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ORIGINAL ARTICLE

Changes in heart rate recovery after high-intensity training in well-trained cyclists

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Abstract Heart rate recovery (HRR) after submaximal exercise improves after training. However, it is unknown if this also occurs in already well-trained cyclists. Therefore, 14 well-trained cyclists (VO_{2max} 60.3 ± 7.2 ml kg^{-1} min^{-1}; relative peak power output 5.2 ± 0.6 W kg^{-1}) participated in a high-intensity training programme (eight sessions in 4 weeks). Before and after high-intensity training, performance was assessed with a peak power output test including respiratory gas analysis (VO_{2max}) and a 40-km time trial. HRR was measured after every high-intensity training session and 40-km time trial. After the training period peak power output, expressed as W kg^{-1}, improved by 4.7% ($P = 0.000010$) and 40-km time trial improved by 2.2% ($P = 0.000007$), whereas there was no change in VO_{2max} ($P = 0.066571$). Both HRR after the high intensity training sessions (7 ± 6 beats; $P = 0.001302$) and HRR after the 40-km time trials (6 ± 3 beats; $P = 0.023101$) improved significantly after the training period. Good relationships were found between improvements in $HRR_{40\text{-km}}$ and improvements in peak power output ($r = 0.73$; $P < 0.0001$) and 40-km time trial time ($r = 0.96$; $P < 0.0001$). In conclusion, HRR is a sensitive marker which tracks changes in training status in already well-trained cyclists and has the potential to have an important role in monitoring and prescribing training.

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Introduction

To achieve a high level of performance, competitive cyclists must strive to find a balance between the most appropriate training load followed by a minimal, though adequate recovery period. While training load can be influenced by intensity, volume, frequency and duration, recovery is influenced by less controllable factors such as stress, sleeping patterns, nutrition and psychological and sociological wellbeing (Jeukendrup 2002; Kenttä and Hassmen 1998). If the training load is too high and/or the recovery period is insufficient, the applied training load cannot be tolerated and symptoms of fatigue will develop. A continuous imbalance in this relationship will lead to 'functional' or 'non-functional' overreaching and can in the long term develop into an overtraining syndrome with detrimental effects on performance (Meeusen et al. 2006).

A variety of measurement tools have been developed in an attempt to predict and monitor changes in training status (Lambert and Borresen 2006). Most of these methods aim, either directly or indirectly, to measure the overall well-being of the athlete in response to the applied training load. However, no single measurement has yet been identified that predicts with consistent precision the imminent symptoms of fatigue (Lambert and Borresen 2006). This limits the practical application of these measurements. A more objective way of quantifying 'coping with training load' might be to analyse the response of the autonomic nervous system to training. The autonomic nervous system consists of parasympathetic and sympathetic components and is interlinked with many other physiological systems

(Borresen and Lambert 2008; Kiviniemi et al. 2007). The responsiveness of the autonomic nervous system may therefore provide useful information about the functional adaptations of the human body. It is known that sympathetic hyperactivity or reduced parasympathetic activity is associated with an increased risk of cardiac disease and overall mortality (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology 1996). Similarly studies have found altered autonomic nervous system function in overtrained athletes (Kuipers 1998; Lehmann et al. 1997, 1998).

Two non-invasive methods that measure autonomic nervous system functioning and modulation are the measurement of heart rate variability (HRV) (Baumert et al. 2006; Kiviniemi et al. 2007; Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology 1996) and heart rate recovery (HRR) after exercise (Borresen and Lambert 2007; Hedelin et al. 2000; Lamberts et al. 2004). HRR is determined as the rate at which heart rate decreases after cessation of moderate to heavy exercise (Shetler et al. 2001). One of the first papers which specifically studied HRR after exhausting exercise was published in 1931 (Boas 1931). Since then it is has become general knowledge that HRR recovers faster in well-trained compared to untrained subjects after similar intensities (Bunc et al. 1988; Short and Sedlock 1997) and recently that a low HRR is associated with a higher risk on overall mortality (Cole et al. 1999). However, only a few studies have investigated the longitudinal effects of training on HRR in healthy subjects (Borresen and Lambert 2007; Sugawara et al. 2001; Yamamoto et al. 2001).

HRR improved in a study of previously untrained men who were exposed to 8 weeks of endurance cycle training and then reverted back to the pre-training levels following the 4 weeks of detraining (Sugawara et al. 2001). Another study showed that resting heart rate decreased and HRR improved after 6 weeks of endurance training (Yamamoto et al. 2001). The authors concluded that the training-induced changes associated with the autonomic control with heart rate occurred sooner in the recovery period after exercise than at rest. This suggests that HRR has the potential to be a sensitive tool that tracks changes in performance parameters.

Buchheit et al. (2008) showed an improved HRR and HRV indices in physically active subjects after 9 weeks of high-intensity training and concluded that HRR indices are more sensitive training markers than HRV indices (Buchheit et al. 2007, 2008). However, it is not known whether the same changes in HRR also occur in already well-trained subjects with improvements in performance.

