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ORIGINAL ARTICLE

The effect of functional overreaching on parameters of autonomic heart rate regulation

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Abstract
Purpose Correlations between fatigue-induced changes in performance and maximal rate of HR increase (rHRI) may be affected by differing assessment workloads. This study evaluated the effect of assessing rHRI at different workloads on performance tracking, and compared this with HR variability (HRV) and HR recovery (HRR).
Methods Performance [5-min cycling time trial (5TT)], rHRI (at multiple workloads), HRV and HRR were assessed in 12 male cyclists following 1 week of light training (LT), 2 weeks of heavy training (HT) and a 10-day taper (T).
Results 5TT very likely decreased after HT (effect size ± 90% confidence interval = −0.75 ± 0.41), and almost certainly increased after *T* (1.15 ± 0.48). rHRI at 200 W likely increased at HT (0.70 ± 0.60), and then likely decreased at *T* (−0.50 ± 0.70). rHRI at 120 and 160 W was unchanged. Pre-exercise HR during rHRI assessments at 120 W and 160 W likely decreased after HT (≤−0.39 ± 0.14), and correlations between these changes and rHRI were large to very large ($r = -0.67 \pm 0.31$ and $r = -0.78 \pm 0.23$). When controlling for pre-exercise HR, rHRI at 120 W very likely slowed after HT (−0.72 ± 0.44), and was moderately correlated with 5TT ($r = 0.35 \pm 0.32$). RMSSD likely increased at HT (0.75 ± 0.49) and likely decreased at *T* (−0.49 ± 0.49). HRR following 5TT likely increased at HT (0.84 ± 0.31) and then likely decreased at *T* (−0.81 ± 0.35).
Conclusions When controlling for pre-exercise HR, rHRI assessment at 120 W most sensitively tracked performance. Increased RMSSD following HT indicated heightened parasympathetic modulation in fatigued athletes. HRR was only sensitive to changes in training status when assessed after maximal exercise, which may limit its practical applicability.

Keywords Heart rate · Overreaching · Athletic performance · Autonomic nervous system

Abbreviations

ANS	Autonomic nervous system
DALDA	Daily analysis of life demands for athletes
ES	Effect size
FOR	Functional overreaching
HR	Heart rate
HRend	Heart rate at the end of exercise
HRR	Heart rate recovery
HRV	Heart rate variability
HT14	Heavy training
Ln RMSSD	Natural logarithm of the root-mean-square difference of successive normal R-R intervals
LT7	Light training
NFOR	Non-functional overreaching
OT	Overtraining
rHRI	Maximal rate of heart rate increase
$rHRI_{120\ W}$	Maximal rate of heart rate increase assessed at 120 watts
$rHRI_{160\ W}$	Maximal rate of heart rate increase assessed at 160 watts

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$rHRI_{200\ W}$	Maximal rate of heart rate increase assessed at 200 watts
$rHRI_{120–200\ W}$	Maximal Rate of Heart Rate Increase assessed during transition from 120 W to 200 watts
SD	Standard deviation
TRIMP	Training impulse
T10	Tapering
W	Watts
5TT	Five-minute time trial
60TT	Sixty-minute time trial

Introduction

The regulation of cardiovascular control by the autonomic nervous system (ANS) has been utilised to detect the accumulation of training-induced fatigue that may occur during periods of high training stress without adequate recovery (Borresen and Lambert 2008; Buchheit 2014). Training periods with these characteristics may result in functional overreaching (FOR), non-functional overreaching (NFOR) or even overtraining (OT), all characterised by decrements in exercise performance (Kellmann 2010; Meeusen et al. 2013). A simple non-invasive marker capable of detecting FOR may assist in optimising athletic performance at important time-points by providing appropriate interpretation of short-term performance decrements representative of this particular training state. Information provided by such a marker could then be used to initiate a period of recovery that may ultimately facilitate supercompensatory performance improvements before fatigue accumulation gives rise to NFOR or OT (Buchheit 2014; Meeusen et al. 2013). The latter training states resulting in long-term performance decrements without supercompensatory performance improvement (Meeusen et al. 2013).

Markers of autonomic HR regulation and athletic training status include resting HR, submaximal HR, maximum HR, HR variability (HRV) and HR Recovery (HRR); however, the potential for HR kinetics at the onset of exercise has also been recently investigated in this context. Initial studies found that the maximal rate of HR increase (rHRI) during the rest-to-exercise transition at the onset of submaximal cycling exercise was slowed in both acutely fatigued (Thomson et al. 2015b) and overreached states (Nelson et al. 2014), and that this slowing of rHRI was positively correlated with fatigue-induced performance reductions.

In a subsequent study to compare the sensitivity of rHRI for tracking performance changes when determined through cycling and running exercise, the exercise intensity at which rHRI was assessed was shown to influence its ability to track performance changes (Bellenger et al. 2015). In that study, cycling rHRI was assessed at 100 W as per Nelson et al. (2014), but despite a small slowing in this parameter following overload training, changes in rHRI did not track fatigue-induced performance decrements as sensitively as demonstrated by Nelson et al. (2014). However, the participants in the latter study (Bellenger et al. 2015) appeared better conditioned than those of Nelson et al. (2014) (~5% greater peak oxygen uptake and time-trial performance) and, given that participants demonstrating superior aerobic fitness require a greater exercise intensity to elicit higher degrees of parasympathetic withdrawal (Tulppo et al. 1998), an intensity of 100 W may not have provided sufficient stress to elicit similar degrees of parasympathetic withdrawal in comparison to that achieved by Nelson et al. (2014). Thus, the differing levels of parasympathetic and sympathetic modulation during rHRI assessment at 100 W in these studies may have affected the sensitivity of this parameter for tracking performance changes. In support of this notion, Bellenger et al. (2015) also found that fatigue-induced reductions in rHRI assessed during running exercise, which elicited a greater steady-state HR and greater change in rest-exercise HR (indicating a greater degree of sympathetic modulation), tracked performance reductions better than the aforementioned changes in cycling rHRI. Consequently, the optimal intensity for determining cycling rHRI to most sensitively track performance changes may be higher than the 100 W used in studies to date.

