Eur J Appl Physiol (2013) 113:371–384
DOI 10.1007/s00421-012-2445-2

ORIGINAL ARTICLE

Consecutive days of cold water immersion: effects on cycling performance and heart rate variability

Jamie Stanley · Jonathan M. Peake · Martin Buchheit

Received: 22 February 2012 / Accepted: 14 June 2012 / Published online: 3 July 2012

Abstract We investigated performance and heart rate (HR) variability (HRV) over consecutive days of cycling with post-exercise cold water immersion (CWI) or passive recovery (PAS). In a crossover design, 11 cyclists completed two separate 3-day training blocks (120 min cycling per day, 66 maximal sprints, 9 min time trialling [TT]), followed by 2 days of recovery-based training. The cyclists recovered from each training session by standing in cold water (10 °C) or at room temperature (27 °C) for 5 min. Mean power for sprints, total TT work and HR were assessed during each session. Resting vagal-HRV (natural logarithm of square-root of mean squared differences of successive *R–R* intervals; ln rMSSD) was assessed after exercise, after the recovery intervention, during sleep and upon waking. CWI allowed better maintenance of mean sprint power (between-trial difference [90 % confidence limits] +12.4 % [5.9; 18.9]), cadence (+2.0 % [0.6; 3.5]), and mean HR during exercise (+1.6 % [0.0; 3.2]) compared with PAS. ln rMSSD immediately following CWI was higher (+144 % [92; 211]) compared with PAS. There was no difference between the trials in TT performance (−0.2 % [−3.5; 3.0]) or waking ln rMSSD (−1.2 % [−5.9; 3.4]). CWI helps to maintain sprint performance during consecutive days of training, whereas its effects on vagal-HRV vary over time and depend on prior exercise intensity.

Keywords Autonomic nervous system · Training block · High-intensity cycling · Sprints · Post-exercise recovery · Hydrotherapy

Abbreviations

CI	Confidence interval
CWI	Cold water immersion
ES	Effect size
HR	Heart rate
$HR_{(ave)}$	Mean heart rate
$HR_{(peak)}$	Peak heart rate
$HR_{(post\text{-}recovery)}$	Heart rate immediately following the recovery intervention
$HR_{(post\text{-}session)}$	Heart rate immediately following the laboratory training session
$HR_{(wake)}$	Heart rate upon waking
HRV	Heart rate variability
ln rMSSD	Natural logarithm of the square-root of mean squared differences of successive *R–R* intervals
ln $rMSSD_{(post\text{-}session)}$	ln rMSSD immediately following the laboratory training session
ln $rMSSD_{(post\text{-}recovery)}$	ln rMSSD immediately following the recovery intervention
ln $rMSSD_{(sleep)}$	ln rMSSD during estimated slow wave sleep
ln $rMSSD_{(wake)}$	ln rMSSD upon waking
PAS	Passive recovery

Communicated by Narihiko Kondo.

J. Stanley · J. M. Peake
Centre of Excellence for Applied Sport Science Research, Queensland Academy of Sport, Brisbane, Australia

J. Stanley (✉)
School of Human Movement Studies, The University of Queensland, Brisbane, QLD 4072, Australia
e-mail: j.stanley@uq.edu.au

J. M. Peake
School of Biomedical Sciences, Queensland University of Technology, Brisbane, Australia

M. Buchheit
Physiology Unit, Sport Science Department, Aspire, Academy for Sports Excellence, Doha, Qatar

PPO	Peak power output
RPE	Rating of perceived exertion
TT	Time-trial
VO_{2peak}	Peak oxygen uptake

Introduction

Elite athletes often train intensely or compete over consecutive days. Cumulative fatigue over such periods of training or competition can reduce athletic performance (Lane and Wenger 2004; Vaile et al. 2008). Adequate recovery between training sessions and/or competitive events is therefore essential to minimise the risk of fatigue and optimise performance.

Often the duration between training sessions or competition events does not allow for complete recovery. To maintain performance, athletes commonly adopt strategies such as massage, active recovery, compression garments and hydrotherapy to accelerate their recovery. Post-exercise cold water immersion (CWI) is a popular form of hydrotherapy and can improve recovery and exercise performance during consecutive days (Lane and Wenger 2004; Vaile et al. 2008). CWI may benefit recovery by altering blood flow (Vaile et al. 2010), thermoregulation (Peiffer et al. 2009; Vaile et al. 2010), and perceptions of recovery (Stanley et al. 2012) which may be reflected by changes in cardiac autonomic activity (Buchheit et al. 2009b; Stanley et al. 2012).

Recording cardiac autonomic activity is an emerging tool to monitor both long-term training adaptations (Borresen and Lambert 2008) and acute recovery from prior exercise (Hautala et al. 2001; James et al. 2002; Kiviniemi et al. 2007). In the long term, increased cardiac parasympathetic activity is generally observed during recovery-based training phases (Pichot et al. 2000) and shows large correlations with changes in athletic performance (Buchheit et al. 2010). However, during prolonged training interventions (Iwasaki et al. 2003) and/or in elite athletes (Iellamo et al. 2002), changes in cardiac autonomic activity may become dissociated from changes in performance. In the short term, changes in cardiac parasympathetic activity after exercise appear to track the time course of restoration of homeostasis (Hautala et al. 2001). Changes in cardiac parasympathetic activity have emerged as a global recovery index that reflects the acute response of the body to exercise. For example, cardiac parasympathetic activity decreases for several hours after exercise (Al Haddad et al. 2009; Myllymäki et al. 2012; Seiler et al. 2007; Stanley et al. 2012) and with adequate recovery, can return to pre-exercise levels during the following days (James et al. 2002; Mourot et al. 2004), or 'rebound' above these levels (Buchheit et al. 2009a; Hautala et al. 2001). This response is likely dependent on training load (exercise duration and intensity, post-exercise blood lactate concentration and ratings of perceived exertion) (Buchheit et al. 2007; Myllymäki et al. 2012; Seiler et al. 2007), age (Sandercock et al. 2005) and training status (Buchheit and Gindre 2006). Therefore, while cardiac autonomic control may not play an active role in the recovery process, its kinetics following exercise may mirror training adaptation responses, as described by the supercompensation theory. The success of training programs guided by changes in daily cardiac parasympathetic (i.e., prescription of high-intensity training only when cardiac parasympathetic activity has returned to a high level) (Kiviniemi et al. 2007) highlights the potential importance of this rebound effect.

The substantial effect of post-exercise CWI on immediate (Buchheit et al. 2009b; Stanley et al. 2012) and delayed (Al Haddad et al. 2011b) cardiac parasympathetic activity may have contrasting implications for recovery and training adaptation. Increased cardiac parasympathetic activity is evident prior to sleep onset (Burgess et al. 1997) and is likely associated with improved sleep quality (Shinar et al. 2006). Therefore, strategies to accelerate sleep onset and/or increase cardiac parasympathetic activity, such as CWI (Stanley et al. 2012), may enhance recovery between exercise sessions (Halson 2008). In elite swimmers, both resting cardiac parasympathetic activity and perceived sleep quality are improved following repeated CWI during an intense training week (Al Haddad et al. 2011b). By contrast, however, the magnitude of cardiac parasympathetic suppression immediately after exercise is reduced with CWI (Buchheit et al. 2009b; Stanley et al. 2012). The response may attenuate the rebound in cardiac parasympathetic activity in the following days (i.e., lower stimuli, lower response), which might reflect compromised readiness to perform and/or limited training adaptations. Until now, the selective effect of CWI on physiological and perceptual recovery, sleep quality, performance and cardiac autonomic activity, during and following a short training block where CWI is used consistently has remained unknown.

The primary aim of this study was to compare the magnitude of the effects of CWI versus passive recovery on high-intensity cycling performance, sleep onset latency, perceptions of recovery and cardiac parasympathetic activity over a 3-day training block. A secondary aim was to compare the magnitude of the effects of CWI versus passive recovery on the time course of changes in cardiac parasympathetic activity in the days following the training block, as a proxy measure of the overall training adaptation/response to the training block.

Methods

Subjects

Eleven endurance-trained male cyclists volunteered to participate in this study [age 27 ± 6 years, body mass

73.4 ± 8.2 kg, height 1.76 ± 0.06 m, peak oxygen uptake ($\dot{V}O_{2peak}$) 64.8 ± 6.0 mL kg^{-1} min^{-1}, peak power output (PPO) 415 ± 39 W]. At the time the study was conducted, six cyclists were in their pre-competition phase, whereas the other five cyclists were at the end of their competitive season. For the duration of the study, the cyclists spent approximately 10–15 h week^{-1} training (including the study requirements) or competing. Prior to the study, all of the cyclists completed a medical screening questionnaire and gave their written informed consent. The experimental procedure was in accordance with the Declaration of Helsinki, and was approved by the Human Research Ethics Committee at The University of Queensland.