Accordingly, the aim of this study was to expose a group of well-trained cyclists to a high-intensity training programme to determine whether their HRR improved as they progressed through the training programme. We hypothesised that HRR would track changes in performance in these cyclists.

Methods

Recruitment

The sample size for this study was determined using the data of Lamberts et al. (2004), which showed that the day-to-day variability of submaximal heart rate and HRR, 60 s after exercise was about 5–6 and 7–8 beats min^{-1}, respectively (these values were defined as the 95% confidence intervals of within subject range), with a standard deviation of 3 beats min^{-1}. As this study was conducted on physically active subjects who maintained their training load, the smallest meaningful difference in the current study was defined as 9 beats min^{-1} with a standard deviation of 6 beats min^{-1} to accommodate possible effects of variations in response to the training. Using these parameters, the sample size was calculated to be $n = 7$ (to achieve a statistical power of 80% and a significance level of 5%) (Altman 1991). However, due to the nature of the training study (high intensity) and the expected high dropout rate, we decided to recruit more subjects to assure sufficient statistical power.

Sixteen well-trained male cyclists, with at least 3 years of cycling experience and a minimal training load of 6 h per week, were recruited to participate in this study. After being fully informed about the risks and stresses associated with the research protocol, all subjects completed a Physical Activity Readiness Questionnaire (PAR-Q) (American College of Sports Medicine 2007), were personally interviewed about their training history and their training logs were analysed. The study was approved by the Ethics and Research Committee of the Faculty of Health Sciences of the University of Cape Town and all subjects signed an informed consent before entering the study.

Study design

Prior to the testing and training period all cyclists performed a 40-km familiarization time trial on an electronically braked cycle ergometer (Computrainer™ Pro 3D, RacerMate, Seattle, USA) over a flat course.

Three days before the start of the training period a peak power output test (PPO) was performed, which included respiratory gas analysis for measurement of maximal oxygen consumption (VO_{2max}), followed by a 40-km time trial (40-km TT) 48 h after the PPO test and 1 day before the start of the training period. After completing the initial

tests, subjects started their training protocol which consisted of two high intensity training (HIT) sessions, two 90 min recovery rides below lactate threshold (Solberg et al. 2005) and three resting days each week, for 4 weeks. During all testing and training subjects were blinded to any feed-back of time, power output, heart rate and speed to prevent them adopting a pacing strategy with the potential of biasing the performance outcomes. The only exception to this was the display of completed distance during the 40-km TT. Additionally, subjects were asked to avoid participating in any racing or prolonged or high-intensity exercise during the study. Each subject was asked to maintain a detailed training diary and to record all heart rate data during all training sessions performed outside of the laboratory for the duration of the study. Subjects were questioned and training logbooks inspected prior to the second testing phase to ensure that they had adhered to the training protocol.

Warming-up and calibration

Before all testing and training sessions subjects performed a self-paced 15 min warm-up ride on a simulated flat 40-km TT course. Testing and training were done on the subject's own bicycle which was mounted on to the Computrainer^{TM} ergometer system. The rear wheel was inflated to 800 kPa after which the system's load generator was calibrated to a rolling resistance of between 0.88 and 0.93 kg. This calibration procedure was done before and directly after the 15 min warming-up period to ensure accurate calibration as recommended by Davidson et al. (2007). The mean overall rolling resistance of the Computrainer system during all testing and training sessions was calibrated to 0.9110 ± 0.0186 kg.

All test and training sessions were done under stable climate conditions (22.3 ± 1.3°C, 53.7 ± 2.4% relative humidity, 101.9 ± 0.8 hPa) and at a similar time of day (within 60 min). Body mass was measured continuously throughout the study whenever a subject visited the laboratory for either a testing or training session. Body fat percentage (Durnin and Womersley 1974) and the sum of seven skinfolds (triceps, biceps, supra-iliac, sub-scapular, calf, thigh and abdomen) measured with the methods described by Ross and Marfell-Jones 1991 before the start of both 40-km TT's. Stature was measured before the first 40-km TT.

Performance tests

Outcomes of the 40-km TT and the PPO test, which included respiratory gas analysis (VO_{2max}), were used as markers of cycling performance (Hawley and Noakes 1992; Lucia et al. 2002a, b; Mujika and Padilla 2001; Padilla et al. 2000). These tests were conducted before training and again after the 28 day training period which was followed by a 10 day low intensity recovery period. This tapering period allowed the cyclists to recover from their HIT session before they were re-tested (Jeukendrup 2002; Shepley et al. 1992).

