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ORIGINAL ARTICLE

Cardiac autonomic response following high-intensity running work-to-rest interval manipulation

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Abstract
The cardiorespiratory, cardiac autonomic (via heart rate variability (HRV)) and plasma volume responses to varying sequences of high-intensity interval training (HIT) of consistent external work were investigated. Twelve moderately trained males underwent three HIT bouts and one control session. The HIT trials consisted of warm-up, followed by 12 min of 15 s, 30 s or 60 s work:relief HIT sequences at an exercise intensity of 100% of the individual velocity at $\dot{V}O_2$max ($v\dot{V}O_2$max), interspersed by relief intervals at 60% $\dot{V}O_2$max (work/relief ratio = 1). HRV was evaluated via the square root of the mean sum of the squared differences between R-R intervals (rMSSD) before, 1 h, 3 h and 24 h after the exercise. Plasma volume was assessed before, immediately after, and 3 h and 24 h after. There were no substantial between-trial differences in acute cardiorespiratory responses. The rMSSD values remained decreased 1 h after the exercise cessation in all exercise groups. The rMSSD subsequently increased between 1 h and 3 h after exercise, with the most pronounced change in the 15/15 group. There were no relationships between HRV and plasma volume. All HIT protocols resulted in similar cardiorespiratory responses with slightly varying post-exercise HRV responses, with the 30/30 protocol eliciting the least disruption to post-exercise HRV. These post-exercise HRV findings suggest that the 30/30 sequence may be the preferable HIT prescription when the between-training period is limited.

Keywords: *Cardiorespiratory, exercise, physiology, running*

Introduction

High-intensity interval training (HIT) is a well-established time-efficient method of enhancing aerobic fitness and improving athletic performance (Laursen & Jenkins, 2002). While practitioners can programme HIT in a variety of different ways, endurance improvements are notable when HIT sessions involve prolonged periods of work above 90% of maximal oxygen consumption (T@$\dot{V}O_2$max) (Billat et al., 2000). Such sessions allow athletes to accumulate substantial amounts of time (i.e. 5–7 min for sport games, 10 min for endurance athletes; Buchheit & Laursen, 2013a) at high exercise intensities close to $\dot{V}O_2$max (Helgerud et al., 2007; Nybo et al., 2010). The observed $\dot{V}O_2$max improvement following HIT interventions (Midgley, McNaughton, & Wilkinson, 2006) is likely due to its high strain on the cardiovascular and metabolic systems (Laursen, 2010), amongst a multitude of other factors (Seiler, Jøranson, Olesen, & Hetlelid, 2013).

Besides T@$\dot{V}O_2$max, up to nine factors can be manipulated within programming to adjust the training stimulus including the exercise/relief intensity and duration (Buchheit & Laursen, 2013a) or the mean load. The physiological effects of different manipulations of these HIT variables, however, are less known. It seems that short work intervals induce a lower cardiorespiratory and metabolic response compared to long work interval durations, and the acute response to the short work sequences might show similarities with a continuous exercise matched for the mean power (Tschakert et al., 2015). However, the specific effect of work interval duration with a fixed work-to-rest ratio in the same group of individuals has not been investigated thus far. Such knowledge would assist the prescription of HIT sessions to be placed

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appropriately within the "programming-puzzle" (Buchheit & Laursen, 2013b).

Heart rate variability (HRV) is one method that has been used to monitor the recovery process following strenuous exercise (Buchheit, 2014; Kiviniemi et al., 2014). HRV is a proxy measure of the autonomic nervous system status, which regulates a number of physiological processes (e.g. thermoregulation and delivery/removal of nutrients and waste products) that are critical during the recovery phase after exercise (Stanley, Peake, & Buchheit, 2013). Accordingly, HRV has been extensively used in the area of physical activity for the purpose of evaluating exercise load (Buchheit & Gindre, 2006), diagnosing non-functional overreaching and training adaptation (Plews, Laursen, Stanley, Kilding, & Buchheit, 2013) and maximising the balance between training and recovery (Botek, McKune, Krejci, Stejskal, & Gaba, 2014).

Accordingly, the aim of this study was to examine the acute cardiorespiratory response and post-exercise cardiac autonomic changes using HRV after varying sequences of HIT of equal external work. In line with other models of stress and recovery (Bannister, 1991), manipulation of the work and/or relief duration and its likely effect is of interest to sports practitioners and coaches looking to maximise the performance outcome from a training programme. Additionally, the possible impact of the exercise-induced changes in blood plasma volume on HRV was monitored within this study, since a relationship between these variables has been established (Buchheit, Laursen, Al Haddad, & Ahmaidi, 2009).

