

One night of partial sleep deprivation impairs recovery from a single exercise training session

Dale E. Rae¹ · Tayla Chin¹ · Kagiso Dikgomo¹ · Lee Hill¹ · Andrew J. McKune^{3,4} · Tertius A. Kohn¹ · Laura C. Roden²

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Abstract

Purpose The effects of sleep deprivation on physical performance are well documented, but data on the consequence of sleep deprivation on recovery from exercise are limited. The aim was to compare cyclists' recovery from a single bout of high-intensity interval training (HIIT) after which they were given either a normal night of sleep (CON, 7.56 ± 0.63 h) or half of their usual time in bed (DEP, 3.83 ± 0.33 h).

Methods In this randomized cross-over intervention study, 16 trained male cyclists (age 32 ± 7 years), relative peak power output (PPO 4.6 ± 0.7 W kg⁻¹) performed a HIIT session at ±18:00 followed by either the CON or DEP sleep condition. Recovery from the HIIT session was assessed the following day by comparing pre-HIIT

variables to those measured 12 and 24 h after the session. Following a 2-week washout, cyclists repeated the trial, but under the alternate sleep condition.

Results PPO was reduced more 24 h after the HIIT session in the DEP (Δ PPO -0.22 ± 0.22 W kg⁻¹; range -0.75 to 0.1 W kg⁻¹) compared to the CON condition (Δ PPO -0.05 ± 0.09 W kg⁻¹, range -0.19 to 0.17 W kg⁻¹, $p=0.008$, $d=-2.16$). Cyclists were sleepier (12 h: $p=0.002$, $d=1.90$; 24 h: $p=0.001$, $d=1.41$) and felt less motivated to train (12 h, $p=0.012$, $d=-0.89$) during the 24 h recovery phase when the HIIT session was followed by the DEP condition. The exercise-induced 24 h reduction in systolic blood pressure observed in the CON condition was absent in the DEP condition ($p=0.039$, $d=0.75$).

Conclusions One night of partial sleep deprivation impairs recovery from a single HIIT session in cyclists. Further research is needed to understand the mechanisms behind this observation.

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✉ Dale E. Rae
Dale.Rae@uct.ac.za

¹ Division of Exercise Science and Sports Medicine, Department of Human Biology, Faculty of Health Sciences, University of Cape Town, PO Box 115, Newlands, Cape Town 7725, South Africa

² Department of Molecular and Cell Biology, Faculty of Science, University of Cape Town, Cape Town, South Africa

³ Research Institute for Sport and Exercise, University of Canberra, Canberra, Australia

⁴ Discipline of Biokinetics, Exercise and Leisure Sciences, School of Health, University of KwaZulu-Natal, Durban, South Africa

Keywords Sleep deprivation · Recovery strategies · Maximal performance · High-intensity interval training · Cyclists

Abbreviations

BP	Blood pressure
CK	Creatine kinase
CON	Control condition: normal night of sleep
DBP	Diastolic blood pressure
DEP	Partial sleep deprivation condition: 50% of normal sleep
HIIT	High-intensity interval training
HR	Heart rate
HR _{max}	Maximum heart rate
IgA	Immunoglobulin A
PPO	Peak power output

RER	Respiratory exchange ratio
RPE	Rating of perceived exertion
SBP	Systolic blood pressure
sIgA	Salivary immunoglobulin A
VO _{2max}	Maximum volume of oxygen uptake

Introduction

In the context of sport and exercise, sleep is recognized as being critical for an athlete's well-being and performance. Much of sleep's value may lie in its role in recovery, from both training and competition, an important factor determining performance. In a recent survey of 890 team sport athletes, sleep was identified as one of the most important recovery strategies (Venter 2014). When elite football players had night matches, their sleep duration was typically reduced on the night of the match and the following day they reported feeling less recovered compared to day matches (Fullagar et al. 2016). Kölling et al. (2016) observed 55 junior, national-level rowers during a 4-week training camp. Following just one night of shortened sleep at the end of the first week of the camp, the rowers reported feeling less recovered and in a worsened emotional state compared to the previous day, which had been preceded by a normal night of sleep (Kölling et al. 2016). Furthermore, the fact that more rowers napped on training days compared to rest days suggests a higher sleep need on training days. These studies provide some evidence that athletes intuitively value sleep in the context of recovery.