All subjects were asked to perform a 90 min submaximal recovery ride 24 h prior to the different tests. The PPO test was performed at a starting work rate of 2.50 W kg^{-1} body mass after which the load was increased incrementally by 20 W each minute until the cyclist could not sustain a cadence greater than 70 rpm or was volitionally exhausted. During this test ventilation volume (V_E), oxygen uptake (VO_2) and CO_2 production (VCO_2) were measured over 15 s intervals using an on-line breath-by-breath gas analyser and pneumotach (Oxycon, Viasis, Hoechberg, Germany). Subjects were verbally encouraged to perform to maximal exhaustion. Maximal PPO was determined as the mean power output during the final minute of the PPO test, whereas VO_{2max} (ml kg^{-1} min^{-1}) was defined as the highest recorded reading over a 30 s period. The PPO test was accepted as being maximal if the subjects had a minimal respiratory exchange ratio of 1.15 and a slow decrease in cadence despite attempting to maintain their power output as they approached the end of the test. The 40-km TT was performed on a simulated 40-km flat time trial course which was programmed into the Computrainer^{TM}. Subjects were allowed to drink water ad libitum throughout the test and were given clear instructions to complete the 40-km distance in the fastest possible time. All subjects refrained from any food in the last 2 h and from any caffeine in the last 3 h before all performance testing and training sessions.

Training sessions

All subjects followed the same structured training programme which included HIT training sessions, rest days and recovery rides. One HIT training session consisted of eight intervals at approximately 80% of PPO, determined for each subject during the initial PPO test. Each interval of 4 min was followed by a 90 s self-paced recovery (Fig. 1). All HIT sessions were closely supervised while speed (km h^{-1}), power output (W), cadence (rpm) and heart rate (beats min^{-1}) were captured at a rate of 34 Hz.

Heart rate and HRR

During all performance tests and training sessions heart rate was recorded continuously at a capturing rate of 34 Hz by the Computrainer software. HRR after the HIT sessions (HRR_{HIT}) was also recorded with the Computrainer software whereas the HRR after the 40-km time trials ($HRR_{40\text{-}km}$) was recorded with a heart rate monitor at 5 s intervals (Polar Sport Tester, Polar Electro, Kempele, Finland).

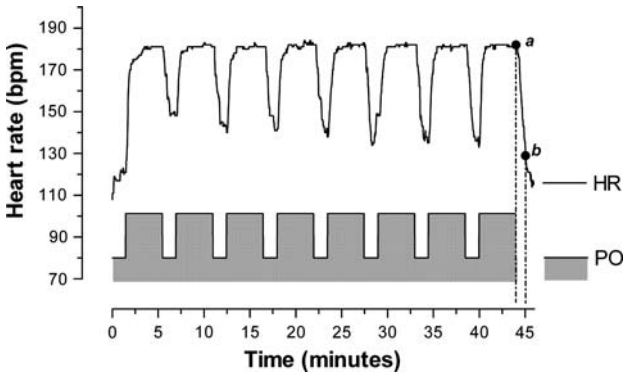

Fig. 1 The average heart rate (HR) response of an arbitrary subject undergoing a high intensity training session [see power output (PO) profile below]. **a** Represents the start of the heart rate recovery measurement (HRR_{HIT}). **b** Represents the end of the heart rate recovery measurement (HRR_{HIT})

HRR was calculated as described previously (Lamberts et al. 2004) and defined as the reduction in heart rate within the first 60 s after the cessation of the eighth interval of an HIT session (HRR_{HIT}) or the 40-km TT ($HRR_{40\text{-km}}$). In an attempt to control factors which could influence heart rate and HRR, subjects were asked to sit passively straight up on their cycles (Gnehm et al. 1997), remain still and were not allowed to talk during the recovery period.

Data analysis

Analysis of performance and training data were performed using CyclingPeaks™ analysis software (WKO+ edition, Version 2.1, 2006, Lafayette, CO, USA) and the Computrainer coaching software (Version 1.5.308, RacerMate, Seattle, USA). Data were expressed as absolute values where changes in performance parameters were expressed in absolute and relative (percentages) values. Percentage change was calculated as;

$$\% \text{ change} = \frac{(\text{Post training value} - \text{Pre training value})}{(\text{Pre training value})} \times 100$$

To compare the changes in VO_{2max} and PPO both these measurements were calculated and expressed per kilogram (ml kg^{-1} min^{-1} and W kg^{-1}, respectively). Heart rate data were analysed with CyclingPeaks™ analysis software and Polar precision performance software (Version 4.03.049, Polar Electro, Kempele, Finland).

Statistical analysis

The data were analysed with STATISTICA version 7.0 (Stat-soft Inc., Tulsa, OK, USA) for any statistical significance ($P < 0.05$). All data are expressed as means ± standard deviations. A *t* test with dependent variables was used to determine any training-induced changes in performance parameters (PPO, VO_{2max}, 40-km TT and $HRR_{40\text{-km}}$). A one-way analysis of variance with repeated measures was used to determine any differences in the HRR measured during the HIT sessions (HRR_{HIT}). Sphericity of the data was tested using the Mauchley test. When the sphericity condition was violated a Greenhouse-Geisser adjustment was made to the degrees of freedom to counter the increased risk of a type 1 error. Relationships between changes in performance parameters and changes in $HRR_{40\text{-km}}$ were established using a Pearsons Product moment correlation (GraphPad Prism version 3.00 for Windows, GraphPad Software, San Diego, CA, USA). Confidence intervals of the correlations were calculated using a spreadsheet designed for this purpose and downloaded from http://www.sportsci.org.