This study therefore evaluated the use of different exercise intensities for determining cycling rHRI to most sensitively track performance changes, and to explore the physiological mechanisms that allow changes in rHRI to track performance changes. With regard to the latter, this study sought to determine whether the processes that allow rHRI to track performance changes are related more to parasympathetic or sympathetic HR modulation. The novel measure of rHRI was also compared to the more established measures of HRV and HRR for detecting changes in training status.

Methods

Participants

Twelve male cyclists/triathletes were recruited from the Adelaide metropolitan area in South Australia. The University of South Australia's Human Research Ethics Committee approved the study, and volunteers provided written informed consent prior to participating.

Experimental overview

Pre-study familiarisation allowed participants to be habituated with study requirements and testing procedures, and determine their peak HR during two cycling performance tests; a five-minute time trial (5TT) and a 60-min time trial (60TT). Assessments of rHRI, HRR, 5TT and 60TT then occurred after seven days of light training (LT7; baseline), 14 days of heavy training (HT14; overreached state) and 10 days of tapering (T10; recovered/adapted state), on the day after the last completed training session. The effect of training on daily measures of HRV and training tolerance were also investigated.

rHRI assessment and calculation

To determine the effect of different workloads on rHRI and its ability to track performance changes, rHRI was assessed during 5 min of cycling at light (120 W; $rHRI_{120\ W}$), moderate (160 W; $rHRI_{160\ W}$) and heavy intensities (200 W; $rHRI_{200\ W}$) (Norton et al. 2010) on an electronically braked cycle ergometer (Lode Excalibur Sport, Lode BV, Groningen, Netherlands).

Contributions of parasympathetic and sympathetic HR modulation to rHRI were explored with a two-stage test based on research from White and Raven (2014), who showed that exercise-induced increases in HR to ~100–120 bpm were primarily the result of parasympathetic modulation (~3.5:1.5 parasympathetic to sympathetic ratio), with subsequent increases becoming more reliant on sympathetic modulation. Therefore, as $rHRI_{120\ W}$ was designed to elicit a steady-state HR of ~65% of peak HR (or ~100–120 bpm), it would primarily reflect parasympathetic withdrawal (Robinson et al. 1966; Rowell and O'Leary 1990; Victor et al. 1987; Warner and Cox 1964; White and Raven 2014). Upon completion of $rHRI_{120\ W}$, the power output was increased to 200 W (designed to elicit a steady-state HR of ~85% of peak HR, or ~150 bpm; $rHRI_{120–200\ W}$), and the subsequent increase in HR would primarily reflect sympathetic activation (Robinson et al. 1966; Rowell and O'Leary 1990; Victor et al. 1987; Warner and Cox 1964; White and Raven 2014).

Ordering of rHRI assessments was randomised at baseline, and held constant at subsequent visits. Exercise onset occurred at random to avoid an anticipatory rise in HR (Krogh and Lindhard 1913). HR data were recorded in beat-to-beat interval mode (RR intervals) for maximal HR curve resolution during rHRI testing using a HR monitor (RS800CX, Polar Electro Oy, Kempele, Finland). A 5-component sigmoidal curve was fit (Eq. 1) to HR data recorded during the 30 s preceding exercise onset (or preceding the change in workload when determining $rHRI_{120–200\ W}$), and throughout the subsequent 5 min of steady-state exercise. rHRI (bpm/sec) was the first derivative maximum of this curve (Eq. 2) obtained using the Solver function in Excel (Microsoft Corporation, NY, USA). Pre-exercise HR (mean HR during the 30 s prior to commencing exercise) and steady-state HR (mean HR during the final 60 s of exercise) were also calculated.

$$\hat{y} = a + \frac{b}{1 + f_x \cdot e^{c(d-x')} + \left(1 - f_x\right) \cdot e^{e(d-x')}}, \tag{1}$$

where

$$f_x = \frac{1}{1 + e^{-\bar{C}_f(d-x')}}$$

defines a logistic weighting function varying smoothly between 0 and 1, centred about d so long as c and e are of the same sign, and where the mean curvature of f is given by

$$\bar{C} = \frac{2 \cdot c \cdot d}{c + d},$$

where a lower HR plateau, b range of HR response, c curvature parameter, d time at which half of the range of HR response was attained, e curvature parameter

$$\boldsymbol{x} = \frac{\boldsymbol{b} \times (\boldsymbol{c} + \boldsymbol{e})}{8} \tag{2}$$

Cycling performance assessment

rHRI testing was followed by 5TT and 60TT, with cycling performance recorded as total work done [kilojoules (kJ)]. 5TT was performed before 60TT, separated by a 60-min rest period.

HRR assessment and calculation

At the conclusion of $rHRI_{160\ W}$, $rHRI_{200\ W}$, 5TT and 60TT, participants dismounted the ergometer and sat quietly in a chair for HRR assessment, calculated as the difference between HR at the end of exercise (HRend; mean of final 5 s) and HR after 60 s of seated recovery (mean over 5 s). Figure 1 depicts the testing protocol.