Preliminary testing

$\dot{V}O_{2peak}$ was measured during an incremental exercise test on an electronically braked cycle ergometer (Schoberer Rad Meßtechnik SRM GmbH, Jülich, Germany). The initial workload of 100 W was sustained for 5 min; thereafter, the workload increased by 25 W every 1 min until volitional fatigue. Gas exchange was measured throughout the entire test (Moxus Modular $\dot{V}O_2$ System, AEI Technologies, Pittsburg, Pennsylvania, USA). Heart rate (HR) was recorded using telemetry (Suunto t6c, Suunto Oy, Vantaa, Finland). PPO was calculated as the power output from the last completed stage of the incremental exercise test, plus the fraction of time spent in the next stage multiplied by 25 W. $\dot{V}O_{2peak}$ was considered as the highest $\dot{V}O_2$ attained over a 30-s period during the incremental test.

Experimental protocol

The study was completed over a 5-week period. An overview of the study design is provided in Fig. 1. To minimise any learning or training effects, the cyclists completed a familiarisation trial (within 2 days of completing the incremental exercise test) where they attended the laboratory and followed all procedures used in the experimental trials for two consecutive days. Eleven days following the familiarisation trial, the cyclists completed two experimental trials in a randomised, counterbalanced, crossover design separated by 11 days. On the day preceding each experimental trial, the cyclists completed light training (i.e., cycling 60–90 min/25–50 km, average HR < 60 %max). Each experimental trial involved completing a laboratory-based training session and a specific recovery intervention on three consecutive days (training block), followed by two consecutive days of recovery-based training (recovery block, 60–90 min/25–50 km, average HR < 60 %max on the first day, and 60–150 min/25–75 km, average HR < 75 %max on the second day). HR was recorded during the cycling protocol, immediately following the cycling protocol, following each recovery intervention, and during sleep each night following the laboratory training session. HR was also recorded upon waking on each day of the training block and for the following 3 days. To ensure that the cyclists commenced each experimental trial in a similar physiological state, food and training diaries were kept throughout the 5-week study period. The cyclists were asked to match food intake, training volume and intensity during the weeks that separated the laboratory sessions. The cyclists refrained from caffeine and alcohol for 7 days commencing 24 h prior to the first laboratory session of each trial. On both days of the familiarisation trial, the cyclists were allowed to consume water ad libitum during the training session, and this volume was recorded. During the experimental trials, the cyclists were provided this same volume of water and consumed it ad libitum during the training session. Following the training session, the cyclists were not allowed to drink or eat until all measurements following the recovery intervention were completed.

Laboratory training sessions

The cyclists arrived at the laboratory between 5:10 pm and 5:40 pm, and commenced the training session between 5:30 pm and 6:00 pm. The laboratory training sessions began with a cycling protocol (adapted from Vaile et al. 2008) that simulated the demands of cycle racing and provided an indication of repeat performance capabilities. All laboratory training was completed on air- and magnetically braked cycling ergometers (Wattbike Ltd., Nottinghamshire, UK). The cycling protocol was 120 min in duration and included 66 maximal effort sprints and three time trials, totalling 9 min of sustained

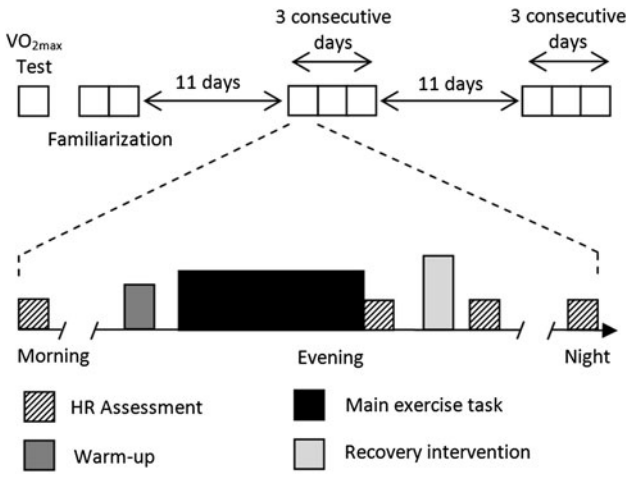

Fig. 1 Schematic of the overall study design (*top*) and intensive training day (*bottom*)

effort (Table 1). The cyclists were instructed to complete as much work as possible during each 2- or 5-min time-trial. The cyclists were not given any information about power output or work completed during the time trials. For all other periods during the cycling protocol, the cycle ergometer computer unit provided instantaneous feedback on power and cadence. Throughout each exercise session of the training blocks, strong verbal encouragement and support was provided to the cyclists in a consistent manner by the same person. Prior to each sprint or time-trial, the same instruction sequence (10- and 5-s warnings preceded the "and…go!" command) was provided to ensure readiness for the upcoming effort. During all trials, a 460-mm pedestal fan (Sampford IXL, Australia) was placed in front of each cyclist while on the cycle ergometer. Laboratory temperature during the training sessions was 23.9 ± 0.5 °C and relative humidity was 73 ± 5 %. Immediately after finishing each exercise session, the cyclists rested quietly for approximately 15 min on chairs positioned behind the cycle ergometers.

Table 1 Laboratory training session including the main exercise task

10-min warm-up (self-selected) + 5-min steady-state
Sprint set 1–12 × 5 s, 1:6 (*W:R*)
Sprint set 2–12 × 5 s, 1:3 (*W:R*)
Sprint set 3–12 × 5 s, 1:1 (*W:R*)
4-min active recovery
2-min TT
4-min active recovery
Sprint set 4–6 × 10 s, 1:6 (*W:R*)
Sprint set 5–6 × 10 s, 1:3 (*W:R*)
Sprint set 6–6 × 10 s, 1:1 (*W:R*)
4-min active recovery
2-min TT
4-min active recovery
Sprint set 7–6 × 15 s, 1:6 (*W:R*)
Sprint set 8–6 × 15 s, 1:3 (*W:R*)
Sprint set 9–6 × 15 s, 1:1 (*W:R*)
5-min active recovery
5-min TT
5-min active recovery

Between the 8th and 10th min of the 10 min warm-up, cyclists were guided through three 3-s sprints at 70, 80, and 90 % then transitioned immediately into 5 min at steady-state of 80 % PPO. 6 min of passive (seated) recovery separated the warm-up and first sprint set. The first sprint of sprint set 1 was commenced from a standing start. Between each sprint set cyclists completed 5 min of active recovery (40–50 % PPO)

W:R work:rest, *TT* time-trial, *PPO* peak power output

Recovery interventions

Approximately 15 min after the cycling protocol, the cyclists completed one of two recovery interventions in a purpose-built hydrotherapy recovery centre. Ambient air temperature within the centre was maintained at 27 °C. In the passive recovery/control trial (PAS), the cyclists remained standing at room temperature for 5 min. In the CWI trial, the cyclists immersed their entire body vertically (excluding head and neck) in 10.1 ± 0.8 °C water for 5 min. We elected to use CWI because of its beneficial effect on exercise performance during consecutive days (Vaile et al. 2008). The specific immersion protocol was selected because this duration and temperature are effective for restoring cardiac parasympathetic activity after exercise (Stanley et al. 2012), while remaining tolerable for athletes (Peiffer et al. 2009). During both recovery interventions, the cyclists assumed similar posture, and limited movement. The cyclists then towel dried and returned to the laboratory for final measurements within 5 min.

Heart rate measurements

HR was assessed upon waking (wake), after the training session (post-session), following the recovery intervention (post-recovery), and during the night (sleep). After waking in the morning of the laboratory training sessions, and for the 3 days following, the cyclists voided their bladder then returned to bed to record $HR_{(wake)}$ in a supine position for 10 min (Kiviniemi et al. 2007). During all resting HR recordings, the cyclists were asked to remain still and not to talk. Immediately following the cycling protocol, the cyclists dismounted the bicycle ergometer and sat passively on an adjacent chair (within 5 s) for 5 min to record $HR_{(post\text{-}session)}$. Likewise, after the recovery intervention, the cyclists returned to the laboratory and sat passively for 5 min to record $HR_{(post\text{-}recovery)}$. Respiratory rate was spontaneous for practicality during the home-based measurements, and because there is little difference in parasympathetic-related HRV indices during controlled or spontaneous breathing (Bloomfield et al. 2001). $HR_{(sleep)}$ was recorded while sleeping each night following the laboratory training sessions. Bed time was not standardised so that we could assess whether the recovery interventions influenced the cyclists' sleep habits.