Results

Two subjects were unable to complete the study as the result of a fractured wrist and a viral illness, respectively, and their data were excluded from further analysis. The general characteristics of the remaining 14 cyclists are shown in Table 1. Based on the mean PPO, power to weight ratios, average amount of training hours per week and years of competitive cycling, all cyclists could be regarded as well-trained (Jeukendrup et al. 2000, 2002). The average training load over the last 6 weeks before the start of the HIT training period was 11 ± 4 h per week which was mainly base training.

Performance

All performance parameters except for VO_{2max} improved after the HIT period (Table 2). Mean PPO expressed as W kg^{-1} improved by 4.7 ± 3.1%, while VO_{2max}

Table 1 Subject characteristics of the 14 cyclists, expressed as $X \pm s$

Variable	
Age (years)	30 ± 6
Stature (cm)	179 ± 7
Body mass (kg)	73.3 ± 8.0
Fat (%)	14.2 ± 3.6
Sum of skinfolds (mm)	57.8 ± 18.8
Peak power output (W)	376 ± 32
Power to weight ratio (W kg^{-1})	5.2 ± 0.6
VO_{2max} (ml kg^{-1} min^{-1})	60.3 ± 7.2
Average 40-km TT time (min.s)	65.26 ± 2.25
Years of competitive cycling (years)	10.2 ± 6.4
Training hours per week (h)	11 ± 4

Table 2 Performance parameters before and after the training period, expressed as $X \pm s$

Variable	Before	After	P value
Peak power output (W)	376 ± 32	389 ± 34	0.000613
Relative peak power (W kg^{-1})	5.2 ± 0.6	5.4 ± 0.5	0.000010
VO_{2max} (ml kg^{-1} min^{-1})	60.3 ± 7.2	61.7 ± 6.5	0.066571
Average 40-km TT time (min.s)	65.26 ± 2.25	63.56 ± 2.00	0.000016
Mean power during the 40-km TT (W)	255 ± 26	270 ± 24	0.000007
Relative power during the 40-km TT (W kg^{-1})	3.5 ± 0.5	3.7 ± 0.4	0.000043

(ml kg^{-1} min^{-1}) only showed a tendency for improvement ($P = 0.066$). Both PPO tests, which were done before and after the training period, produced similar maximal heart rates (189 ± 9 vs. 189 ± 10 beats min^{-1}) with a maximal difference of three beats. The respiratory exchange ratios of 1.24 ± 0.04 and 1.25 ± 0.04 (respectively) assured that both tests were performed at maximal effort. Mean time for the 40-km TT improved by 2.2 ± 1.2%.

Heart rate recovery after the 40-km TT ($HRR_{40\text{-km}}$)

$HRR_{40\text{-km}}$ improved by 6 ± 3 beats ($P = 0.023101$) after completing the HIT period. The average $HRR_{40\text{-km}}$ improved from 29 ± 6 beats before to 35 ± 4 beats after the training period, which is equivalent to a 21 ± 16% improvement ($P = 0.000023$). The intensity (%HR_{max}) and heart rate from which $HRR_{40\text{-km}}$ was measured was similar before and after the training period [97 ± 1% (183 ± 10 beats min^{-1}) vs. 98 ± 2% (184 ± 9 beats min^{-1}) respectively].

Relationships between $HRR_{40\text{-km}}$ and change in performance parameters

When analysing the improvements in $HRR_{40\text{-km}}$ and the improvements in performance parameters two strong relationships were found (Fig. 2). Improvement in 40-km TT time, both absolute and when expressed as a percentage, correlated very well with improvements in $HRR_{40\text{-km}}$, $r = 0.97$; 95% CI 0.91–0.99 ($P < 0.0001$) and $r = 0.96$; 95% CI 0.88–0.99 ($P < 0.0001$), respectively. A similar relationship, but slightly weaker, was found between $HRR_{40\text{-km}}$ and improvement in absolute and relative PPO and when expressed as percentage improvement ($r = 0.67$; 95% CI 0.22–0.89 ($P < 0.0081$) and ($r = 0.73$; 95% CI 0.33–0.91 ($P < 0.0027$), respectively.

Heart rate recovery during high intensity training (HRR_{HIT})

The intensity (%HR_{max}) at the end of each HIT session remained constant throughout the eight HIT sessions (93 ± 2% (175 ± 2 beats min^{-1})). HRR_{HIT} improved throughout the entire training period, being significantly higher at the fifth, sixth, seventh and eighth HIT session compared to the first HIT session (Table 3).

HRR_{HIT} improved by 7 ± 6 beats ($P = 0.001302$) beats per minute between the eighth and first HIT session. The largest change with the lowest standard deviation was found between the sixth and the first HIT session, 7 ± 4 beats ($P = 0.000018$) after which the changes in HRR_{HIT} tended to plateau.

Discussion

This is the first study to investigate changes in HRR during and after 4 weeks of HIT in a group of well-trained cyclists. The results show that all cyclists responded well to the HIT protocol with a significant improvement in all performance parameters, except for VO_{2max}. These improvements in performance parameters are in accordance with other studies, using similar training protocols that have shown the benefits of HIT in well and highly trained cyclists (Laursen et al. 2002; Laursen and Jenkins 2002; Stepto et al. 1999). This finding confirms that the HIT protocol used in this study was correctly implemented.