HRV assessment and calculation

RR intervals were recorded daily during 3 min of quiet rest in a standing posture (Bellenger et al. 2016b; Le Meur et al. 2013) at home upon wakening and after emptying the urinary bladder using a personal HR monitor. Data were downloaded to Polar Protrainer 5 software (Polar Electro Oy, Kempele, Finland) where Polar's automatic filtering removed any artefacts. Data were then exported to HRV analysis software (Kubios HRV Analysis, version 2.0 beta

Fig. 1 Testing protocol flowchart. *HRR* heart rate recovery, *LT7* light training, *min* minute, $rHRI_{120\ W}$ maximal rate of heart rate increase assessed at 120 W (increase in heart rate due primarily to parasympathetic nervous system activation), $rHRI_{160\ W}$ maximal rate of heart rate increase assessed at 160 W, $rHRI_{200\ W}$ maximal rate of heart rate increase assessed at 200 W, $rHRI_{120–200\ W}$, maximal rate of heart rate increase assessed during transition from 120 W to 200 W (increase in heart rate due primarily to sympathetic nervous system activation), *5TT* cycling performance during a 5-min time trial; *60TT* cycling performance during a 60-min time trial

1, The Biomedical Signals Analysis Group, University of Kuopio, Finland) where remaining artefacts were manually removed, and the final 2 min analysed. Vagal-related HR modulation was analysed via the root-mean-square difference of successive normal RR intervals (RMSSD) (Buchheit 2014), along with RR interval and RMSSD:RR interval. These indices were analysed as rolling 7 day averages and presented as values on the final day of LT (LT7), the seventh day of HT (HT7), the 14th day of HT (HT14), the fifth day of T (T5) and the 10th day of T (T10).

Subjective training tolerance assessment

Training tolerance was determined daily via a Daily Analysis of Life Demands for Athletes (DALDA) questionnaire, and perceptions of mood state, energy levels, stress, fatigue and muscle soreness as previously described (Bellenger et al. 2016b).

Training intervention

Training was conducted on each participant's bicycle attached to a stationary trainer. LT required 6 days of cycling exercise for 30–60 min per day at 65–85% of peak HR, but no training on day seven preceding post-LT testing, such that it would allow participants to be rested and recovered from any pre-study training prior to completing HT. HT required 124 min of cycling per day, with 34% of the training performed above 88% of peak HR, and was intended to induce substantial fatigue from which participants would not recover by testing on the day following the final training session. Details of the HT programme have been provided previously (Nelson et al. 2014). Tapering lasted 10 days with rest days one and nine. Seven of eight training sessions required 30–60 min per day at 65–85% of peak HR, with one interval session (four repeats of 3 min at 69–81% peak HR followed by 2 min at 88–92% peak HR) conducted on day seven to provide some variety in training. Training HR data were recorded at 15 s intervals to determine training load via Training Impulse (TRIMP; duration in minutes multiplied by % of peak HR) (Banister 1991).

Statistical analysis

Data were analysed using PASW Statistics 18.0 (SPSS, Chicago, IL, USA) and presented as mean ± standard deviation (SD), and also effect size (ES) with 90% confidence intervals. Data were log transformed to reduce bias arising from non-uniformity of error (Hopkins et al. 2009). Outcome measures were compared across time-points using repeated measures analysis of variance with Bonferroni post hoc comparison (statistical significance of $p < 0.05$), and also through magnitude-based inferences (Hopkins et al. 2009), which uses a modified statistical spreadsheet (Hopkins 2006) to calculate ES between time-points using pooled standard deviation (Cohen 2010). Thresholds for ES statistics were ≤0.2 (trivial), >0.2 (small), >0.6 (moderate), >1.2 (large), >2.0 (very large) and >4.0 (extremely large) (Hopkins et al. 2009). Probabilities to establish differences as lower, similar or higher than the smallest worthwhile change were interpreted as: <1%, almost certainly not; 1–5%, very unlikely; 5–25%, unlikely; 25–75%, possibly; 75–95%, likely; 95–99%, very likely and >99%, almost certain. If the chance of higher and lower differences was >5%, the true difference was unclear. Within-subject correlations between HR parameters and performance were evaluated using univariate analysis of covariance (Bland and Altman 1995), with *r* values evaluated according to Hopkins et al. (2009). Other inter-variable relationships were assessed using Pearson's correlation and presented as r value with 90% confidence intervals.

Results

Effect of training on cycling performance

Twelve participants completed the study (age 33.8 ± 10.2 years, body mass 76.7 ± 12.4 kg). Daily TRIMP almost certainly increased from 2841 ± 434 units at LT7 to 9283 ± 558 units at HT14 (ES ± 90% confidence interval = 10.92 ± 0.76; $p < 0.001$), and then almost certainly decreased to 1859 ± 652 units from HT14 to T10 (ES = −15.39 ± 2.30; $p < 0.001$). Work done during 5TT

and 60TT was 100.73 ± 7.78 kJ and 795.44 ± 91.70 kJ at LT7, while Peak HR during 5TT and 60TT was 180.67 ± 10.50 bpm and 178.50 ± 9.65 bpm at LT7. Figure 2a, b depict changes in these variables, respectively.