Methods

Participants

Twelve moderately trained males (age 22.8 ± 1.7 years, height 183.9 ± 7.8 cm, weight 77.0 ± 8.4 kg, body fat 9.9 ± 4.0%, 6.0 ± 2.5 h per week of self-reported exercise activity) participated in the study. None of the participants were clinically diagnosed with any chronic or acute cardiovascular, metabolic, respiratory, immunological or musculoskeletal system disorders. Those excluded from the study included smokers and those on medication or dietary supplements of various types. Prior to the participant's involvement, the local Ethics Committee of the University approved the experimental protocol and the investigation conformed to the principles outlined in the Declaration of Helsinki. All participants were fully informed about the study details and provided written informed consent.

Research design

The participants visited the laboratory on five separate occasions on a 1–2 week interval. During this time they first performed a maximal treadmill test, followed by three interval exercise sessions and one control session. The order of the exercise and control sessions was chosen at random. All sessions were performed in the morning and were conducted by the same researchers in a thermally controlled laboratory room.

Procedures

All the participants were informed about the experimental procedure during the first laboratory visit. During this time they also filled out a short questionnaire about physical activity, acute or chronic diseases and the use of dietary supplements/medication. The anthropometric assessment and body composition analysis then followed (Tanita BC418MA; Japan).

In order to determine their maximum aerobic power ($\dot{V}O_2max$) and the minimal running speed required to elicit $\dot{V}O_2max$ ($v\dot{V}O_2max$) (Table I), participants performed a graded exercise test (GXT). The GXT protocol began with 3 min at 8 $km.h^{-1}$; the speed then increased to 12 $km.h^{-1}$ and subsequently increased by 1 $km.h^{-1}$ every minute to voluntary exhaustion. Expired air was continuously monitored for an analysis of O_2 and CO_2 concentrations during the GXT by the use of a breath-by-breath system (ZAN600Ergo; Germany). Prior to each test, the gas analyser was calibrated in accordance with the manufacturer's instructions. The ambient conditions were automatically recorded by a ZAN 600 Ergo and maintained by air-conditioning at 21°C. It was declared that the participants had reached their $\dot{V}O_2max$, when at least two of the following criteria were met: (1) a plateau in the $\dot{V}O_2$ or increase less than 2.1 $ml.kg^{-1}.min^{-1}$ despite the increasing running load, (2) a final respiratory exchange ratio (RER) higher than 1.10; (3) an attainment of 95% of the age-predicted maximal heart rate. The $\dot{V}O_2max$ was based on the highest average O_2 consumption measured over a 30 s period. Gas-exchange measurements were also used to quantify the second ventilatory threshold (VT_2), which was defined as the point where ventilation increased disproportionately to oxygen consumption (Kenney, Wilmore, & Costill, 2012). Apart from the ventilatory curve, V-slope technique (Beaver, Wassermann, & Whipp, 1986) and RER were also considered the supplementary methods for the VT_2 determination. The final incremental test speeds reached at the end of the test ($v_{inc.t.}$) and at the $\dot{V}O_2max$ were calculated according to Kohn, Essén-Gustavsson and Myburgh

Table I. Pre-intervention graded exercise test results

Parameter	$N = 12$
$\dot{V}O_2$max (l.min^{-1})	4.4 ± 0.5
$\dot{V}O_2$max (ml.kg^{-1}.min^{-1})	57.2 ± 6.3
$V_{inc.t.}$ (km.h^{-1})	19.8 ± 1.4
v$\dot{V}O_2$max (km.h^{-1})	18.6 ± 1.4
HRmax (bpm)	196.7 ± 6.1
VT_2 (bpm)	176.3 ± 10.6
VT_2 (%)	84.7 ± 4.8
GXT duration (min:s)	11:49 ± 1:27
RPE	17.9 ± 1.5

Notes: $\dot{V}O_2$max – maximal oxygen consumption, $v_{inc.t.}$ – peak incremental test speed, v$\dot{V}O_2$max – minimal running speed required to elicit $\dot{V}O_2$max, HRmax – maximal heart rate, VT_2 – second ventilatory threshold in bpm or % of $\dot{V}O_2$max, GXT – graded exercise test, RPE – rating of perceived exertion. Values are expressed as mean ± standard deviation.

(2011). The heart rate was measured using a chest belt (Polar Electro; Finland).

HRV analysis

The last 5 min epochs of the 10 min supine rest ECG recording data were analysed using VarCorPF8 (DimeaGroup, Czech Republic). This diagnostic system enables a routine short-term HRV evaluation with respect to Task Force (1996) findings and recommendations. ECG was sampled at 1000 Hz and the accuracy of the measurements was 1 ms. The RR data was visually validated by the researcher prior to analysis, that is, assessment for stationary, ectopic, missing data or aberrant beats. Ectopic beats were excluded.