For all resting HR recordings, *R–R* intervals were recorded continuously with a Suunto t6c wrist top computer (Suunto Oy, Vantaa, Finland) at a sampling frequency of 1,000 Hz. During the cycling protocol, exercise HR was recorded directly on a PC using a Suunto PC Pod (Suunto Oy, Vantaa, Finland). To ensure quality of *R–R* signal, the cyclists applied electrode gel (Lectron II, Pharmaceutical Innovations Inc., New Jersey, USA) to the HR chest strap prior to each recording.

HR data analysis

R–R interval data files were transferred to the computer using Suunto Team Manager software (Suunto Oy, Vantaa, Finland) (Kaikkonen et al. 2010). Further signal processing was performed using a dedicated HRV analysis program (Kubios HRV Analysis version 2.0 beta 1, The Biomedical Signals Analysis Group, University of Kuopio, Finland). An experienced investigator visually identified and manually removed any occasional ectopic beats and artefacts. Although removing artefacts may potentially disrupt the rhythm of the signal (and potentially alter the spectral energy or overall variability) we consider that this approach provides greater certainty that the remaining data are physiologically 'real' compared with extrapolation. The natural logarithm of the square-root mean of the sum of the squared differences between adjacent normal *R–R* intervals (ln rMSSD) was calculated to provide an index of cardiac parasympathetic activity (Task-Force 1996). Analysis was restricted to ln rMSSD because time domain indices are more reliable under ambulatory trials of variable respiration rate compared with power spectral analysis techniques (Al Haddad et al. 2011a). ln $rMSSD_{(wake)}$ was calculated from the last 5 min of the 10-min morning recordings (Task-Force 1996), ln $rMSSD_{(post\text{-}session)}$ and ln $rMSSD_{(post\text{-}recovery)}$ from the last 3 min of the 5-min (seated) recordings (Buchheit et al. 2007). Sleep onset latency was visually determined using both HR and HRV trends within the first hour after bed time (Shinar et al. 2006). Two experienced investigators independently assessed sleep onset latency. If there was any discrepancy, both investigators reassessed the data until they agreed on the sleep onset latency. ln $rMSSD_{(sleep)}$ was calculated from the most stationary 5 min of the first period of slow wave sleep as described elsewhere (Brandenberger et al. 2005).

Subjective measures

During each laboratory training session, the cyclists rated their perceived exertion (RPE) using Borg's scale of 6 (no exertion) to 20 (maximal exertion) at the end of each sprint set and at the end of the 5-min time-trial (Vaile et al. 2008). Tiredness was rated on a scale of 1 (not tired at all) to 5 (very tired) immediately prior to going to bed each night following a laboratory training session. Perceptions of general fatigue, mental recovery, and leg soreness were rated on a scale of 1 (minimal) to 10 (maximal) (Stanley et al. 2012) immediately following the waking HRV recording each morning following a laboratory training session (i.e., days 2–4).

Data analysis

Data were assessed for practical significance using magnitude-based inferences (Hopkins et al. 2009). We used this qualitative approach because traditional statistical approaches often do not indicate the magnitude of an effect, which is typically more relevant to athletic performance than any statistically significant effect. For any variables that included data for fewer than 11 of the cyclists, the number of data sets is noted with the relevant result. All data were log-transformed prior to analysis to reduce bias arising from non-uniformity of error. Performance, HR and HRV indices before CWI, and RPE for day 1 were compared separately between trials to establish that the cyclists were in a similar state at the beginning of the two trials. Perceptions of recovery (general fatigue, mental recovery, leg soreness) on day 2 were compared separately between trials to determine the initial effect of each recovery intervention. Baseline (pre-experimental trial) characteristics were included as a covariate for between- and within-trial analysis. PPO was used as a covariate for performance variables and age was used as a covariate for HRV responses due to the effect of age on baseline HRV and the training-induced HRV response (Sandercock et al. 2005).

For each variable, we calculated within-trial changes during the 3-day training block using within-subject modelling (Hopkins 2010). This approach involved calculating a slope for the change from days 1–3, and using this slope to predict a change score for the final day. We then compared these predicted change scores between the two trials. Using this approach, we also calculated within-trial changes for waking HR and HRV over the 3-day training block and over the three mornings of the recovery block (i.e., over the 6 consecutive days).

To compare within-trial changes between trials (i.e., between-recovery conditions effect), we used a modified statistical spreadsheet (Hopkins 2006). This spreadsheet calculates the between-trial standardised differences or effect sizes (ES, 90 % confidence interval [CI]) using the pooled standard deviation (Cohen 1988). Threshold values for ES statistics were $\leq$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). In addition, we calculated probabilities to establish whether the true (unknown) differences were lower, similar or higher than the smallest worthwhile change or difference. The smallest worthwhile change/difference for time-trial and sprint performance was set at 1 % because this represents the smallest worthwhile enhancement for cyclists competing in track and time-trial events (Paton and Hopkins 2006). For all other variables, the smallest worthwhile change/difference was calculated as 0.2 multiplied by the between-subject standard deviation based on Cohen's effect size principle (Cohen 1988). Quantitative chances of higher or lower differences were evaluated qualitatively as follows: <1 % almost certainly not, 1–5 % very unlikely, 5–25 % unlikely, 25–75 % possible, 75–95 % likely, 95–99 % very

likely, >99 % almost certain. If the chance of higher or lower differences was >5 %, the true difference was assessed as 'unclear'. Effect size statistics were also used to determine the magnitude of difference in power output within each session between CWI and PAS trials for each set of sprints and time trials during corresponding sessions. Pearson's product–moment correlation analysis was used to compare the association between individual changes in HR-derived indices, individual changes in performance variables, and individual changes in subjective measures. Individual responses were calculated as a percentage of the individual's mean response on comparable days for each trial [e.g., an individual's mean ln $rMSSD_{(wake)}$ for CWI and PAS on day 2]. Pearson's product–moment correlation analysis was also used to compare the association between the effect of CWI versus PAS recovery on the changes in the different variables [e.g., the difference in the changes in sprint power output between CWI and PAS trials vs. the difference in the change in ln $rMSSD_{(post\text{-}recovery)}$ on the preceding day between CWI and PAS trials]. The following criteria were adopted to interpret the magnitude of the correlation (r) between test measures: ≤0.1 trivial, >0.1–0.3 small, >0.3–0.5 moderate, >0.5–0.7 large, >0.7–0.9 very large, and >0.9–1.0 almost perfect. If the 90 % CI overlapped small positive and negative values, the magnitude of the correlation was deemed 'unclear'; otherwise, the magnitude of the correlation was deemed to be the observed magnitude (Hopkins et al. 2009).

Results

Performance

As expected, there were no substantial or clear differences between trials (CWI vs. PAS) on day 1 for performance, HR or perceived exertion during the laboratory session (Table 2). Changes in performance variables for the duration of the experimental trials are illustrated in Fig. 2. Differences between trials for within-trial changes in performance variables are depicted in Fig. 3a. The decrease in sprint power was almost certainly smaller from days 1 to 3 of the CWI trial compared with the PAS trial (within-trial change ±90 % confidence limit, +2.4 ± 2.3 vs. −9.6 ± 5.0 % for CWI vs. PAS, chances that the true difference was higher/trivial/lower, 99/1/0 %). Sprint cadence decreased from days 1 to 3 in both trials but the decrease was likely smaller during the CWI trial (−2.1 ± 1.5 vs. −4.1 ± 1.8 % for CWI vs. PAS, 94/6/0 %). No clear difference between trials for changes in time-trial performance (−0.6 ± 2.0 vs. −0.4 ± 2.1 % for CWI vs. PAS, 25/41/34 %) was observed from days 1 to 3. There was a possibly greater decrease in time-trial cadence from days 1 to 3 in the CWI trial compared with the PAS trial (−0.4 ± 1.3 vs. +0.4 ± 2.1 % for CWI vs. PAS, 4/25/71 %). Mean HR [$HR_{(ave)}$] during the cycling protocol decreased from days 1 to 3 in both trials, but the decrease was possibly smaller during the CWI trial compared with the PAS trial (−2.3 ± 1.3 vs. −3.9 ± 1.4 % for CWI vs. PAS, 86/12/2 %). There was no clear difference between trials for changes in peak HR [$HR_{(peak)}$] during exercise (−0.9 ± 1.1 vs. −2.0 ± 0.5 % for CWI vs. PAS, 87/7/6 %) from days 1 to 3. Within each training session, differences in power output between the CWI trial and the PAS trial were greater at the start of the session, where the shorter duration sprints occurred (i.e., 5- or 10-s sprints, sprints sets 1–6; Fig. 4).