Despite this, sleep complaints are prevalent in athletes (Swinbourne et al. 2015; Gupta et al. 2016). Not only does competition and travel interfere with their sleep (Fullagar et al. 2016; Gupta et al. 2016), but also training itself may reduce sleep quality and quantity. Training periods in which intensity is increased or early morning sessions are scheduled have both been shown to reduce sleep duration in athletes (Sargent et al. 2013; Schaal et al. 2015; Kölling et al. 2016). Thus athletes and coaches are challenged to balance training programmes, travel and competition schedules, and sleep routines to facilitate optimal performance. Pertinently, a recent consensus statement from the International Olympic Committee called on coaches to ensure sufficient rest and recovery periods in training programmes for youth athletes to “encourage positive adaptations and progressive athletic development” (Bergeron et al. 2015).

A single bout of exercise perturbs the homeostasis of the skeletal muscle, cardiovascular, nervous, endocrine, metabolic and immune systems (Reilly and Ekblom 2007; Heinonen et al. 2014; Walsh and Oliver 2016) and the ensuing recovery of these systems can be viewed as an adaptation to the specific training stimulus in anticipation of future exposures. One of the functions of sleep is

to provide an opportunity for both neurological and physiological systems to recover and repair (Akerstedt and Nilsson 2003). Both complete and partial sleep deprivation are understood to compromise various physiological systems (Zhong 2005; Van Cauter et al. 2008; Ruiz et al. 2012) and reduce physical performance the next day (Souissi et al. 2003; Oliver et al. 2009; Temesi et al. 2013). Reducing sleep only partially (4 h for 6 days) has been shown to decrease glucose tolerance, increase evening cortisol levels, elevate sympathovagal balance, and disrupt the normal pattern of growth hormone at night (Akerstedt and Nilsson 2003). Reduced sleep may interfere with muscle recovery due to increased signalling to initiate proteolysis, essentially creating a more catabolic state (Dattilo et al. 2011). The fact that resting systolic blood pressure increases after partial sleep deprivation (± 3.5 h), potentially indicates that sympathetic nervous system activity is altered by reduced sleep (Tochikubo et al. 1996). Sleep deprivation is also understood to disrupt the immune system (Faraut et al. 2012), with effects similar to those of intense exercise on both the innate and acquired immune systems (Walsh and Oliver 2016). Sleep loss also reduces mood and motivation (Reilly and Piercy 1994; Scott et al. 2006), both of which may impact effort produced during a subsequent training session. Collectively, these data suggest that recovery from exercise may be impaired by sleep deprivation through the compromised function of any number of systems.

While numerous recovery enhancing modalities such as nutrition (Beelen et al. 2010), ice (Poppendieck et al. 2013) and compression (MacRae et al. 2011) having been investigated, studies measuring the effect of sleep deprivation on recovery from exercise are scant (Skein et al. 2013). As early as 1984 recovery from a 20 min training session was assessed in five males, under both normal sleep and complete sleep deprivation conditions. While submaximal performance was not compromised with sleep deprivation, recovery was hampered, as indicated by altered profiles of blood glucose, lactate, minute ventilation and oxygen uptake during submaximal exercise (McMurray and Brown 1984). More recently, recovery of lower limb power in rugby players who underwent complete sleep deprivation the night after a match was shown to be impaired compared to a normal night of sleep (Skein et al. 2013).

Both of these previous studies (McMurray and Brown 1984; Skein et al. 2013) made use of complete sleep deprivation models. While they are useful designs to understand the extreme effects of sleep restriction, athletes may be more likely to experience poor sleep or reduced sleep duration following competition or during training phases (Sargent et al. 2013; Schaal et al. 2015; Kölling et al. 2016; Fullagar et al. 2016). Therefore, investigation of the effects of partial sleep deprivation on recovery from exercise would

expand previous research in this area by providing information on a sleep scenario many athletes may face.

The aim of this study was to determine whether partial sleep deprivation impairs recovery from training. The objective was to compare cyclists' recovery from a single bout of high-intensity interval training (HIIT) after which they were given either a normal night of sleep or were partially sleep deprived. Recovery was assessed by monitoring motivation to train; muscle soreness and tiredness; sleepiness; resting heart rate and blood pressure; maximal power, heart rate and oxygen uptake; creatine kinase activity; leukocyte cell counts and salivary IgA secretion rate during the 24 h after the HIIT session. The hypothesis was that a full, normal night of sleep after a HIIT session would be necessary to facilitate recovery, assessed by the ability to perform at close to maximum in a peak power output test 24 h after the HIIT. Specifically, it was hypothesised that even one night of partial sleep deprivation would be sufficient to impair recovery.