The assessment of the post-exercise cardiac autonomic regulation focused on the vagal-related parameter rMSSD (the square root of the mean sum of the squared differences between RR intervals; ms), since HRV has not been demonstrated as an appropriate method of assessing cardiac sympathetic modulation (Task Force, 1996). The changes in the cardiac parasympathetic activity following exercise show the time course of restoration of homeostasis, which is believed to be training strain dependent (Buchheit, 2014; Stanley et al., 2013). HRV analysis was also limited to rMSSD since it has a much greater reliability than other spectral indices (Al Haddad, Laursen, Chollet, Ahmaidi, & Buchheit, 2011), particularly during 'free-running' ambulatory conditions (Penttila et al., 2001).

Exercise intervention

The second, third, fourth and fifth visits to the laboratory consisted of the interval exercise interventions (or rest in case of the control). Participants always came to the laboratory between 7 and 9 am, after a night of fasting (i.e. no breakfast was consumed). Participants performed the HIT intervention which consisted of the following parts: (1) 8 min of a warm-up at the speed of 50% v$\dot{V}O_2$max; (2) interval exercise: total duration 12 min, work/relief ratio = 1, work intensity 100% v$\dot{V}O_2$max, relief intensity 60% v$\dot{V}O_2$max, work and relief duration 15 s, 30 s, or 60 s; (3) cool-down: 3 min at 5 km.h^{-1}. All interval exercise interventions were identical to the total duration, work/relief ratio, relative work and relief intensity. The between-session difference was the work:relief interval duration (15 s vs. 30 s vs. 60 s). Ventilatory parameters and heart rate were monitored during the exercise. Training load for each HIT intervention was also assessed via the heart rate-based Training Stress Score (hrTSS) using Trainingpeaks.com; in order to describe the potential between-trial differences hrTSS was calculated for all HIT trials as follows:

$$\text{hrTSS} = T\ x\ \frac{\text{HRex}\ \times \text{IF}}{\text{HR}_{\text{VT2}}\ \times 3600}\ \times 100,$$

where T is the duration of the exercise, HRex is the average HR during exercise, IF is the intensity factor calculated from the ratio of HRex to HR_{VT2} and HR_{VT2} is the heart rate at the second ventilatory threshold. For the Control trial, data collection was identical to the HIT trial, but the exercise intervention was replaced by 23 min of rest.

Recovery monitoring

The participants remained resting in the laboratory for 3 h post testing to assess the recovery process. HRV was measured in the supine position before the exercise, 1 h, 3 h and 24 h after the exercise intervention. Experienced medical staff drew the blood samples from the antecubital vein with the participant in a sitting position. Venous blood was collected four times (prior to exercise, immediately after exercise (POST), 3 h and 24 h after exercise cessation) from all the individuals during all the exercise (and control) sessions. The period between 3 h and 24 h measurement was not personally controlled. However, participants were instructed to avoid alcohol drinking, any strenuous physical activity and keep common sleep duration.

Fluid and food ingestion during each testing session was standardised. Accordingly, each participant was provided with carbohydrate-rich, low-fat food (plain sponge biscuits 240 g; 75.0 g CHO,

11.0 g protein and 4.9 g fat per 100 g; 390 kcal per 100 g) and 1.5 l of sweet mineral water (21.4 kcal per 100 ml).

Venous blood sampling and blood analysis

The blood sample was allowed to clot for 30 min and subsequently centrifuged at 2000 G for 10 minutes in order to separate the serum. The blood serum was consequently divided into three 1 ml aliquots, which were frozen at −70°C until analysis. The S-Monovette® system (Sarstedt, Germany) was used for blood sample collection. Blood samples were analysed for the total circulating protein concentration in serum (TCP; $g.l^{-1}$).

Statistical analysis

Data are presented as means and standard deviations (SD). All HRV and biochemical data were log-transformed prior to analysis. The standardised changes in mean (Effect size, ES) and 90% confidence limits (90% CL) were calculated for the between groups as well as for the between time points changes. Threshold values for ES statistics were <0.2 (*trivial*), ≥0.2 (*small*), ≥0.6 (*moderate*), ≥1.2 (*large*), ≥2.0 (*very large*), ≥4.0 (*nearly perfect*). The exact probabilities were expressed and magnitude of the difference was also evaluated qualitatively as follows: 25–75% *possibly*, 75–95% *likely*, 95–99% *very likely*, >99% *almost certain* (Batterham & Hopkins, 2005; Hopkins, Marshall, Batterham, & Hanin, 2009). The smallest worthwhile change/difference is considered 0.2 of the between-individual standard deviation. If the chance of higher or lower differences was >5%, then the true difference was assessed as *unclear*. Statistical analyses were performed using the statistical spreadsheet (Hopkins, 2006).