HIT and $HRR_{40\text{-km}}$

The main finding from this study was that $HRR_{40\text{-km}}$ improved (6 ± 3 beats) after 4 weeks of HIT in a group of well-trained cyclists. This finding is in accordance with the data of Buchheit et al. (2008), who showed a similar magnitude of change in HRR in a group of fit adolescents, who also participated in a HIT programme. The change in HRR after HIT was less than the value which we regarded as meaningful when we calculated the sample size (9 beats) in planning the study (Lamberts et al. 2004). The original assumption, however, was formed from data using a different protocol with a different exercise intensity and duration prior to the measurement of recovery heart rate, which could have influenced the day-to-day variation in heart rate. We performed a post-study

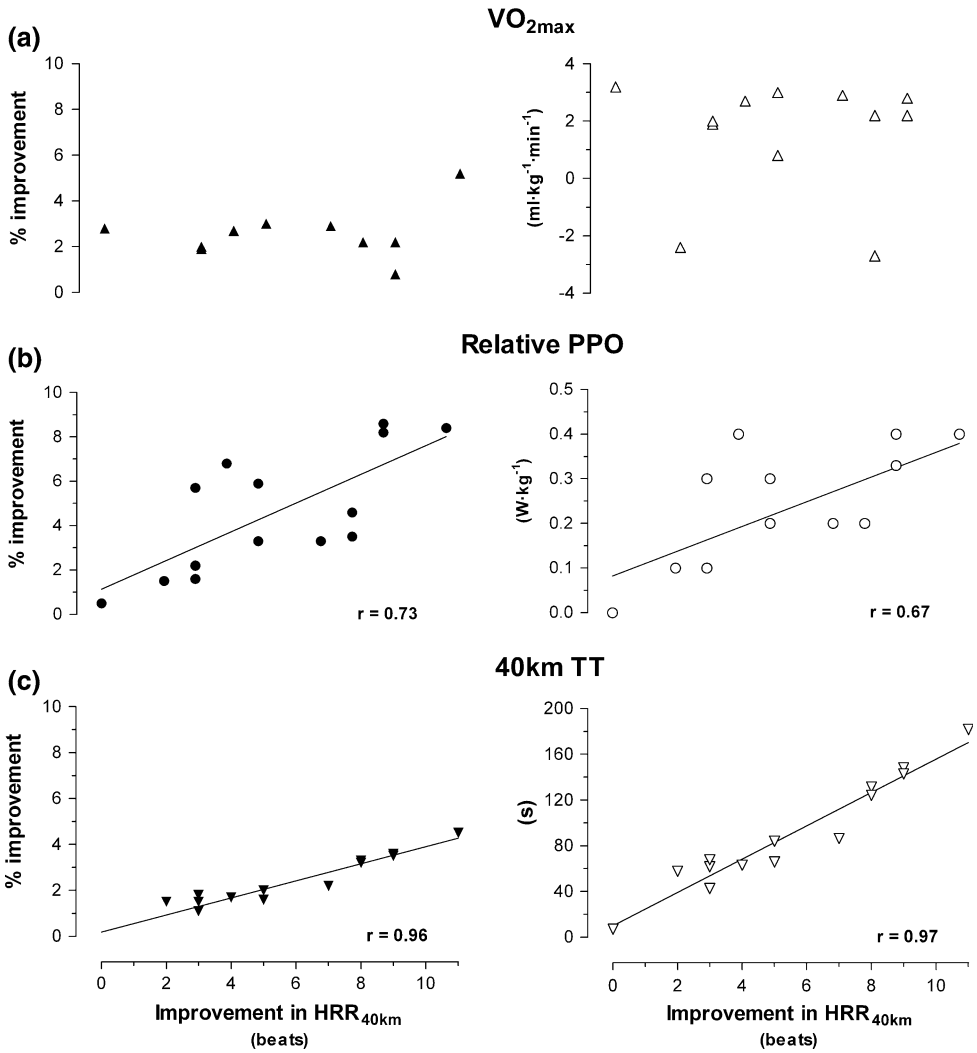

Fig. 2 Improvement in $HRR_{40\text{-km}}$ versus absolute (*open symbols*) and relative (*closed symbols*) improvements in **a** VO_{2max}, **b** relative PPO and **c** 40-km TT time

Table 3 Heart rate recovery after each of the eight high intensity training sessions (HRR_{HIT})

HIT session	1	2	3	4	5	6	7	8
HRR_{HIT} (beats)	36 ± 8	38 ± 9	39 ± 9	40 ± 9	42 ± 8$^{\#}$	43 ± 9$^{\$}$	43 ± 8$^{\$}$	43 ± 8$^{\$}$
Difference versus session 1 (beats)	0 ± 0	2 ± 3	3 ± 5	5 ± 3	5 ± 5	7 ± 4	6 ± 4	7 ± 6

Data ($X \pm s$) are expressed as change in heart rate within the first 60 s after completing the HIT sessions and the difference in HRR compared to session 1 ($n = 14$)

$^{\#}$ $P < 0.01$, $^{\$}$ $P < 0.001$ when compared with training session 1

power analysis (Altman 1991) to confirm if the magnitude of change in $HRR_{40\text{-km}}$ could be considered real. This calculation revealed a 100% power at a 5% significant level, which confirms that we were not making a type II error. These findings suggest that relatively small changes in recovery heart rate can be measured in a group of already well-trained cyclists. However, this is only true if the same level of control is used when performing the testing protocol.