Effect of training on subjective training tolerance

Fatigue, muscle soreness, energy levels, mood state, stress and the number of 'worse than normal' scores on the DALDA were 3.46 ± 1.39, 3.17 ± 1.53, 7.25 ± 1.12, 7.40 ± 1.30, 3.63 ± 1.94 and 1.59 ± 1.66, respectively, at LT7. Figure 3 shows the changes in these variables.

Effect of training on HRV

RR interval, Ln RMSSD and Ln RMSSD:RR interval were 826 ± 79, 3.31 ± 0.22 ms and 3.99 ± 0.53 units, respectively, at LT7. Changes in RR interval and Ln RMSSD:RR interval were possible to almost certainly trivial ($p \geq 0.02$). Changes in Ln RMSSD are shown in Fig. 4a.

Effect of training on rHRI

$rHRI_{120\ W}$, $rHRI_{160\ W}$, $rHRI_{200\ W}$ and $rHRI_{120–200\ W}$ were 6.81 ± 2.47, 5.21 ± 1.99, 4.55 ± 1.68 and 1.12 ± 1.16 bpm/s, respectively, at LT7, and changes in these variables are shown in Fig. 4b.

Pre-exercise HR during $rHRI_{120\ W}$, $rHRI_{160\ W}$, $rHRI_{200\ W}$ and $rHRI_{120–200W}$ was 36.09 ± 3.35, 36.36 ± 5.38, 36.07 ± 4.12 and 67.27 ± 5.63% of peak HR, respectively, at LT7. Steady-state HR during $rHRI_{120\ W}$, $rHRI_{160\ W}$, $rHRI_{200\ W}$ and $rHRI_{120–200\ W}$ was 66.33 ± 5.23, 71.84 ± 3.83, 77.04 ± 4.48 and 79.47 ± 5.00% peak HR, respectively, at

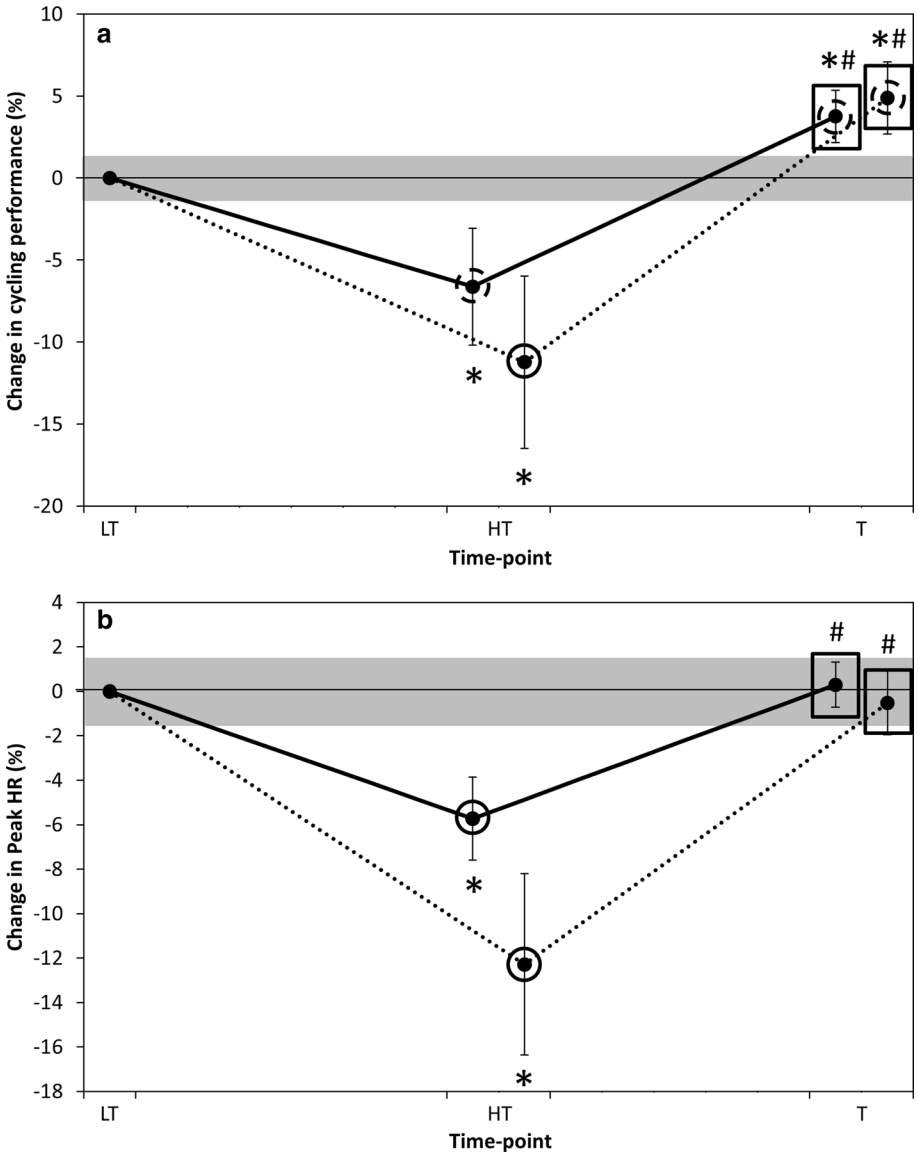

Fig. 2 Percentage change in **a** cycling performance and **b** peak heart rate from LT7. Data are mean ± 90% confidence level. *Grey shaded* areas represent the smallest worthwhile change. *HR* heart rate, *LT7* light training, *HT14* heavy training, *T10* tapering. *Continuous line*, cycling performance during a 5-min time trial; dotted line, cycling performance during a 60-min time trial; *dashed circle*, very likely chance of practically meaningful difference in value from LT7; *continuous circle*, almost certain chance of practically meaningful difference in value from LT7; *continuous rectangle*, almost certain chance of practically meaningful difference in value from HT14; (*asterisk*) significantly different ($p < 0.05$) from LT7; (*hash*) significantly different ($p < 0.05$) from HT14