Pearson's product–moment correlation analysis was used to compare the association between the ln rMSSD and ln TCP values. To do this we used within-subject linear modelling which expresses the percentage change between PRE and after exercise (immediately POST, 1 h, 3 h, and 24 h) values. The following criteria were used to interpret the magnitude of the correlation (*r*) between ln rMSSD and ln TCP: <0.1 *trivial*, ≥0.1 *small*, ≥0.3 *moderate*, ≥0.5 *large*, ≥0.7 *very large*, and ≥0.9 *nearly perfect*. If the 90% CI overlapped small positive and negative values, the magnitude of the correlation was considered *unclear*; otherwise, the magnitude of the correlation was deemed to be the observed magnitude (Hopkins et al., 2009).

Results

Aerobic fitness assessment and exercise intervention characteristics

The pre-intervention graded exercise test results are shown in Table I. All participants successfully completed the HIT trials as prescribed. The standardised differences of the cardiorespiratory response to the different HIT interventions are presented in Table II. The physiological responses (HR, $\dot{V}O_2$, $\dot{V}CO_2$, RER) can be viewed in Figure 1.

HRV measures

The pre-exercise between-group differences of the ln rMSSD were *trivial* (15/15 vs. 30/30; 15/15 vs. 60/60; 30/30 vs. 60/60; ES ± 90% CL: −0.10 ± 0.48; −0.18 ± 0.53; −0.08 ± 0.59, respectively) or *small* (15/15 vs. Control; 30/30 vs. Control; 60/60 vs. Control; +0.27 ± 0.43; +0.28 ± 0.35; +0.33 ± 0.46, respectively). All standardised changes in the mean ln rMSSD during the time course of recovery are shown in Figure 2. The ln rMSSD values remained decreased 1 h after the exercise cessation in all exercise groups. The ln rMSSD subsequently increased between 1 h and 3 h after exercise in all HIT trials, with the most pronounced change observed in the 15/15 group (+1.28 ± 0.69; 99/1/0%). It was *unclear* if ln rMSSD changed from baseline 3 h after HIT in the 15/15 group (+0.13 ± 0.34; 36/58/6%) or 24 h after exercise (−0.17 ± 0.38; 6/49/45%). There was a *possibly small* increase in ln rMSSD 3 h after exercise for both 30/30 (+0.21 ± 0.26; 52/47/1%) and 60/60 (+0.30 ± 0.35; 68/30/1%) trials in relation to the PRE-exercise level. In the 15/15 trial (−0.17 ± 0.29; 6/49/45%) and 60/60 trial (+0.19 ± 0.53; 48/41/11%), there were *unclear* changes in ln rMSSD after 24 h of HIT. Conversely, there was a *possibly small* increase in ln rMSSD (+0.31 ± 0.28; 74/25/0%) 24 h after exercise in the 30/30 trial. The ln rMSSD values in the Control trial did not reveal a meaningful change over time.

Total circulating protein (TCP)

The PRE-exercise between-group differences of the ln TCP were *likely trivial* (60/60 vs. Control; −0.07 ± 0.26), *possibly* and *likely small* (15/15 vs. 30/30; 30/30 vs. 60/60; 30/30 vs. Control; −0.39 ± 0.29; −0.35 ± 0.41; −0.33 ± 0.25, respectively) or *almost certainly moderate* (15/15 vs. 60/60; 15/15 vs. Control; −0.75 ± 0.26; −0.85 ± 0.23, respectively). All standardised changes in mean ln TCP during the time course of recovery can be viewed in Figure 2. Immediately POST exercise intervention,

Table II. The between-trial differences (Warm-up and cool-down excluded)