Methods

Participants

Sixteen trained male cyclists participated in this study. To be included they needed to be 20–50 years old, usually sleep >6 h per night, train at least three times per week (past 6 months) and have ridden the Cape Town Cycle Tour, an annual 110 km road race in South Africa, within the last year in a time of <3 h 45 min. Exclusion criteria were diagnosed chronic cardiovascular or metabolic disease, psychiatric condition, sleep disorder, or any other condition known to affect sleep; current or recent use of sleep medication (past 2 months); recent trans-meridian travel exceeding three time zones (past 2 months); and shift work. The Human Research Ethics Committee of the Faculty of Health Sciences, University of Cape Town approved this study (HREC No: 272/2014) and all participants provided written informed consent. Descriptive characteristics of the participants are presented in Table 1.

Study overview

An overview of the design of this randomized crossover intervention study is depicted in Fig. 1. Briefly, in session 1 (17:30) participants completed a questionnaire to document medical and cycling histories, assess chronotype using the Horne–Östberg morningness–eveningness personality questionnaire (Horne and Östberg 1976) and measure sleep quality using the Pittsburgh Sleep Quality Index (Buysse et al. 1989). They also practiced the peak power output (PPO) test. For the next seven consecutive days participants wore a

Table 1 Participant characteristics ($n=16$)

	Mean \pm SD	Range
Age (years)	32.3 \pm 7.1	22–47
Height (m)	1.80 \pm 0.08	1.62–1.94
Weight (kg)	76.9 \pm 9.5	60.6–95.7
BMI (kg m ⁻²)	23.8 \pm 1.6	21.2–27.4
Chronotype score	63 \pm 9	44–76 ^a
Sleep quality	4 \pm 1	2–7
VO _{2max} (ml min ⁻¹ kg ⁻¹)	57.6 \pm 8.3	45.1–79.3
Peak power output (W kg ⁻¹)	4.61 \pm 0.70	3.69–6.37

BMI body mass index, VO_{2max} maximum oxygen uptake volume

^a11 of the cyclists were morning-types (69%), 5 were neither-types (31%), and there were no evening-types

wrist actigraph (Actiwatch AW2, Philips Respironics, Bend, OR, USA) to establish their usual sleep habits. Sleep timing was verified using a diary. They were asked to refrain from training 48 h before and to avoid caffeine and alcohol 24 h before session 2. A dietary log completed in the 24 h period prior to sessions 2 and 5 was used to verify this. There was 94% compliance to the 24 h alcohol restriction for both the CON and DEP conditions, and 100 and 88% compliance to the caffeine restriction prior to the CON and DEP sessions, respectively. Participants also used the dietary log to match their food intake times, content and quantity before session 5 to that of session 2.

Participants fasted for 3 h before session 2 (17:30) at which baseline measures (Pre) were taken. These included weight, sleepiness, motivation to train, muscle soreness and tiredness scores, resting heart rate and blood pressure, saliva and blood samples, and a PPO test. This was followed immediately by the HIIT session at 18:00. Participants then slept in the laboratory under either control sleep (CON) or partial sleep deprivation (DEP) conditions. Follow-up sessions 3 (06:30–07:00) and 4 (18:30) took place 12 and 24 h after session 2, respectively, during which all baseline measures were repeated, with the exception of the PPO test, which was only performed at session 4. After a 2-week washout, cyclists repeated the trial (sessions 5–7) under the alternate sleep condition. The order in which participants underwent the intervention was randomized. While it was not possible to blind participants to the condition (CON or DEP), they were only told their condition after the first HIIT session.