Sleep onset latency

Sleep onset latency was not different between trials on day 1 (Table 2). There was also no clear difference between trials for changes in sleep onset latency (+12.7 ± 18.5 vs. +10.6 ± 36.1 % for CWI vs. PAS, 29/48/23 %) from days 1 to 3 (Fig. 3b).

HRV measures

There were no clear differences in ln $rMSSD_{(wake)}$, $HR_{(wake)}$, ln $rMSSD_{(post\text{-}session)}$, and $HR_{(post\text{-}session)}$ between trials on day 1 (Table 2). However, ln $rMSSD_{(post\text{-}recovery)}$ was almost certainly higher, $HR_{(post\text{-}recovery)}$ was almost certainly lower, and ln $rMSSD_{(sleep)}$ was likely lower in the CWI trial compared with the PAS trial on day 1 (Table 2). Figure 5 illustrates the time course of cardiac parasympathetic activity for the duration of each trial. Differences between trials for changes in HRV measures are depicted in Fig. 3b. The increase in ln $rMSSD_{(post\text{-}recovery)}$ was likely smaller from days 1 to 3 in the CWI trial compared with the PAS trial (+3.2 ± 8.1 vs. +15.2 ± 6.7 % for CWI vs. PAS, 1/6/93 %) (Fig. 3b). There were no clear differences between the trials for changes in ln $rMSSD_{(post\text{-}session)}$ (+25.7 ± 10.2 vs. +28.5 ± 18.2% for CWI vs. PAS, 59/33/8 %), $HR_{(post\text{-}session)}$ (−4.9 ± 3.8 vs. −7.1 ± 4.3 % for CWI vs. PAS, 59/33/8 %), $HR_{(post\text{-}recovery)}$ (−5.7 ± 2.7 vs. −6.3 ± 4.5 % for CWI vs. PAS, 34/46/20 %), and ln $rMSSD_{(sleep)}$ (+3.7 ± 3.9 vs. −0.3 ± 6.8 % for CWI vs. PAS, 72/15/13 %) from days 1 to 3. From days 1 to 6 (i.e., training block + recovery-based training) there were no clear differences between the trials for changes in ln $rMSSD_{(wake)}$ (+2.2 ± 1.4 vs. +3.2 ± 2.0 % for CWI vs. PAS, 21/22/57 %) and $HR_{(wake)}$ (−4.9 ± 4.8 vs. −6.9 ± 7.4 % for CWI vs. PAS, 46/42/12 %).

Subjective measures

There was no clear difference between trials in RPE or pre-sleep tiredness on day 1 (Table 2). There was also no clear

Table 2 Performance, physiological, and subjective measures on the first day of assessment during the cold water immersion (CWI) and passive recovery (PAS) trials

Variable	PAS	CWI	Standardized (Cohen) differences (90 % CL) for CWI versus PAS	% chances for CWI values to be higher/trivial/lower than PAS	Rating of the difference
ln $rMSSD_{(wake)}$ (ms)	4.3 ± 0.5	4.3 ± 0.3	+0.02 (−0.36; 0.40)	20/64/16	Unclear
$HR_{(wake)}$ (% HR_{max})	25.2 ± 3.4	24.6 ± 3.0	−0.16 (−0.57; 0.24)	6/50/44	Unclear
$Power_{(sprint)}$ (W)	638.5 ± 71.8	635.3 ± 94.8	−0.08 (−0.50; 0.34)	25/26/49	Unclear
$Cadence_{(sprint)}$ (rev min^{-1})	121 ± 5	123 ± 6	+0.25 (−0.54; 1.05)	55/29/16	Unclear
$Work_{(TT)}$ (kJ)	184.2 ± 27.4	180.9 ± 26.5	−0.10 (−0.27; 0.07)	6/27/67	Unclear
$Cadence_{(TT)}$ (rev min^{-1})	110 ± 5	110 ± 5	−0.01 (−0.38; 0.37)	17/64/19	Unclear
$HR_{(ave)}$ (% HR_{max})	80.1 ± 2.6	80.4 ± 3.5	+0.11 (−0.39; 0.60)	37/48/15	Unclear
$HR_{(peak)}$ (% HR_{max})	97.0 ± 2.8	97.3 ± 2.8	+0.08 (−0.18; 0.34)	21/75/4	*Likely trivial difference*
ln $rMSSD_{(post\text{-}session)}$ (ms)	2.3 ± 0.5	2.2 ± 0.2	−0.04 (−0.55; 0.47)	20/51/29	Unclear
$HR_{(post\text{-}session)}$ (% HR_{max})	48.6 ± 2.7	48.4 ± 3.4	−0.08 (−0.79; 0.64)	25/37/38	Unclear
RPE (6−20)	19 ± 1	19 ± 1	+0.19 (−0.36; 0.74)	49/40/11	Unclear
ln $rMSSD_{(post\text{-}recovery)}$ (ms)	2.9 ± 0.5	3.8 ± 0.5	+1.82 (1.32; 2.31)	100/0/0	*Almost certain large difference*
$HR_{(post\text{-}recovery)}$ (% HR_{max})	42.3 ± 3.2	33.7 ± 3.5	−2.76 (−3.57; −1.96)	0/0/100	*Almost certain very large difference*
Pre-sleep tiredness (1−5)	3 ± 1	3 ± 1	−0.20 (−1.01; .61)	19/31/50	Unclear
SOL (min)	8.0 ± 4.6	8.1 ± 3.2	+0.11 (−0.31; 0.52)	34/55/11	Unclear
ln $rMSSD_{(sleep)}$ (ms)	4.1 ± 0.2	3.9 ± 0.3	−0.53 (−0.79; −0.28)	1/2/97	*Likely small difference*
General Fatigue (1−10)	6 ± 2	5 ± 2	−0.19 (−0.68; 0.31)	9/43/48	Unclear
Mental Recovery (1−10)	5 ± 2	5 ± 2	+0.28 (−0.21; 0.76)	61/34/5	Unclear
Leg Soreness (1−10)	6 ± 2	5 ± 3	−0.49 (−0.97; −0.01)	1/14/85	*Likely small difference*

Values are mean ± SD for the natural logarithm of the square-root of the mean sum of the squares of differences between adjacent normal *R–R* intervals upon waking [ln $rMSSD_{(wake)}$], immediately following the session [ln $rMSSD_{(post\text{-}session)}$], and immediately following the recovery intervention [ln $rMSSD_{(post\text{-}recovery)}$], heart rate upon waking [$HR_{(wake)}$], peak [$HR_{(peak)}$] and mean [$HR_{(ave)}$] during the session, following the session [$HR_{(post\text{-}session)}$], following the recovery intervention [$HR_{(post\text{-}recovery)}$], sprint power output [$Power_{(sprint)}$], sprint cadence [$Cadence_{(sprint)}$], work performed during the time trials [$Work_{(TT)}$], time-trial cadence [$Cadence_{(TT)}$], mean rating of perceived exertion during the session (RPE), sleep onset latency (SOL) [$n = 11$ except for ln $rMSSD_{(wake)}$ and $HR_{(wake)}$ where $n = 9$, pre-sleep tiredness, SOL, general fatigue, mental recovery, and leg soreness where $n = 10$, and ln $rMSSD_{(sleep)}$ where $n = 4$]. See "Methods" for rating of differences