$HRR_{40\text{-km}}$ and change in performance parameters

The second important finding of this study was that the improvements in $HRR_{40\text{-km}}$ correlate well with some improvements in performance parameters. For example, about 93% of the variation in the improvements in $HRR_{40\text{-km}}$ ($r = 0.96$) could be accounted for by the variation in the improvement in 40-km TT time. A weaker correlation was found between the variation in the improvements in PPO,

which could only account for about 54% of the change in $HRR_{40\text{-km}}$ ($r = 0.73$). The stronger correlation between $HRR_{40\text{-m}}$ and 40-km TT time might be explained by the fact that $HRR_{40\text{-km}}$ was directly measured after the 40-km TT and is therefore more closely related to 40-km TT performance. These findings, are similar to a study which found a correlation ($r = 0.62$) between change in indices of HRR and improvements in repeated-sprint ability (Buchheit et al. 2008). The lack of relationship between change in $HRR_{40\text{-km}}$ and change in VO_{2max} is not novel (Buchheit and Gindre 2006), and confirms that VO_{2max} is of limited value in tracking or predicting athletic ability (Lamberts et al. 2008; Noakes 2008).

HIT and HRR_{HIT}

The third finding of this study was that HRR_{HIT} improved throughout the HIT sessions. The first significant differences in HRR_{HIT} (compared to the values of the first training session) occurred after the fifth HIT session (5 ± 5 beats) and kept on improving until the eighth and final HIT session (7 ± 6 beats).

$HRR_{40\text{-km}}$ and HRR_{HIT}

Although $HRR_{40\text{-km}}$ and HRR_{HIT} improved similarly over the training period, the HRR was faster after the HIT sessions when compared to the 40-km TT ($P = 0.0118$). This is in accordance with Kaikkonen et al. (2008) who recently reported a faster HRR after a similar interval training protocol (7 times 3 min exercise at 85% of VO_{2max}, with 2-min rest intervals) compared to a continuous protocol at the same intensity and duration.

Factors that have shown to influence autonomic nervous system response after exercise, measured through HRV, are the mode of exercise (Heffernan et al. 2006), the proceeding workload intensity (Kaikkonen et al. 2008) and duration (Seiler et al. 2007). As HRR after moderate to heavy exercise is a consequence of parasympathetic reactivation and sympathetic withdrawal (autonomic nervous system), HRR responses in this study might also have been influenced by the intensity at the end of the proceeding workload (after the interval training or 40-km TT). A recent study showed that recovery after maximal exercise is mainly caused by parasympathetic reactivation where sympathetic activation can even carry on into the early phases of recovery (Kannankeril et al. 2004). As subjects sprinted during the last kilometer of the 40-km TT, in an attempt to set the fastest possible time, sympathetic drive was further stimulated. Therefore, $HRR_{40\text{-km}}$ was measured from an intensity of 98 ± 2% of HR_{max}. This higher intensity compared to an intensity of 93 ± 2% of HR_{max} after HRR_{HIT} might also have contributed to the slower $HRR_{40\text{-km}}$ compared to the HRR_{HIT}. A third contributing factor might have been the duration of cycling at high intensities which was ±65 min for the 40-km TT and 32 min (8 × 4 min) per HIT session.

The objective of this study was to determine if changes in HRR were sufficiently sensitive to track changes in performance in already well-trained cyclists. As HRR after exercise is an easy and non-invasive method to collect data representing the functioning of the autonomic nervous system, and appears to be sensitive to direct changes in training markers, HRR can possible play an important role in prescribing and fine tuning training (Borresen and Lambert 2008; Buchheit et al. 2007; Lamberts and Lambert 2009). However, it is recommended that HRR needs to be measured after a standardised test, as factors of duration, intensity an exercise mode influence the rate of HRR.

In conclusion, this study shows that HRR changes in a group of already well-trained cyclists who improved their performance parameters after a period of HIT. The changes in $HRR_{40\text{-km}}$ were associated with changes in 40-km TT time (s) and relative peak power (W kg^{-1}). This relationship indicates that $HRR_{40\text{-km}}$ and possibly HRR measured after a standardised warming up period, may be a sensitive monitoring tool to track changes over time in performance parameters in already well-trained cyclists.