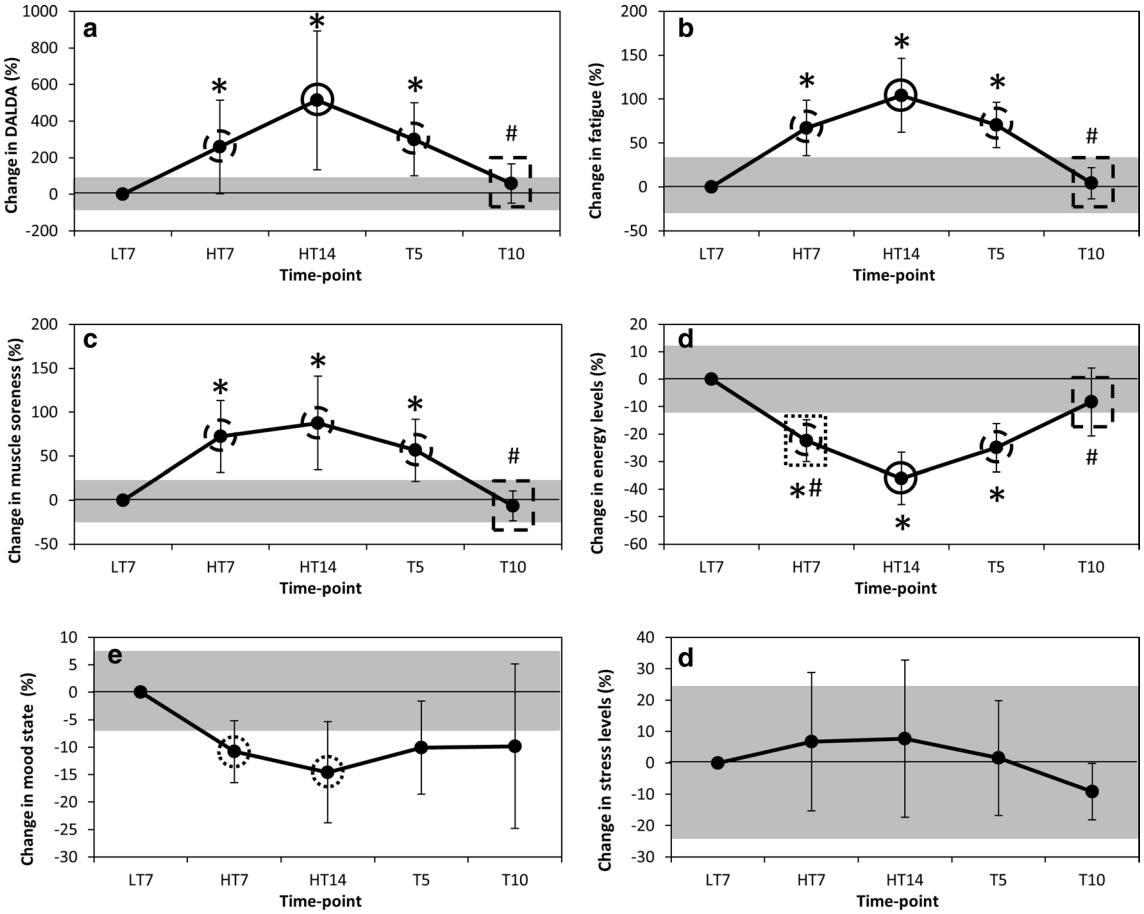

Fig. 3 Percentage change in **a** DALDA 'worse than normal' score, **b** fatigue, **c** muscle soreness, **d** energy levels, **e** mood state and **f** stress from LT7. Data are mean ± 90% confidence level. *Grey shaded areas* represent the smallest worthwhile change. *DALDA* daily analysis of life demands for athletes questionnaire, LT7 rolling 7 day average on the seventh day of light training, HT7 rolling 7 day average on the seventh day of heavy training, HT14 rolling 7 day average on the fourteenth day of heavy training, T5 rolling 7 day average on the fifth day of tapering, T10 rolling 7 day average on the tenth day of tapering. *Dotted circle*, likely chance of practically meaningful difference in value from LT7; *dashed circle*, very likely chance of practically meaningful difference in value from LT7; *continuous circle*, almost certain chance of practically meaningful difference in value from LT7; *dotted rectangle*, likely chance of practically meaningful difference in value from HT14; *dashed rectangle*, very likely chance of practically meaningful difference in value from HT14; *continuous rectangle*, almost certain chance of practically meaningful difference in value from HT14; (*asterisk*), significantly different ($p < 0.05$) from LT7; (*hash*), significantly different ($p < 0.05$) from HT14

LT7. Changes in pre-exercise and steady-state HR in these variables are shown in Fig. 4c, d.