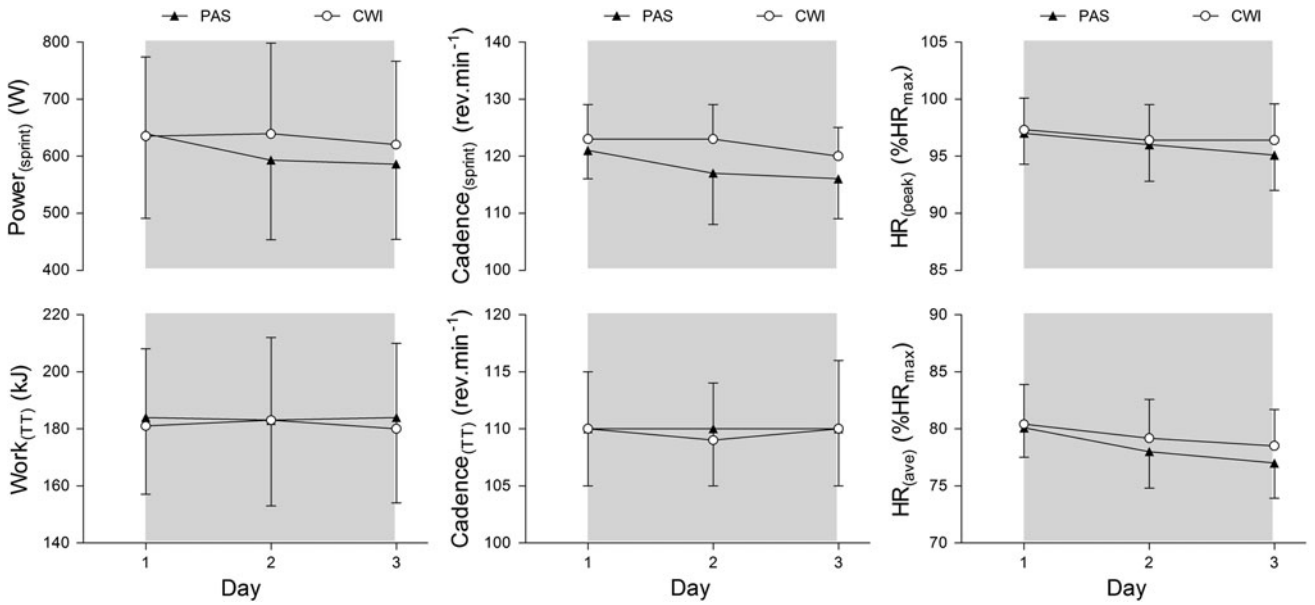

Fig. 2 Time course of performance variables during the 6-day experimental trials. *Shaded area* denotes the training block (laboratory training sessions). Data are presented at mean ± SD

difference between trials in general fatigue or mental recovery on day 2, but leg soreness was likely lower in the CWI trial compared with the PAS trial (Table 2). Changes in subjective measures of recovery for the duration of the experimental trials are depicted in Fig. 6. Differences between trials for within-trial changes in subjective measures are depicted in Fig. 3c. Despite substantially lower values on day 2 compared with the PAS trial (Table 2), there was likely a greater increase in leg soreness from days 2 to 4 of the CWI trial (+31.9 ± 52.2 vs. −10.2 ± 63.2 % for CWI vs. PAS, 87/13/0 %). There were no clear differences between trials for changes in general fatigue (+11.8 ± 36.6 vs. +17.6 ± 33.9 % for CWI vs. PAS, 13/54/13 %), or mental recovery (−11.9 ± 25.4 vs. −21.5 ± 40.0 % for CWI vs. PAS, 47/36/17 %) from days 2 to 4. There were also no clear differences for changes in RPE during exercise (+1.0 ± 1.5 vs. +2.4 ± 2.3 % for CWI vs. PAS, 10/29/61 %) or pre-sleep tiredness (+18.2 ± 15.1 vs. −0.8 vs. 26.4 %, 68/24/8 %) from days 1 to 3.

Correlations between variables

For the comparisons associating individual changes, when data from the training blocks of both trials were pooled, there was a large correlation between sprint performance and ln $rMSSD_{(post\text{-}recovery)}$ from the preceding day ($r = 0.51$ [90 % confidence limits, 0.30; 0.68], $n = 44$) (Fig. 7) and a moderate correlation between sprint performance and session $HR_{(peak)}$ ($r = 0.30$ [0.10; 0.48], $n = 66$). There was also a small correlation between sprint performance and session $HR_{(ave)}$ ($r = 0.29$ [0.09; 0.47], $n = 66$) and a small correlation between general fatigue and ln $rMSSD_{(post\text{-}recovery)}$ from the preceding day ($r = -0.25$ [−0.44; −0.03], $n = 60$). No other variables or comparisons associating the effect of CWI versus PAS recovery on the changes in the different variables clearly correlated with each other.

Discussion

In the present study we investigated how CWI affects high-intensity cycling performance and the recovery process during a short 3-day training block by assessing, sleep onset latency, cardiac parasympathetic activity (as a global recovery index), and subjective recovery. The primary finding was that CWI allowed better maintenance of sprint performance during the intensive training block compared with passive recovery. This beneficial effect of CWI occurred despite lower nocturnal cardiac parasympathetic activity and no effect on sleep onset latency or waking cardiac parasympathetic activity.

Sprint cycling performance—as indicated by higher average sprint power output—decreased less during the training block in the CWI trial compared with the passive recovery trial (Figs. 2, 3a). Better maintenance of power and cadence during the sprints in the CWI trial (Fig. 2) suggests faster recovery of neuromuscular function ~24 h after CWI. This notion is supported by the differences in power output (CWI trial vs. PAS trial) for each sprint set and time-trial observed during corresponding sessions

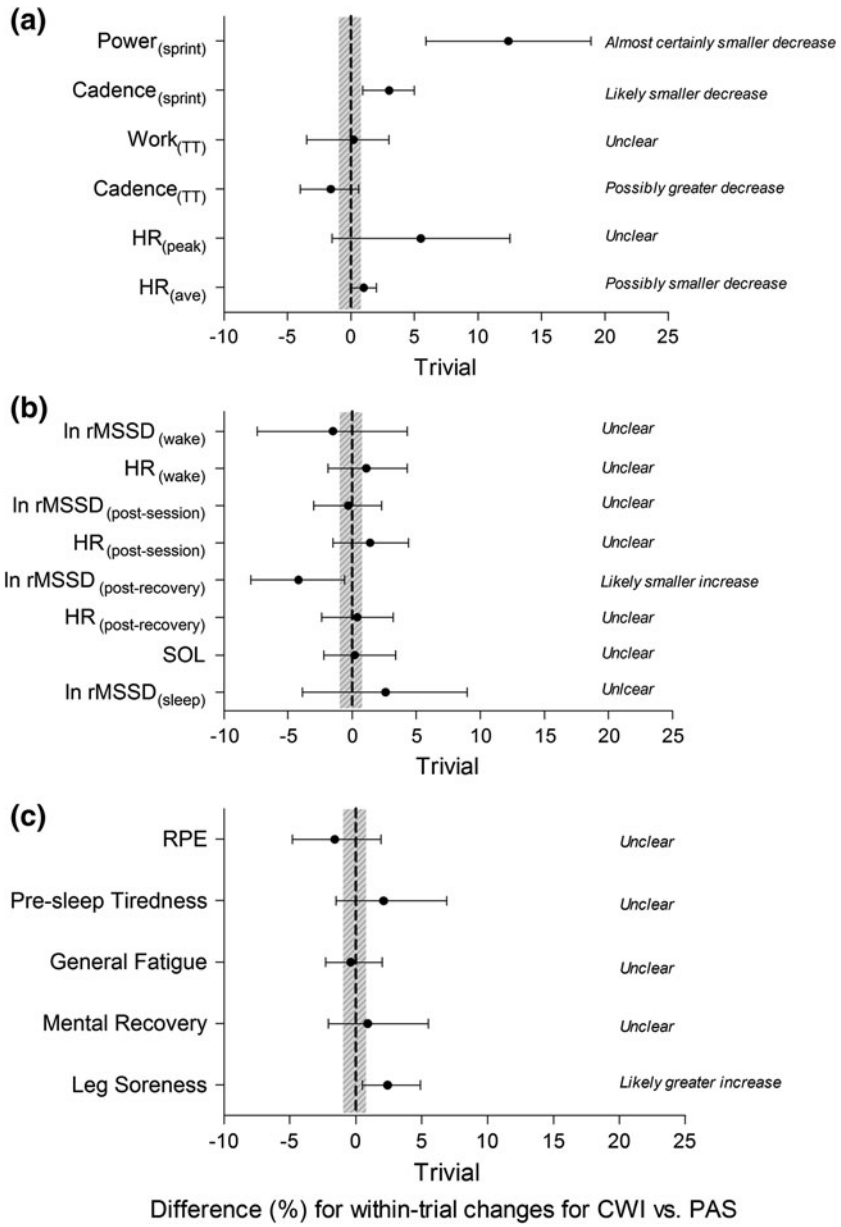

Fig. 3 The difference in within-trial change during the cold water immersion (*CWI*) compared with passive (*PAS*) recovery ($n = 11$ unless otherwise stated). Differences are for changes in performance-related variables (**a**) sprint power output [$Power_{(sprint)}$], sprint cadence [$Cadence_{(sprint)}$], work performed during the time trials [$Work_{(TT)}$], time-trial cadence [$Cadence_{(TT)}$], peak [$HR_{(peak)}$] and mean [$HR_{(ave)}$] during the session; recovery related HR variables (**b**) the natural logarithm of the square-root of the mean sum of the squares of differences between adjacent normal *R–R* intervals upon waking [ln $rMSSD_{(wake)}$, $n = 9$], immediately following the session [ln $rMSSD_{(post\text{-}session)}$], immediately following the recovery intervention [ln $rMSSD_{(post\text{-}recovery)}$], and during slow wave sleep [ln $rMSSD_{(sleep)}$, $n = 4$], heart rate upon waking [$HR_{(wake)}$, $n = 9$], following the session [$HR_{(post\text{-}session)}$], sleep onset latency (*SOL*, $n = 10$); subjective variables (**c**) mean rating of perceived exertion during the session (RPE), perceived tiredness prior to sleep (Pre-sleep Tiredness, $n = 10$), perceptions of recovery (General Fatigue, Mental Recovery, Leg Soreness, Physical Recovery, $n = 10$). Note that for Mental Recovery and Physical Recovery, a higher score is beneficial for recovery whereas for General Fatigue and Leg Soreness, a lower score is beneficial for recovery (see "Methods"). *Error bars* indicate uncertainty in the true mean changes with 90 % confidence intervals. The *shaded area* represents the smallest worthwhile change (see "Methods"). For figure clarity, changes are represented as a factor of the smallest worthwhile change