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References

Altman DG (1991) Practical statistics for medical research. Chapman and Hall, London

American College of Sports Medicine (2007) Preparticipation health screening and risk stratification. In: Whaley MH, Brubaker PH, Otto RM (eds) ACSM's guidelines for exercise testing and prescription. Lippincott Williams & Wilkins, Baltimore, p 26

Baumert M, Brechtel L, Lock J, Hermsdorf M, Wolff R, Baier V, Voss A (2006) Heart rate variability, blood pressure variability, and baroreflex sensitivity in overtrained athletes. Clin J Sports Med 16:412–417. doi:10.1097/01.jsm.0000244610.34594.07

Boas EP (1931) The heart rate of boys during and after exhausting exercise. J Clin Invest 10:145–152. doi:10.1172/JCI100335

Borresen J, Lambert MI (2007) Changes in heart rate recovery in response to acute changes in training load. Eur J Appl Physiol 101:503–511. doi:10.1007/s00421-007-0516-6

Borresen J, Lambert MI (2008) Autonomic control of heart rate during and after exercise—measurements and implications for monitoring training status. Sports Med 28:633–646. doi: 10.2165/00007256-200838080-00002

Buchheit M, Gindre C (2006) Cardiac parasympathetic regulation: respective associations with cardiorespiratory fitness and training load. Am J Physiol Heart Circ Physiol 291:H451–H458. doi: 10.1152/ajpheart.00008.2006

Buchheit M, Papelier Y, Laursen PB, Ahmaidi S (2007) Noninvasive assessment of cardiac parasympathetic function: postexercise