A large inverse correlation was found between changes (HT14 minus LT7) in $rHRI_{120\ W}$ and changes in pre-exercise HR during the assessment of this parameter ($r = -0.67 \pm 0.31$; $p = 0.02$). Similarly, a very large inverse correlation was found between changes in $rHRI_{160\ W}$ and changes in pre-exercise HR during its assessment ($r = -0.78 \pm 0.23$; $p = 0.003$). Smaller correlations were found between changes in $rHRI_{120\ W}$ and $rHRI_{160\ W}$, and changes in steady-state HR during their assessments ($r = -0.52 \pm 0.39$; $p = 0.08$, and -0.52 ± 0.40; $p = 0.09$, respectively). Changes in $rHRI_{120–200\ W}$ following HT were also inversely correlated with changes in steady-state HR ($r = -0.58 \pm 0.38$; $p = 0.06$).

Given these relationships, changes in pre-exercise and steady-state HR were controlled for when analysing the difference in rHRI from LT7 to HT14. $rHRI_{120\ W}$ and $rHRI_{160\ W}$ very likely ($ES = -0.72 \pm 0.44$; $p = 0.02$) and almost certainly ($ES = -1.28 \pm 0.44$; $p = 0.003$) slowed, respectively, at HT14 when controlled for their changes in pre-exercise HR. Similarly, $rHRI_{120–200\ W}$ likely slowed at HT14 when changes in steady-exercise HR were controlled for ($ES = -0.68 \pm 0.65$; $p = 0.06$).

Effect of training on HRR

HRR after $rHRI_{160\ W}$, $rHRI_{200\ W}$, 5TT and 60TT was 56.07 ± 11.95 bpm, 57.10 ± 14.43 bpm, 38.40 ± 7.94 bpm and 45.40 ± 8.00 bpm, respectively,