$N = 12$	15/15 mean ± SD	30/30 mean ± SD	60/60 mean ± SD	15/15 vs. 30/30	15/15 vs. 60/60	30/30 vs. 60/60
Mean HR (bpm)	182.5 ± 6.9	179.6 ± 5.6	180.8 ± 7.2	−0.38 (−0.77; 0.01) Likely small	−0.23 (−0.52; 0.06) Possibly small	0.15 (−0.16; 0.45) Possibly trivial
Maximal HR (bpm)	191.4 ± 8.1	188.3 ± 5.7	191.3 ± 7.8	−0.35 (−0.65; −0.04) Likely small	−0.02 (−0.30; 0.26) Unclear	0.33 (0.06; 0.60) Likely small
Mean $\dot{V}O_2$ (ml.kg. min^{-1})	50.2 ± 4.2	49.8 ± 3.7	49.5 ± 3.6	−0.16 (−0.44; 0.11) Possibly trivial	−0.13 (−0.32; 0.06) Possibly trivial	−0.07 (−0.21; 0.34) Unclear
Mean $\dot{V}CO_2$ (ml.kg. min^{-1})	45.9 ± 4.3	46.8 ± 5.0	46.5 ± 4.6	0.11 (−0.76; 0.99) Unclear	0.11 (−0.13; 0.35) Possibly trivial	0.06 (−0.85; 0.97) Unclear
RER	0.92 ± 0.03	0.94 ± 0.02	0.94 ± 0.03	0.67 (−0.13; 1.47) Likely moderate	0.63 (0.01; 1.26) Likely moderate	0.20 (−0.55; 0.95) Unclear
RPE	16.8 ± 1.4	15.8 ± 1.8	16.7 ± 0.9	−0.65 (−1.15; −0.16) Likely moderate	−0.03 (−0.46; 0.39) Unclear	0.62 (0.08; 1.16) Likely moderate
hrTSS	21.6 ± 2.6	21.0 ± 2.4	21.2 ± 2.8	−0.24 (−0.47; 0.00) Possibly small	−0.13 (−0.30; 0.04) Possibly trivial	0.10 (−0.08; 0.28) Likely trivial

Notes: HR – heart rate, $\dot{V}O_2$ – oxygen consumption, $\dot{V}CO_2$ – carbon dioxide production, RER – respiratory exchange ratio, RPE – rating of perceived exertion, hrTSS – heart rate Training Stress Score, 15/15–15 s at 100% v$\dot{V}O_2$max and 15 s at 60% v$\dot{V}O_2$max, 30/30–30 s at 100% v$\dot{V}O_2$max and 30 s at 60% v$\dot{V}O_2$max, 60/60–60 s at 100% v$\dot{V}O_2$max and 60 s at 60% v$\dot{V}O_2$max. The between-trial differences are expressed as standardised (Cohen) difference of the mean (90% confidence limits) and rating of the difference.