(Fig. 4a). Within each session, increased power output was observed early in the session (5- and 10-s sprints sets) but not later in the session (15-s sprint sets and time trials) following CWI compared with PAS recovery (Fig. 4b). These findings may indicate that the benefits of CWI are smaller during longer sprints compared to short sprints.

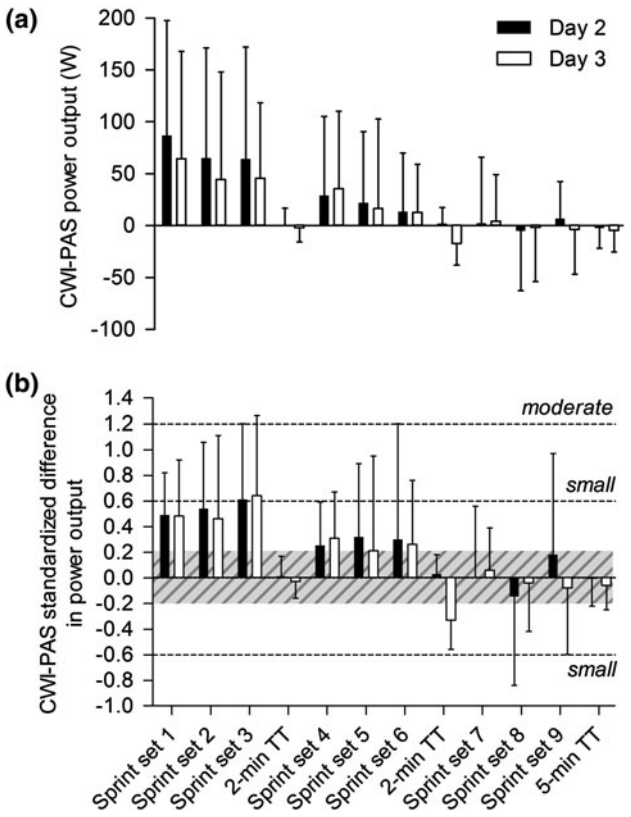

Fig. 4 Between-trial differences at each stage of the training session protocol for cold water immersion (*CWI*) versus passive (*PAS*) recovery as **a** mean power output (±SD), and **b** standardised differences (±90 % CI). *Dashed lines* represent threshold values for ES statistics with the *shaded area* representing a trivial difference

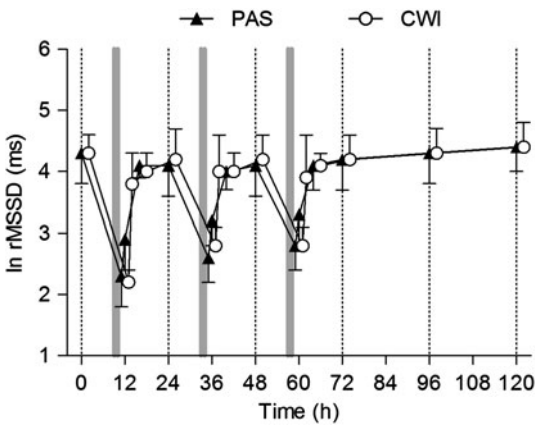

Fig. 5 Time course of cardiac parasympathetic activity during the 6-day experimental trials. *Shaded areas* denote the laboratory training sessions. *Vertical dashed lines* indicate waking HRV time points. Data are presented as mean ± SD

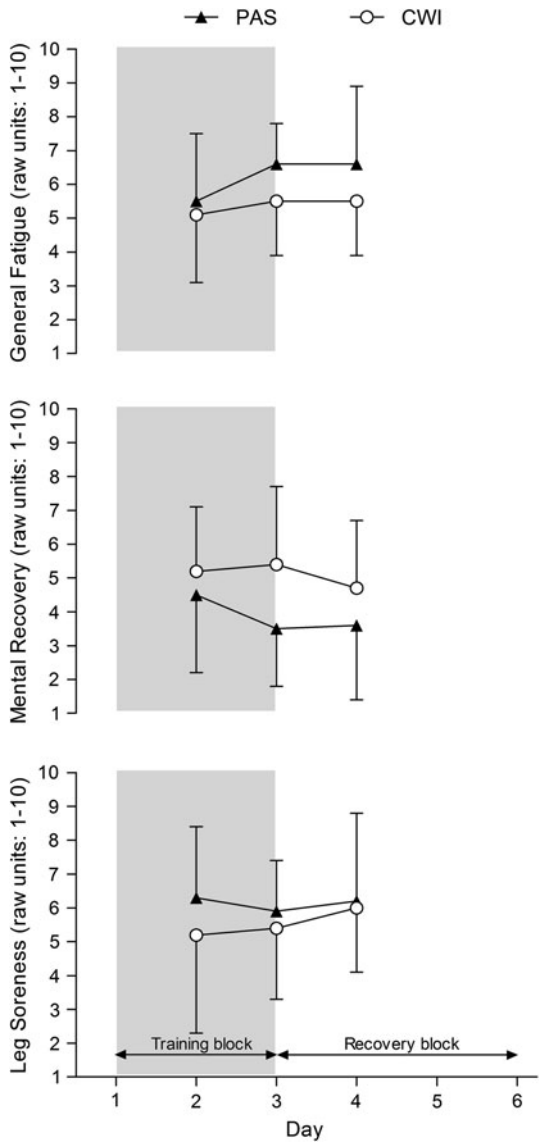

Fig. 6 Time course of subjective measures of recovery during the 6-day experimental trials. *Shaded area* denotes the training block (laboratory training sessions). Data are presented as mean ± SD

However, because the longer sprints occurred at the end of the cycling protocol, it is also possible that the benefits of CWI simply gradually diminish as a result of cumulative fatigue during exercise. Pointon et al. (2011) reported that cold water improved short-term (i.e., 5 min post-exercise), but not long-term (i.e., 24 h post-exercise) recovery of maximal strength of the quadriceps. Differences in the CWI protocol (i.e., legs only vs. legs and upper body) and the assessment of muscle function (i.e., single vs. multiple muscle groups) may partially explain the differences between our findings and those of Pointon et al. (2011). Our observation that CWI benefited sprint performance is consistent with other reports where exercise bouts were separated by approximately 24 h (Lane and Wenger 2004; Vaile et al. 2008). Lane and Wenger (2004) found that compared with other recovery modalities (e.g., active recovery, passive recovery or massage) immersion of the legs for 15 min in 15 °C water resulted in better

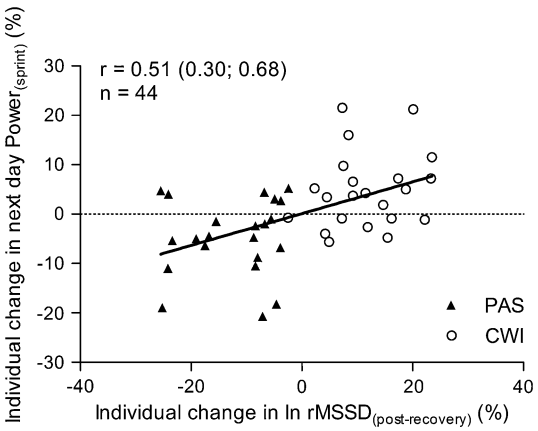

Fig. 7 Relationship between the change in ln $rMSSD_{(post\text{-}recovery)}$ and the change in sprint power output ($Power_{(sprint)}$) on the following day. Within-trial correlations were unclear (PAS, $r = 0.09$ [−0.28; 0.43], $n = 22$; CWI, $r = 0.20$ [−0.17; 0.53], $n = 22$)

maintenance of work (with slight improvement) during an 18-min intermittent cycling protocol. Vaile et al. (2008) reported that over five consecutive days of high-intensity cycling, whole-body immersion for 10 min in 14 °C water following exercise resulted in better maintenance of sprint and time-trial performance. We observed no difference in time-trial performance (Table 2; Figs. 2, 3a). Similarly, Buchheit et al. (2011a) observed improved sprint performance but not sustained high-speed running performance 48 h after exercise in young soccer players when they used a spa treatment (recovery using a combination of sauna, CWI, and Jacuzzi) after exercise. This difference may reflect the greater reliance on muscle metabolism rather than neuromuscular function during the sustained effort of time trials. Differences in the exercise protocol (e.g., warm-up, bicycle set-up) and nature of the recovery intervention (5 min at 10 °C vs. 14 min at 14 °C) may partially account for the variation between our findings and those of Vaile et al. (2008).