heart rate recovery or heart rate variability? Am J Physiol Heart Circ Physiol 293:H8–H10. doi:10.1152/ajpheart.00335.2007
Buchheit M, Millet GP, Parisy A, Pourchez S, Laursen PB, Ahmaidi S (2008) Supramaximal training and postexercise parasympathetic reactivation in adolescents. Med Sci Sports Exerc 40:362–371
Bunc V, Heller J, Leso J (1988) Kinetics of heart rate responses to exercise. J Sports Sci 6:39–48
Cole CR, Blackstone EH, Pashkow FJ, Snader CE, Lauer MS (1999) Heart-rate recovery immediately after exercise as a predictor of mortality. N Engl J Med 341:1351–1357. doi:10.1056/NEJM199910283411804
Davidson RCR, Corbett J, Ansley L (2007) Influence of temperature and protocol on the calibration of the computrainer electromagnetically braked cycling ergometer. J Sports Sci 25:257–258
Durnin JVGA, Womersley J (1974) Body fat assessed from total body density and its estimation from skinfold thickness: measurements on 481 men and women aged from 16 to 72 years. Br J Nutr 32:77–97. doi:10.1079/BJN19740060
Gnehm P, Reichenbach S, Altpeter E, Widmer H, Hoppeler H (1997) Influence of different racing positions on metabolic cost in elite cyclists. Med Sci Sports Exerc 29:818–823. doi:10.1097/00005768-199706000-00013
Hawley JA, Noakes TD (1992) Peak power output predicts maximal oxygen uptake and performance time in trained cyclists. Eur J Appl Physiol Occup Physiol 65:79–83. doi:10.1007/BF01466278
Hedelin R, Kenttä G, Wiklund U, Bjerle P, Henriksson-Larsen K (2000) Short-term overtraining: effects on performance, circulatory responses, and heart rate variability. Med Sci Sports Exerc 32:1480–1484. doi:10.1097/00005768-200008000-00017
Heffernan KS, Kelly EE, Collier SR, Fernhall B (2006) Cardiac autonomic modulation during recovery from acute endurance versus resistance exercise. Eur J Cardiovasc Prev Rehabil 13:80–86. doi:10.1097/00149831-200602000-00012
Jeukendrup A (2002) High-performance cycling. Human Kinetics Publishers, Inc, Champaign
Jeukendrup AE, Craig NP, Hawley JA (2000) The bioenergetics of world class cycling. J Sci Med Sport 3:414–433. doi:10.1016/S1440-2440(00)80008-0
Kaikkonen P, Rusko H, Martinmaki K (2008) Post-exercise heart rate variability of endurance athletes after different high-intensity exercise interventions. Scand J Med Sci Sports 18:511–519
Kannankeril PJ, Le FK, Kadish AH, Goldberger JJ (2004) Parasympathetic effects on heart rate recovery after exercise. J Investig Med 52:394–401. doi:10.2310/6650.2004.00611
Kenttä G, Hassmen P (1998) Overtraining and recovery. A conceptual model. Sports Med 26:1–16. doi:10.2165/00007256-199826010-00001
Kiviniemi AM, Hautala AJ, Kinnunen H, Tulppo MP (2007) Endurance training guided individually by daily heart rate variability measurements. Eur J Appl Physiol 101:743–751. doi:10.1007/s00421-007-0552-2
Kuipers H (1998) Training and overtraining: an introduction. Med Sci Sports Exerc 30:1137–1139. doi:10.1097/00005768-199807000-00018
Lambert MI, Borresen J (2006) A theoretical basis of monitoring fatigue: a practical approach for coaches. Int J Sports Sci Coaching 1:371–388. doi:10.1260/174795406779367684
Lamberts RP, Lambert MI (2009) Day-to-day variation in heart rate at different levels of submaximal exertion: implications for monitoring training. J Strength Cond Res (in press)
Lamberts RP, Lemmink KA, Durandt JJ, Lambert MI (2004) Variation in heart rate during submaximal exercise: implications for monitoring training. J Strength Cond Res 18:641–645. doi:10.1519/1533-4287(2004)18<641:VIHRDS>2.0.CO;2
Lamberts RP, Swart J, Woolrich RW, Noakes TD, Lambert MI (2008) Measurement error associated with performance testing in well-trained cyclists; application to the precision of monitoring changes in training status. Int Sports Med J (in press)
Laursen PB, Jenkins DG (2002) The scientific basis for high-intensity interval training: optimising training programmes and maximising performance in highly trained endurance athletes. Sports Med 32:53–73. doi:10.2165/00007256-200232010-00003
Laursen PB, Shing CM, Peake JM, Coombes JS, Jenkins DG (2002) Interval training program optimization in highly trained endurance cyclists. Med Sci Sports Exerc 34:1801–1807. doi:10.1097/00005768-200211000-00017
Lehmann MJ, Lormes W, Opitz-Gress A, Steinacker JM, Netzer N, Foster C, Gastmann U (1997) Training and overtraining: an overview and experimental results in endurance sports. J Sports Med Phys Fitness 37:7–17
Lehmann M, Foster C, Dickhuth HH, Gastmann U (1998) Autonomic imbalance hypothesis and overtraining syndrome. Med Sci Sports Exerc 30:1140–1145. doi:10.1097/00005768-199807000-00019
Lucia A, Hoyos J, Santalla A, Perez M, Chicharro JL (2002a) Kinetics of VO_2 in professional cyclists. Med Sci Sports Exerc 34:320–325. doi:10.1097/00005768-200203000-00021
Lucia A, Rivero JL, Perez M, Serrano AL, Calbet JA, Santalla A, Chicharro JL (2002b) Determinants of VO_2 kinetics at high power outputs during a ramp exercise protocol. Med Sci Sports Exerc 34:326–331. doi:10.1097/00005768-200203000-00021
Meeusen R, Duclos M, Gleeson M, Rietjens G, Steinacker J, Urhausen A (2006) Prevention, diagnosis and treatment of the overtraining syndrome. Eur J Sport Sci 6:1–14. doi:10.1080/17461390600617717
Mujika I, Padilla S (2001) Physiological and performance characteristics of male professional road cyclists. Sports Med 31:479–487. doi:10.2165/00007256-200131070-00003
Noakes TD (2008) Testing for maximum oxygen consumption has produced a brainless mode of human exercise performance. Br J Sports Med 42:551–555. doi:10.1136/bjsm.2008.046821
Padilla S, Mujika I, Orbananos J, Angulo F (2000) Exercise intensity during competition time trials in professional road cycling. Med Sci Sports Exerc 32:850–856. doi:10.1097/00005768-200004000-00019
Ross WD, Marfell-Jones MJ (1991) Kinanthropometry. In: MacDougall JD, Wenger HA, Green HS (eds) Physiological testing of the high performance athlete. Human Kinetics, Champaign, pp 223–308
Seiler S, Haugen O, Kuffel E (2007) Autonomic recovery after exercise in trained athletes: intensity and duration effects. Med Sci Sports Exerc 39:1366–1373. doi:10.1249/mss.0b013e318060f17d
Shepley B, MacDougall JD, Cipriano N, Sutton JR, Tarnopolsky MA, Coates G (1992) Physiological effects of tapering in highly trained athletes. J Appl Physiol 72:706–711
Shetler K, Marcus R, Froelicher VF, Vora S, Kalisetti D, Prakash M, Myers J, Do D (2001) Heart rate recovery: validation and methodologic issues. J Am Coll Cardiol 38:1980–1987. doi:10.1016/S0735-1097(01)01652-7
Short KR, Sedlock DA (1997) Excess postexercise oxygen consumption and recovery rate in trained and untrained subjects. J Appl Physiol 83:153–159
Solberg G, Robstad B, Skjønsberg OH, Borchsenius F (2005) Respiratory gas exchange indices for estimating the anaerobic threshold. J Sports Sci Med 4:29–36
Stepto NK, Hawley JA, Dennis SC, Hopkins WG (1999) Effects of different interval-training programs on cycling time-trial performance. Med Sci Sports Exerc 31:736–741. doi:10.1097/00005768-199905000-00018

Sugawara J, Murakami H, Maeda S, Kuno S, Matsuda M (2001) Change in post-exercise vagal reactivation with exercise training and detraining in young men. Eur J Appl Physiol 85:259–263. doi:10.1007/s004210100443

Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology (1996) Heart rate variability: standards of measurement, physiological interpretation and clinical use. Circulation 93:1043–1065

Yamamoto K, Miyachi M, Saitoh T, Yoshioka A, Onodera S (2001) Effects of endurance training on resting and post-exercise cardiac autonomic control. Med Sci Sports Exerc 33:1496–1502. doi:10.1097/00005768-200109000-00012