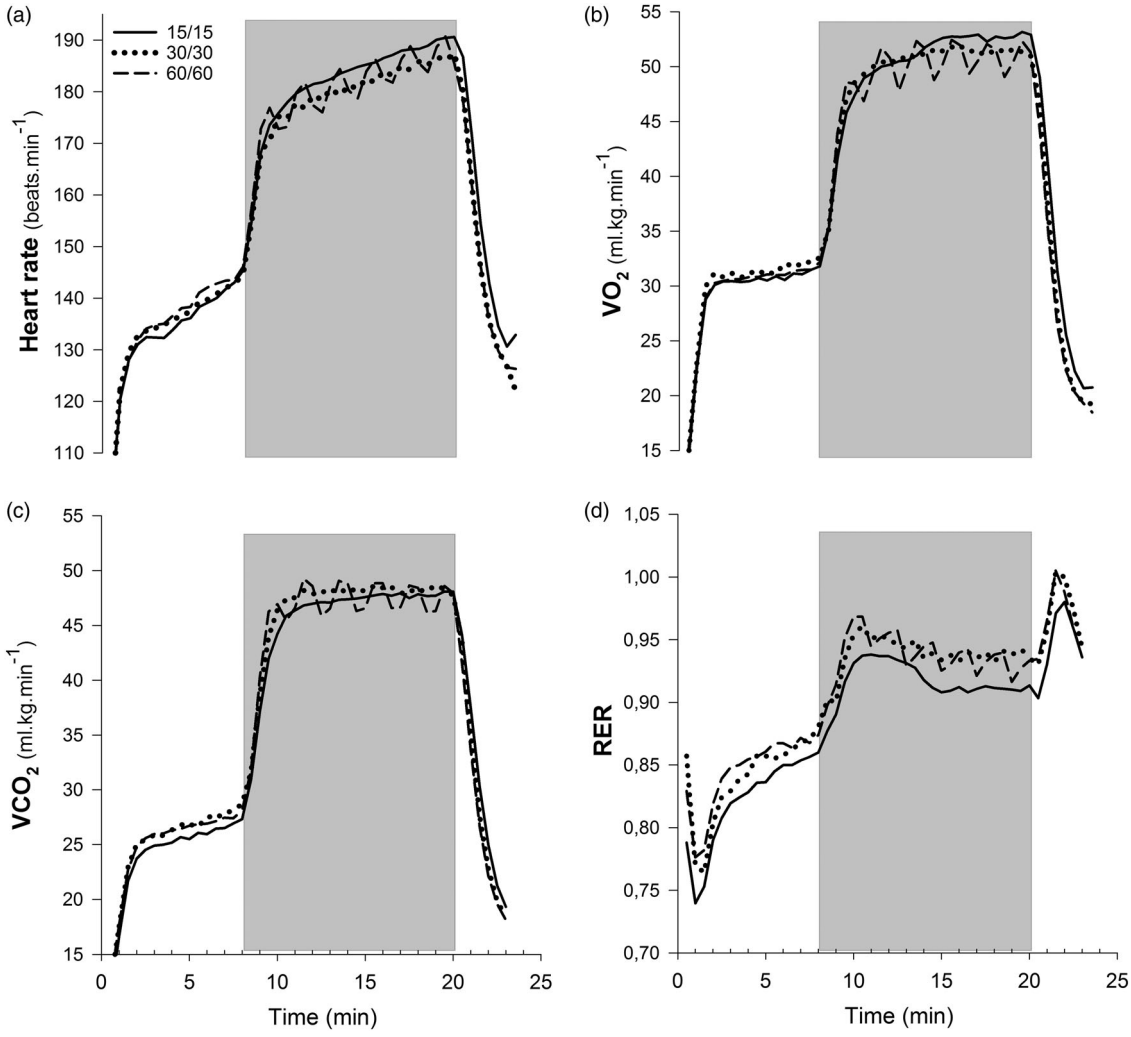

Figure 1. (a–d). Mean physiological responses during the three high-intensity interval training sessions (15/15, 30/30 and 60/60) for heart rate (a), oxygen uptake ($\dot{V}O_2$) (b), carbon dioxide production ($\dot{V}CO_2$) (c) and respiratory exchange ratio (RER) (d). 296 × 418 mm (300 × 300 DPI).

there was an *almost certainly very large* (15/15 and 60/60; +2.02 ± 0.31, +2.54 ± 0.22, respectively) and *large* (30/30; +1.70 ± 0.27) increase in ln TCP for all exercise groups. Subsequently, an *almost certain large* decrease was observed in all exercise groups between immediately POST and 3 h after the

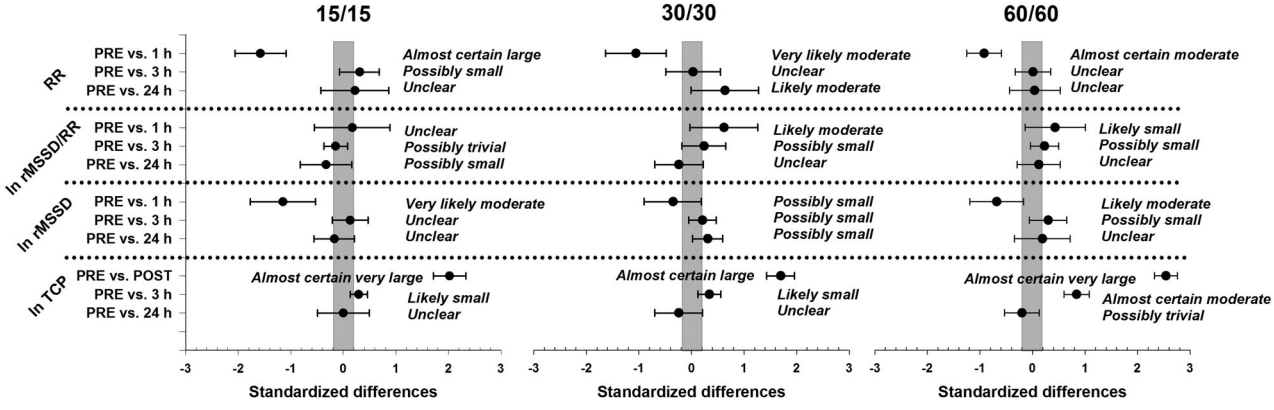

Figure 2. Standardised change of the mean and the rating of the difference for the RR, ln rMSSD/RR, ln rMSSD and TCP values during the 15/15, 30/30 and 60/60 HIT sessions. 296 × 209 mm (300 × 300 DPI).

intervention (15/15; 30/30; 60/60; −1.72 ± 0.33; −1.35 ± 0.23; −1.70 ± 0.23; respectively). There was a *possibly trivial* change in ln TCP 24 h after the exercise compared to the PRE-exercise level in the 60/60 trial while these changes in other trials (15/15, 30/30 and Control) were *unclear*.

Correlations between ln rMSSD and Total circulating protein

Most correlations were deemed *unclear*. The only exception was the 24 h vs. PRE comparison, where the correlation was *large* ($r = 0.52$).