Within-trial changes during the training block indicate that average HR during exercise was maintained during the CWI trial, whereas it decreased in the passive recovery trial (Fig. 3a). Despite similar ratings of perceived exertion in both trials (Table 1; Fig. 3c), the higher average HR and higher sprint power output suggest that the cyclists sustained a higher overall exercise intensity over the course of the CWI trial. This concept is supported by the moderate positive relationship between session $HR_{(peak)}$ and sprint performance ($r = 0.30$), and small positive relationship between session $HR_{(ave)}$ and sprint performance ($r = 0.29$). These findings suggest that CWI may increase sprint capacity, independent of subjective perceptions of effort. This has important implications, not only for performance maintenance in athletic competition over successive days but also for possible training adaptations when athletes use CWI during intensive training blocks (i.e., CWI enables athletes to train harder).

Immediately after the recovery interventions, cardiac parasympathetic activity was substantially higher in the CWI trial compared with the passive trial (Table 2; Fig. 6). This finding consolidates the concept that CWI promotes faster reactivation of cardiac parasympathetic activity following intense exercise (Buchheit et al. 2009b; Stanley et al. 2012). During the training block, the level of parasympathetic reactivation following CWI remained stable each day. By contrast, it increased following passive recovery each day (Figs. 3b, 6). This difference may simply reflect the lower exercise intensity as indicated by the lower exercise HR in the passive recovery trial (Figs. 2, 3a). Evidence supporting this notion is that greater exercise intensity is related to slower post-exercise parasympathetic reactivation (Seiler et al. 2007). Measuring blood lactate concentration, which correlates with post-exercise HRV (Buchheit et al. 2007), would have helped to confirm this hypothesis.

The present findings contrast with the notion that cardiac parasympathetic activity may be increased following CWI for up to 12 h (Al Haddad et al. 2011b). We postulated that the accelerated recovery kinetics of cardiac parasympathetic reactivation in response to CWI would be predictive of improved overnight recovery. However, CWI did not affect sleep onset latency in any obvious way (Table 1; Fig. 3b). This finding suggests that by the time the cyclists retired to bed (approximately 2.5 h after the recovery intervention), any effects of CWI on the different factors that may promote rapid sleep onset had likely abated. In agreement with this, nocturnal cardiac parasympathetic activity was not higher following CWI compared with passive recovery during the training block, but tended to be lower (Table 2; Figs. 3b, 6). Because cardiac parasympathetic activity is inversely related to the intensity of prior exercise (Al Haddad et al. 2009; Myllymäki et al. 2012), the likely higher exercise intensity during the CWI trial (see above) may have attenuated the expected increase in cardiac parasympathetic activity after CWI. In turn, this response may have reduced nocturnal parasympathetic activity. However, the overnight rest was probably long enough for the autonomic nervous system function to recover, as illustrated by the lack of any clear difference in waking cardiac parasympathetic activity or HR between trials (Table 2; Figs. 3b, 6). Our findings suggest that CWI enabled athletes to train harder while maintaining a similar morning cardiac parasympathetic activity as they would if they trained less intensely, and without CWI. Therefore, the present study supports the use of CWI during intense training blocks (because a progressive decrease in cardiac parasympathetic activity would be expected with accumulated fatigue). Our results may appear to conflict with

those of Al Haddad et al. (2011b). This group observed that cardiac parasympathetic activity upon waking increased over five consecutive days of recovery with CWI, while it decreased over the same period with passive recovery. One explanation for this difference is that the cyclists in the present study exercised at variable self-selected intensities, whereas the athletes in the study by Al Haddad et al. (2011b) exercised at a fixed intensity. Together, these findings suggest that the effect of CWI on cardiac parasympathetic activity recovery is time-dependent (e.g., immediately post-exercise vs. nocturnal measures) and may also depend on the work performed during prior exercise. The decreased nocturnal parasympathetic activity and unchanged sleep onset latency observed here suggest that, although athletes are able to train harder with repeated CWI treatments, other recovery interventions might still be required to promote sleep quality (e.g., relaxation, specific snacks, etc.; Halson 2008).

We could not detect any clear effects of CWI on perceived tiredness immediately prior to sleep or subjective measures of recovery. CWI did reduce leg soreness after the first training session (Table 2; Figs. 3c, 5). We have previously found that CWI immediately improved subjective recovery (Buchheit et al. 2009b; Stanley et al. 2012), which was associated with higher cardiac parasympathetic activity (Stanley et al. 2012). We also found a moderate correlation between cardiac parasympathetic activity and leg soreness ($r = -0.50$) during the $\sim$2 h following CWI (Stanley et al. 2012). In the present study, we observed a small negative correlation ($r = -0.25$) between cardiac parasympathetic activity immediately following the recovery intervention and general fatigue. The magnitude of the correlation suggests that caution should be exercised when interpreting the data.

We compared the effects of CWI on cardiac parasympathetic activity during the three recovery days following the intensive training block. To our knowledge, this experimental design is unique, because other researchers have only examined autonomic responses to single sessions of high-intensity exercise over a limited time period (i.e., a maximum of 72 h) (Al Haddad et al. 2009; Buchheit et al. 2009a; Hautala et al. 2001; James et al. 2002; Mourot et al. 2004). Cardiac parasympathetic activity is transiently suppressed after intense exercise, but with adequate recovery, it can 'rebound' above pre-exercise values. The magnitude of this rebound effect likely depends on how much cardiac parasympathetic activity is suppressed after prior exercise. We found that CWI allowed the cyclists to exercise at a higher self-selected intensity (Figs. 2, 3a). Normally, this response would have caused greater suppression of cardiac parasympathetic activity after exercise. However, CWI also minimised the suppression of cardiac parasympathetic activity following exercise (Table 1; Fig. 6). Consequently, the time course of recovery of cardiac parasympathetic activity upon waking following the training block was similar for each trial (Fig. 6). When considering cardiac parasympathetic activity as a global recovery index, we interpret these findings as evidence that post-exercise CWI may reduce the magnitude of physiological adaptation following intense training. The training block and recovery period in the present study was relatively short. Further research is needed to determine whether manipulating cardiac parasympathetic activity using CWI during recovery influences long-term training adaptation (Yamane et al. 2006).

We found no clear effect of CWI on cardiac parasympathetic activity during sleep or upon waking, but the level of parasympathetic reactivation immediately following the recovery intervention correlated with sprint performance the following day ($r = 0.51$ [0.30, 0.68]) (Fig. 7). A correlation of this magnitude requires careful interpretation. However, it suggests that cardiac parasympathetic activity during recovery from exercise can be indicative of an athlete's readiness to perform, i.e., high-intensity performance capacity. It remains unknown whether changes in cardiac parasympathetic activity mediate, or simply reflect physiological processes related to recovery. Nevertheless, our data support the concept that cardiac parasympathetic activity is a useful tool to monitor physical performance (Borresen and Lambert 2008; Buchheit et al. 2011b).

Several practical considerations arose while collecting HR and sleep data that warrant brief discussion. To keep the experimental design as practical as possible, rather than sleeping overnight in the laboratory, the cyclists slept at home and recorded their HR while sleeping and upon waking. Unfortunately, noise resulting from home-based HRV recording and our requirement for complete HRV data sets meant that only $\sim$80 % of the waking HRV data and $\sim$40 % of the sleep HRV data were suitable for analysis. We chose not to control the time that the cyclists retired to bed so that we could assess whether the recovery interventions influenced their natural sleep habits. If we had controlled sleeping time, we may have observed greater differences in sleep onset latency. In reality, however, the time that athletes go to sleep and the period they sleep for is variable. For these reasons, it is difficult to generalise about the relationships between cardiac parasympathetic activity and sleep onset and quality. Factors beyond our control such as personal commitments (i.e., work or study) may also have influenced the natural sleeping duration and autonomic responses of the cyclists. They were instructed to standardise their diet and training from week to week, and we are confident that variation in diet or training between the two trials did not substantially influence our results.