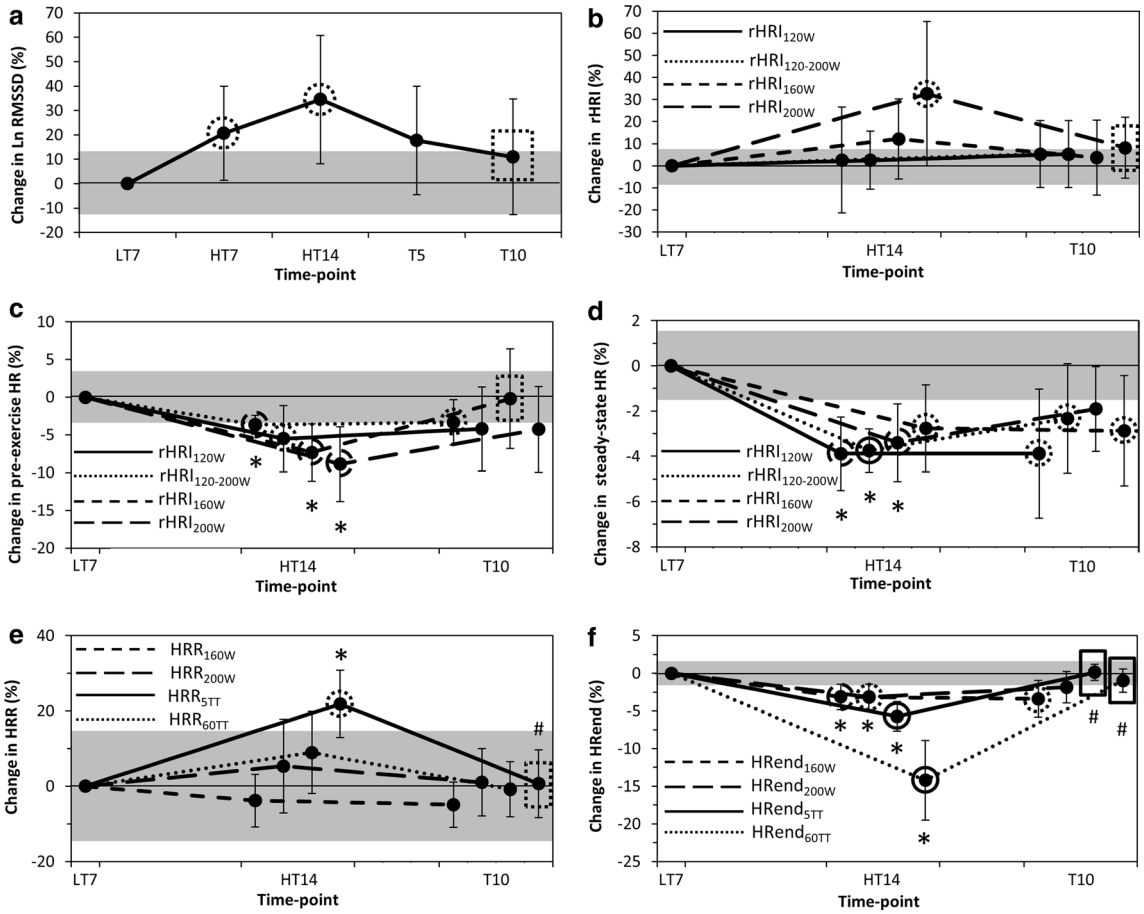

Fig. 4 Percentage change in **a** Ln RMSSD, **b** rHRI, **c** pre-exercise HR during rHRI assessment, **d** steady-state HR during rHRI assessment, **e** HRR and **f** HR at the end of exercise from LT7. Data are mean ± 90% confidence level. *Grey shaded areas* represent the smallest worthwhile change for each variable. *HR* heart rate, *HRR* heart rate recovery, $HRR_{160\ W}$ heart rate recovery assessed following exercise at 160 W, $HRR_{200\ W}$ heart rate recovery assessed following exercise at 200 W, HRR_{5TT} heart rate recovery assessed following a 5-min cycling time trial, HRR_{60TT} heart rate recovery assessed following a 60-min cycling time trial, *HRend*, heart rate at the end of exercise, $HRend_{160\ W}$ heart rate at the end of exercise at 160 W, $HRend_{200\ W}$ heart rate at the end of exercise at 200 W, $HRend_{5TT}$ heart rate at the end of a 5-min cycling time trial, $HRend_{60TT}$ heart rate at the end of a 60-min cycling time trial, HT7 seventh day of heavy training, *HT14* fourteenth day of heavy training, *Ln RMSSD* natural logarithm of the root-mean-square difference of successive normal R-R intervals, *LT7* seventh day of light training, *rHRI* maximal rate of heart rate increase, $rHRI_{120\ W}$ maximal rate of heart rate increase assessed at 120 W, $rHRI_{160\ W}$ maximal rate of heart rate increase assessed at 160 W, $rHRI_{200\ W}$ maximal rate of heart rate increase assessed at 200 W, $rHRI_{120–200\ W}$ maximal rate of heart rate increase assessed during transition from 120 W to 200 W, *T5* fifth day of tapering, *T10* tenth day of tapering. *Dotted circle*, likely chance of practically meaningful difference in value from LT7; *dashed circle*, very likely chance of practically meaningful difference in value from LT7; *continuous circle*, almost certain chance of practically meaningful difference in value from LT7; dotted rectangle, likely chance of practically meaningful difference in value from HT14; *continuous rectangle*, almost certain chance of practically meaningful difference in value from HT14; (*asterisk*), significantly different ($p < 0.05$) from LT7; (*hash*), significantly different ($p < 0.05$) from HT14

at LT7. HRend during $rHRI_{160\ W}$, $rHRI_{200\ W}$, 5TT and 60TT was 130.75 ± 12.19 bpm, 139.35 ± 13.44 bpm, 179.95 ± 10.19 bpm and 177.75 ± 9.59 bpm, respectively. Changes in HRR and HRend in these variables are shown in Fig. 4e, f.

Within-subject correlations between cycling performance and HR parameters

Within-subject correlations (using LT7, HT14 and T10 data-points) were trivial to small between performance

and modes of $rHRI_{160\ W}$, $rHRI_{200\ W}$ and $rHRI_{120–200\ W}$ ($r \leq 0.33 \pm 0.41$; $p \geq 0.12$), and were unchanged when controlling for pre-exercise or steady-state HR ($r \leq 0.34 \pm 0.42$; $p \geq 0.12$). Correlations between $rHRI_{120W}$ and 5TT ($r = 0.22 \pm 0.56$; $p = 0.29$) and 60TT ($r = 0.32 \pm 0.40$; $p = 0.12$) were strengthened when controlling for pre-exercise HR ($r = 0.35 \pm 0.32$; $p = 0.09$ and $r = 0.43 \pm 0.32$; $p = 0.03$, respectively).

A large within-subject correlation was found between 5TT performance and 5TT-derived HRR ($r = -0.61 \pm 0.24$; $p = 0.001$). 5TT-derived HRR was also correlated with peak HR during 5TT ($r = 0.71 \pm 0.24$; $p < 0.001$), and when controlling for the effect of peak HR, the correlation between 5TT and 5TT-derived HRR became trivial ($r = -0.02 \pm 0.18$; $p = 0.94$). Within-subject correlations were trivial to small between performance and other HR parameters ($r \leq 0.31 \pm 0.40$; $p \geq 0.13$).

Discussion

The present study showed that HT-induced changes in pre-exercise and steady-state HR affected rHRI assessment, such that rHRI was unchanged following HT unless these moderating variables were controlled for. Additionally, controlling for pre-exercise HR in the within-subject relationships between $rHRI_{120W}$ and exercise performance resulted in moderate-strength correlations, suggesting that rHRI assessed at ~65% of peak HR provided the most sensitive measure for tracking performance changes in the present context.

rHRI

In response to overload training and subsequent taper, which produced moderate attenuation of performance followed by large performance improvement, $rHRI_{120\ W}$, $rHRI_{120–200\ W}$ and $rHRI_{160\ W}$ remained unchanged, while $rHRI_{200\ W}$ increased and then decreased. Unattenuated $rHRI_{120\ W}$ following HT conflicts previous research demonstrating reductions in rHRI (ES −0.33 to −0.65) assessed at similar relative intensities (i.e. ~65% of peak HR) (Bellenger et al. 2015; Nelson et al. 2014). However, the large inverse correlation between changes in $rHRI_{120\ W}$ and changes in pre-exercise HR during its assessment indicated that participants experiencing greater decreases in pre-exercise HR also experienced greater increases in rHRI. The effect of altered pre-exercise HR on rHRI is intuitive; since previous studies of cardiovascular control demonstrated that exercise-induced increases in HR up to ~100–120 bpm were primarily the result of parasympathetic withdrawal (Robinson et al. 1966; Victor et al. 1987; Warner and Cox 1964; White and Raven 2014), an increase in parasympathetic modulation prior to exercise, as evidenced by reductions in pre-exercise HR in the present study, was likely to affect rHRI assessment unless controlled for, and doing so ultimately resulted in a moderate slowing in $rHRI_{120\ W}$ following HT. Similar reductions in $rHRI_{160\ W}$ and $rHRI_{120–200\ W}$ were evident when controlling for changes in pre-exercise HR and steady-state HR, respectively, during their assessment. Together, HT-induced reductions in these rHRI when controlling for changes in pre-exercise and steady-state HR support the earlier findings of rHRI (Bellenger et al. 2015; Nelson et al. 2014); however, it is not immediately clear why changes in these variables had a profound effect on rHRI in the present study. It does, however, provide novel insight into the mechanistic properties of rHRI. Specifically, since pre-exercise and steady-state HR as measures of ANS *activity* remained unchanged despite a slowing in rHRI following HT in previous studies (Bellenger et al. 2015; Nelson et al. 2014), it was hypothesised that rHRI solely assessed the *reactivity* (or responsiveness) of the ANS. The present study suggests, however, that the reactivity of the ANS is also influenced by its level of activity, since decreases in pre-exercise and steady-state HR were found in the assessment of rHRI following HT, and these decreases appeared to diminish any slowing in rHRI.