Discussion

Our investigation brought several major findings. First, the various HIT protocols of short intervals applied over fixed work/relief ratios, all common prescriptions used by coaches, produced comparable mean cardiorespiratory responses, even if the pattern was shown to be different (Table II and Figure 1). These similar mean cardiorespiratory responses shown across all HIT exercise interventions confirm a commonly heard saying within the training literature, that there is "more than one way to skin the cat" when it comes to exercise programming and prescription (Buchheit & Laursen, 2013a). Second, the 30/30 exercise intervention resulted in the smallest disruption of the post-exercise cardiac autonomic modulation compared with the shorter (15 s) or longer (60 s) work/relief intervals. As such, coaches and sports practitioners wishing to target specific physiological adaptations from HIT (e.g. maximal aerobic capacity) may be able to vary the type of HIT interval whilst achieving a similar training response.

Exercise interventions

The HIT protocols used in this study were built to the recommendations of Buchheit and Laursen (2013a, 2013b). These authors, and others, suggest that exercise intensity should reach at least 90% of $\dot{V}O_2$max for several minutes to elicit the maximal cardiovascular and peripheral adaptations needed to increase $\dot{V}O_2$max effectively, assuming that is the goal of the session. Indeed, a work intensity of 95% v$\dot{V}O_2$max with varying relief intervals allows $\dot{V}O_2$max to be attained for prolonged periods of time (Midgley & McNaughton, 2006). Accordingly, v$\dot{V}O_2$max was assessed via the graded exercise test prior to the HIT prescription to allow for the individual relative exercise intensity of each HIT session to be calibrated appropriately for all participants. The only variable of manipulation between the HIT sessions was the duration of the work performed during each interval (with the ratio and total work performed remaining identical throughout). Indeed, the interaction between work duration, intensity and recovery is considered important for inducing the desirable physiological adaptations of those training sessions (Seiler et al., 2013). The design of the HIT sessions implemented here allowed for ~6 min of T@$\dot{V}O_2$max; duration recommended as appropriate for conditioning in team sports and for maintenance during unloading or recovery periods in the programmes of endurance athletes (Buchheit & Laursen, 2013a).

The mean cardiorespiratory response to the HIT trials over the entire 12 min of intermittent exercise (Table II and Figure 1) showed mostly *trivial* or *small* differences between the 15/15, 30/30 and 60/60 HIT protocols. The average HR and $\dot{V}O_2$ during all 12 min interval exercise protocols exceeded VT_2 (Tables I and II), with higher relative HR (> 91% of HRmax) and $\dot{V}O_2$ values (> 86% of $\dot{V}O_2$max). Since the exercise intensity during recovery was quite high (60% v$\dot{V}O_2$max), it is not surprising that the mean HR in the 15/15 trial was the highest. However, these differences were only *small*. Similarly, the differences in maximal HR were *small* at the most, which are in contrast to Tschakert et al. (2015). The identical exercise work, relief, and mean intensity and quite short duration of all HIT protocols are believed to be the reasons for this. The *likely moderate* difference was observed between 15/15 vs. 30/30 and 60/60 trials for the respiratory exchange ratio (RER), with the gradual decrease of the RER in all three HIT trials (Figure 1d), which might be explained by an inhibition of glycolysis (Gaitanos, Williams, Boobis, & Brooks, 1993).

The heart rate Training Stress Score (hrTSS), predicted the training load dose-response nature, was also calculated to quantify the predicted dose-response nature of the HIT trials. Based on the hrTSS, there were *trivial* or *small* differences between the HIT trials. This method of analysis, however, has limitations, particularly with very short duration maximal exercise conditions, where the HR does not precisely correspond to the high exercise intensity (Hayes & Quinn, 2009).

Post-exercise HRV changes

Notwithstanding methodological limitations surrounding the assessment of HRV (Cipryan & Litschmannova, 2013), the monitoring of the post-exercise HRV can be useful for evaluating the degree of strain

associated with an acute exercise session (Buchheit, 2014). As mean and peak HR are influenced by sympathetic nervous system activation (i.e. higher mean and maximal HRs that require greater sympathetic nervous system activity, and thereby lower post-exercise HRV (Buchheit, Laursen, & Ahmaidi, 2007)), we evaluated the relationship between HR and vagal-related HRV variable ln rMSSD. In this study, the 15/15 HIT was associated with slightly higher HRs (*likely small* increase 15/15 vs. 30/30, and *possibly small* increase 15/15 vs. 60/60). It was therefore not surprising that ln rMSSD was at its lowest after 15/15 HIT (*very likely* decrease) compared with 30/30 (*possible* decrease) and 60/60 (*likely* decrease) HIT sessions 1 h post exercise. However, on average this difference was only 2 beats (15/15 to 30/30) and 1 beat (15/15 to 60/60), which are unlikely to be considered meaningful by coaches and sports practitioners within a practical setting. This shows the potential sensitivity of ln rMSSD in assessing the overall training stress of a session compared with mean and maximal heart rate alone.

