via future research into different protocols for combining these environmental parameters.

Although T_c was increased, RPE was lower in E:R_{1:4} compared to E:R_{1:2}, and thermal sensation was comparable between conditions. This indicates that E:R_{1:4} was better tolerated perceptually than E:R_{1:2} despite increasing T_c to a greater extent. This outcome may be related to the observed differences between E:R_{1:4} and E:R_{1:2} in power output across the session. The perception of increased exertion in E:R_{1:2} may have translated into an unwillingness to sustain repeated maximal efforts (Flouris & Schlader, 2015), thus contributing to the reduced power output. In turn, this would likely have an adverse impact on the efficacy of the implementation of the RSH protocol. Increases in T_{sk} across sets were not different between conditions, and this may be related to the well-established connection between elevated T_{sk} and increases in perceptual feelings of thermal sensation (Flouris & Schlader, 2015), which did not differ between conditions. Heart rate increased over time globally, which may have been induced by the higher thermoregulatory demand placed on the body as T_c increased above baseline levels (Casa, 1999). As such, thermoregulatory HR increases may have masked any independent effect of E:R on HR. Finally, the increase in BLa from baseline to the end of exercise was larger for E:R_{1:2} than in E:R_{1:4}. This observation may relate to the increased recovery time within sets (180-s vs 80-s) in E:R_{1:4}, which would allow for greater clearance of the by-products from anaerobic glycolysis from the blood. This may aid in explaining the differences in performance between conditions, with an increased BLa being previously correlated with increased muscle fatigue (Douris, 1993).

Limitations and additional considerations

This investigation explored the physiological responses of trained athletes (tier 4 in the Participant Classification Framework as classified by McKay et al., 2022) to a combination of heat and RSH. Of course, there is merit for future investigations to assess the outcomes on highly trained athletes (tier 3 or above), given the potential for different responses in such groups. Additionally, this investigation may have benefitted from the inclusion of experimental conditions that were performed in thermoneutral, laboratory conditions. Future

research featuring such an experimental design would allow elucidation of the environmental effects separate to the E:R manipulation. Further, future research might also investigate the effects of E:R manipulation on chronic adaptations to combined heat and RSH training, allowing the determination of the optimal protocol design for adaptation that coaches and practitioners can use to prescribe this type of training.

Conclusion

In summary, utilising an E:R of 1:4 (shorter sprints) during RSH in hot conditions augmented mechanical power output compared to 1:2 (longer sprints). These results occurred concurrent with a higher T_c (despite remaining below hyperthermic levels) and a reduction in RPE throughout the session, indicating a greater tolerance to the 1:4 protocol. However, both protocols had different effects on measures of muscle oxygenation, with E:R_{1:2} generating greater muscle oxygen extraction and E:R_{1:4} producing more muscle oxygenation flux, which are both important signals for peripheral adaptation. As such, the prescription of RSH combined with hot ambient temperatures may vary in E:R (1:2–1:4) and sprint length (5–10-s) depending on desired session outcomes.

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