Conclusions

The present study confirms that CWI post-exercise allows for better maintenance of sprint cycling performance during a short training block that comprises three consecutive days of intense exercise. In practice, CWI may allow athletes to perform better over consecutive days of competition (or train harder in preparation for competition), while maintaining acceptable levels of cardiac parasympathetic activity upon waking, and perceptions of recovery. The decreased nocturnal parasympathetic activity and unchanged sleep onset latency observed here suggest that although CWI allows athletes to train/compete more intensely, other recovery interventions might still be required to promote sleep quality, and, in turn, overall recovery.

Acknowledgments The authors would like to acknowledge and thank the cyclists for their generous time commitment and effort throughout the study. This study was supported by the Centre of Excellence for Applied Sport Science Research at the Queensland Academy of Sport, Brisbane.

References

Al Haddad H, Laursen P, Ahmaidi S, Buchheit M (2009) Nocturnal heart rate variability following supramaximal intermittent exercise. Int J Sports Physiol Perform 4:435–447

Al Haddad H, Laursen PB, Chollet D, Ahmaidi S, Buchheit M (2011a) Reliability of resting and postexercise heart rate measures. Int J Sports Med 32:598–605

Al Haddad H, Parouty J, Buchheit M (2011b) Effect of daily cold water immersion on heart rate variability and subjective ratings of well-being in highly trained swimmers. Int J Sports Physiol Perform Aug 30 (Epub ahead of print)

Bloomfield DM, Magnano A, Bigger JT Jr, Rivadeneira H, Parides M, Steinman RC (2001) Comparison of spontaneous vs. metronome-guided breathing on assessment of vagal modulation using RR variability. Am J Physiol Heart Circ Physiol 280:H1145–H1150

Borresen J, Lambert MI (2008) Autonomic control of heart rate during and after exercise: measurements and implications for monitoring training status. Sports Med 38:633–646

Brandenberger G, Buchheit M, Ehrhart J, Simon C, Piquard F (2005) Is slow wave sleep an appropriate recording condition for heart rate variability analysis? Auton Neurosci 121:81–86

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

Buchheit M, Laursen PB, Ahmaidi S (2007) Parasympathetic reactivation after repeated sprint exercise. Am J Physiol Heart Circ Physiol 293:H133–H141

Buchheit M, Laursen PB, Al Haddad H, Ahmaidi S (2009a) Exercise-induced plasma volume expansion and post-exercise parasympathetic reactivation. Eur J Appl Physiol 105:471–481

Buchheit M, Peiffer JJ, Abbiss CR, Laursen PB (2009b) Effect of cold water immersion on postexercise parasympathetic reactivation. Am J Physiol Heart Circ Physiol 296:H421–H427

Buchheit M, Chivot A, Parouty J, Mercier D, Al Haddad H, Laursen PB, Ahmaidi S (2010) Monitoring endurance running performance using cardiac parasympathetic function. Eur J Appl Physiol 108:1153–1167

Buchheit M, Horobeanu C, Mendez-Villanueva A, Simpson BM, Bourdon PC (2011a) Effects of age and spa treatment on match running performance over two consecutive games in highly trained young soccer players. J Sports Sci 29:591–598

Buchheit M, Simpson M, Al Haddad H, Bourdon P, Mendez-Villanueva A (2011b) Monitoring changes in physical performance with heart rate measures in young soccer players. Eur J Appl Physiol 112:1–13

Burgess HJ, Trinder J, Kim Y, Luke D (1997) Sleep and circadian influences on cardiac autonomic nervous system activity. Am J Physiol Heart Circ Physiol 273:H1761–H1768

Cohen J (1988) Statistical power analysis for behavioral sciences. Lawrence Erlbaum Associates, Hillsdale

Halson SL (2008) Nutrition, sleep and recovery. Eur J Sport Sci 8:119–126

Hautala AJ, Tulppo MP, Mäkikallio TH, Laukkanen R, Nissilä S, Huikuri HV (2001) Changes in cardiac autonomic regulation after prolonged maximal exercise. Clin Physiol 21:238–245

Hopkins WG (2006) Spreadsheets for analysis of controlled trials with adjustment for a subject characteristic. Sportscience 10:46–50. http://sportsci.org/2006/wghcontrial.htm

Hopkins WG (2010) Linear models and effect magnitudes for research, clinical and practical applications. Sportscience 14: 49–57. http://sportsci.org/2010/wghlinmod.htm

Hopkins WG, Marshall SW, Batterham AM, Hanin J (2009) Progressive statistics for studies in sports medicine and exercise science. Med Sci Sports Exerc 41:3–13

Iellamo F, Legramante JM, Pigozzi F, Spataro A, Norbiato G, Lucini D, Pagani M (2002) Conversion from vagal to sympathetic predominance with strenuous training in high-performance world class athletes. Circulation 105:2719–2724

Iwasaki K, Zhang R, Zuckerman JH, Levine BD (2003) Dose-response relationship of the cardiovascular adaptation to endurance training in healthy adults: how much training for what benefit? J Appl Physiol 95:1575–1583

James DVB, Barnes AJ, Lopes P, Wood DM (2002) Heart rate variability: response following a single bout of interval training. Int J Sports Med 23:247–251

Kaikkonen P, Hynynen E, Mann T, Rusko H, Nummela A (2010) Can HRV be used to evaluate training load in constant load exercises? Eur J Appl Physiol 108:435–442

Kiviniemi A, Hautala A, Kinnunen H, Tulppo M (2007) Endurance training guided individually by daily heart rate variability measurements. Eur J Appl Physiol 101:743–751

Lane KN, Wenger HA (2004) Effect of selected recovery conditions on performance of repeated bouts of intermittent cycling separated by 24 hours. J Strength Cond Res 18:855–860

Mourot L, Bouhaddi M, Tordi N, Rouillon J-D, Regnard J (2004) Short- and long-term effects of a single bout of exercise on heart rate variability: comparison between constant and interval training exercises. Eur J Appl Physiol 92:508–517

Myllymäki T, Rusko H, Syväoja H, Juuti T, Kinnunen M-L, Kyröläinen H (2012) Effects of exercise intensity and duration on nocturnal heart rate variability and sleep quality. Eur J Appl Physiol 112:801–809

Paton CD, Hopkins WG (2006) Variation in performance of elite cyclists from race to race. Eur J Sport Sci 6:25–31

Peiffer J, Abbiss C, Watson G, Nosaka K, Laursen P (2009) Effect of cold-water immersion duration on body temperature and muscle function. J Sports Sci 27:987–993

Pichot V, Roche F, Gaspoz JM, Enjolras F, Antoniadis A, Minini P, Costes F, Busso T, Lacour JR, Barthélémy JC (2000) Relation between heart rate variability and training load in middle-distance runners. Med Sci Sports Exerc 32:1729–1736

Pointon M, Duffield R, Cannon J, Marino F (2011) Cold water immersion recovery following intermittent-sprint exercise in the heat. Eur J Appl Physiol. doi:10.1007/s00421-011-2218-3

Sandercock GR, Bromley PD, Brodie DA (2005) Effects of exercise on heart rate variability: inferences from meta-analysis. Med Sci Sports Exerc 37:433–439

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

Shinar Z, Akselrod S, Dagan Y, Baharav A (2006) Autonomic changes during wake-sleep transition: a heart rate variability based approach. Auto Neurosci 130:17–27

Stanley J, Buchheit M, Peake JM (2012) The effect of post-exercise hydrotherapy on subsequent exercise performance and heart rate variability. Eur J Appl Physiol 112:951–961

Task-Force (1996) Heart rate variability: standards of measurement, physiological interpretation, and clinical use. Circulation 93:1043–1065

Vaile J, Halson S, Gill N, Dawson B (2008) Effect of hydrotherapy on recovery from fatigue. Int J Sports Med 29:539–544

Vaile J, O'Hagan C, Stefanovic B, Walker M, Gill N, Askew CD (2010) Effect of cold water immersion on repeated cycling performance and limb blood flow. Br J Sports Med. doi:10.1136/bjsm.2009.067272

Yamane M, Teruya H, Nakano M, Ogai R, Ohnishi N, Kosaka M (2006) Post-exercise leg and forearm flexor muscle cooling in humans attenuates endurance and resistance training effects on muscle performance and on circulatory adaptation. Eur J Appl Physiol 96:572–580