A decrease in ln rMSSD over the first 60 min following high-intensity exercise was the expected response (Stanley et al., 2013). The greatest decrease of the ln rMSSD 1 h after HIT, compared to the pre-exercise level, was shown in the 15/15 trial (ES ± 90% CL; −1.15 ± 0.62; 0/1/99%). Interestingly, participants evaluated this HIT protocol as most challenging on the RPE scale (mean ± SD: 16.8 ± 1.4). In contrast, the 30/30 HIT protocol resulted in the least decrease to ln rMSSD (−0.35 ± 0.55; 5/26/69) and was least strenuous in terms of the RPE values (15.8 ± 1.8). The between-group differences of the RPE and cardiorespiratory variables were, however, assessed as *likely small* (mean HR, RPE) or *possibly trivial* (mean $\dot{V}O_2$). As such HRV may be a sensitive and objective measure of subsequent training load following HIT. These data suggest that HRV is potentially a more sensitive tool for evaluating training load compared with other physiological variables (e.g. HR and $\dot{V}O_2$) (Buchheit & Laursen, 2013a).

The influence of heart rate on HRV has been previously reported (Sacha, 2014). This should be, therefore, considered when the HRV results are presented. The between-trial RR interval and ln rMSSD/RR differences in all time points (PRE, 1 h, 3 h and 24 h post-exercise) were, however, revealed *unclear* or *small* (Figure 2).

Changes in total circulating protein

It has been shown that HRV indices are sensitive to changes in blood plasma volume (Buchheit et al., 2009) and therefore may be of interest when assessing changes in HRV after exercise. Changes in blood plasma volume after HIT would be expected due, in part, to fluid shifts due to sweating and plasma shifts between the blood and interstitial fluid space. In this study, we chose to measure total circulating protein (TCP) to assess possible changes in exercise-induced blood plasma volume between the different HIT sequences (Convertino, 1991). As capillary membranes are relatively permeable to plasma electrolytes, increases in TCP represent osmotic compartmentalisation of water in the vascular space, where 1 g of circulating plasma protein binds 14–15ml of water (Scatchard, Batchelder, & Brown, 1944). The present results showed that TCP *almost certainly* increased immediately after exercise termination in all HIT trials. These changes were more evident for all HIT trials compared with the Control trial. Interestingly, the smallest change in both ln rMSSD and ln TCP (PRE vs. 1 h / POST exercise) was observed in the 30/30 trial. Accordingly, the *very large* increases of the TCP may be related to the *moderate* decreases shown in ln rMSSD for the 15/15 and 60/60 trials, while "only" the *large* increase of the TCP was associated with the *small* decrease of the ln rMSSD in the 30/30 trial. As a result, the magnitude of the change in HRV appears somewhat related to the change in TCP; the larger the decrease in ln rMSSD the greater the increase in TCP.

Despite the abovementioned findings between ln rMSSD and ln TCP, we showed no direct relationship between the two variables when expressed as an individual relative change (% difference from the individual mean). These finding are in contrast to Buchheit et al. (2009), who observed a very large ($r = 0.85$) relationship between the relative change in ln rMSSD and blood plasma volume. There may be several reasons for this difference. First, Buchheit et al. (2009) assessed blood plasma volume expansion via changes in haematocrit and haemoglobin measures, whereas we inferred it via assessment of ln TCP. Second, the time course of assessment in the study by Buchheit et al. (2009) was over 2 days, compared with the shorter time course used in our study (24 h). Thus, individual factors such as plasma epinephrine (Perini et al. 1989), blood lactate (Buchheit, Al Haddad, Mendez-Villanueva, Quod, & Bourton, 2011), blood acidosis (Buchheit et al., 2007) and arterial oxygenation (Ba, Delliaux, Bregeon, Levy, & Jammes, 2009) may still affect ln rMSSD levels, potentially clouding possible associations between variables.

Conclusions

The main aim of this study was to further understand the cardiorespiratory stress of various types of HIT where the workload was identical. We found that three different short-interval HIT protocols of fixed work/relief ratios caused similar mean cardiorespiratory responses and slightly different HRV responses, with the 30/30 HIT protocol eliciting the least post-exercise HRV disruption, compared with the shorter (15/15) and longer (60/60) HIT designs. In this regard, post-exercise HRV findings from the present study suggest that the 30/30 sequence might be the preferable HIT prescription when a between-training recovery period needs to be reduced due to multiple-phase training programming.

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Disclosure statement

No potential conflict of interest was reported by the authors.

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