The sensitivity of rHRI for tracking changes in exercise performance also appeared to be influenced by pre-exercise HR. Indeed, relationships between $rHRI_{120\ W}$ and performance were strengthened when controlling for pre-exercise HR, and were such that slower values of rHRI resulted in reduced performance, which is consistent with the findings of earlier studies (Bellenger et al. 2015; Nelson et al. 2014). Thus, rHRI assessed at 120 W or ~65% of peak HR, which is theoretically reflective of predominantly parasympathetic HR modulation, resulted in the most sensitive tracking of performance (when controlling for pre-exercise HR).

HRV

The HT-induced decreases in pre-exercise and steady-state HR, representative of increased parasympathetic modulation prior to and during exercise, respectively, were supported by heightened RMSSD following HT. Increased RMSSD has previously been demonstrated in athletes experiencing FOR (Bellenger et al. 2016b; Le Meur et al. 2013), and may be considered paradoxical given parasympathetic modulation is also heightened following training leading to improved performance (Bellenger et al. 2016a). With regard to FOR, however, increased parasympathetic modulation may limit full sympathetic engagement during high intensity exercise (as evidenced by the reduction in peak HR during 5TT and 60TT in this study), thereby attenuating maximal cardiac output, and potentially contributing

to the reduction in maximal performance characteristic of FOR (Le Meur et al. 2013).

Following taper, the increased parasympathetic modulation was overcome, which coincided with a return of peak HR during 5TT and 60TT to LT7 levels and an improvement in performance. Thus, while previous research on the effect of overreaching on HRV may be considered equivocal (Bellenger et al. 2016a), perhaps due to the naturally large day-to-day variation in isolated measures of HRV (Al Haddad et al. 2011), this study's results further support the utilisation of rolling 7-day average HRV as a means of overcoming day-to-day variations in HRV that may provide consistencies in future studies.

HRR

The moderate increase in 5TT-derived HRR following HT is supportive of increases in HRR assessed after maximal performance tests lasting 5–30 min (ES = 0.46–0.82) (Aubry et al. 2015; Dupuy et al. 2013; Nelson et al. 2014; Thomson et al. 2015a). However, $rHRI_{160\ W}$, $rHRI_{200\ W}$ and 60TT-derived HRR remained unchanged throughout this study's intervention. Since HRR is the result of both sympathetic withdrawal and parasympathetic reactivation, with the early and rapid deceleration in HR following exercise caused primarily by parasympathetic reactivation, and a slower deceleration in HR caused by sympathetic withdrawal (Kannankeril et al. 2004; Pierpont and Voth 2004), excessive sympathetic stimulation during exercise may cause a slower HRR when withdrawn, and lower sympathetic stimulation during exercise may cause a faster HRR (Borresen and Lambert 2008). This understanding may explain the greater increase in 5TT-derived HRR at HT14 in comparison to HRR following $rHRI_{160\ W}$ and $rHRI_{200\ W}$, since the decrease in HRend (indicating reduced sympathetic stimulation) was greater in 5TT (ES = −0.81) in comparison to $rHRI_{160\ W}$ (ES = −0.27) and $rHRI_{200\ W}$ (ES = −0.31). However, this theory cannot explain the trivial changes in 60TT-derived HRR at HT14, since change in HRend following this test (ES −1.56) was greater than all other assessments. Consequently, the duration of effort may have also influenced the assessment of 60TT-derived HRR.

In any case, since the aim of HR parameter assessment was to detect changes in athletic training status (for which the gold standard measure is exercise performance), the finding of a practically meaningful change in HRR following maximal exercise is essentially redundant given that a direct measure of training status (i.e. time-trial performance) was also measured. Thus, an important practical finding from the present study was that HRR assessed after submaximal exercise was not sensitive for detecting changes in training status.

Conclusion

rHRI assessed at a moderate workload, and at workloads theoretically reflecting contributions of parasympathetic and sympathetic HR modulation were slowed following HT, but only after controlling for concurrent changes in pre-exercise or steady-state HR. Within-subject correlations between $rHRI_{120\ W}$ and performance were strengthened when controlled for pre-exercise HR, suggesting this method provides the most sensitive rHRI measure for tracking performance. Increased resting RMSSD following HT supported the heightened parasympathetic modulation found during rHRI assessment, while submaximally derived HRR was not sensitive to changes in training status, which potentially limits its practical applicability for athletic monitoring.

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Compliance with ethical standards

Conflict of interest The University of South Australia has applied for a patent on the rHRI technology described in this manuscript, and researchers Davison and Buckley are employees of the University. Researcher Karavirta is an employee of Polar Electro Oy.

Ethics approval Was granted by the University of South Australia's Human Research Ethics Committee, and volunteers provided written informed consent prior to participating. All procedures performed in this study involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

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