Detailed testing procedures

Subjective, heart rate (HR) and blood pressure (BP) measurements

The Epworth Sleepiness Scale was used to measure sleepiness (Johns 1991). Participants rated their “motivation to

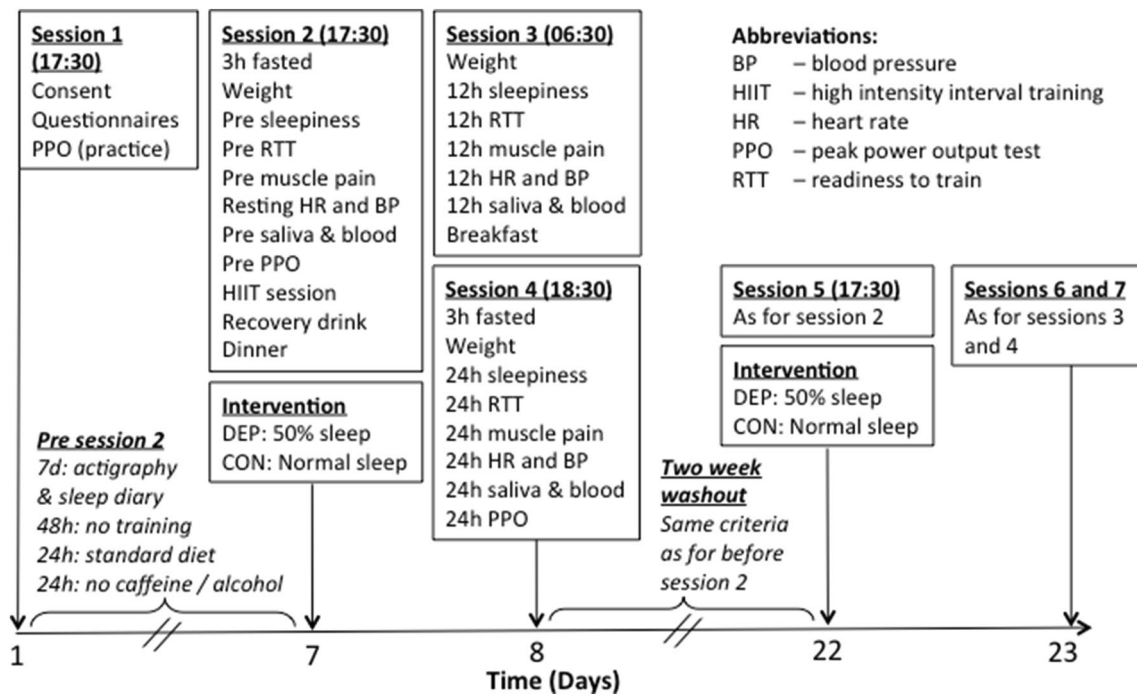


Fig. 1 Study overview

train” on a 10-point scale (1: “not into it” through to 10: “extremely motivated”). Leg muscle soreness and tiredness were assessed when standing, during a quadriceps stretch and immediately after climbing two flights of stairs (2 steps per second), since each scenario may elicit soreness or tiredness to different extents in different individuals. These ratings made use of an 11-point scale (0: “no soreness or tiredness” through to 10: “extreme soreness or tiredness”), and the global leg muscle soreness and tiredness scores summed all three measurements. Only the global values are presented in the results. Resting HR and BP measurements represent the average of three values taken after 10 min of seated rest using an automated monitor (Omron HEM-907, Omron Health Care, Kyoto, Japan).

Immune function and muscle damage

Saliva samples were collected from participants during sessions 2–7 by unstimulated passive drool to assess immune function. Salivary immunoglobulin A (sIgA) secretion rate ($\mu\text{g min}^{-1}$) was determined using an indirect enzyme immunoassay kit (Salimetrics, State College, USA). Details of this procedure have been published previously (McKune et al. 2006). Venous blood samples were obtained from participants to measure indices of muscle damage (creatine kinase (CK) activity) and immune function (white blood cell count, WBC, and differential). Samples for CK activity were collected into lithium heparin vacutainer

tubes, placed on ice, centrifuged at $3000\times g$ for 10 min at 4°C , after which the plasma was removed and stored in microfuge tubes at -80°C . CK activity was determined using a commercial enzymatic assay kit (CK-NAC-activated, Boehringer Mannheim, Meylan, France) and a spectrophotometer (Beckman Instruments DU-530, Fullerton, CA, USA). Samples for WBC analyses were collected into EDTA vacutainers and immediately sent to a medical diagnostic laboratory (Metropolis, Cape Town) that performed the leukocyte counts.

Peak power output (PPO) test

The cyclists performed PPO tests during sessions 1, 2, 4, 5 and 7. The PPO test in session 1 was for familiarisation and to establish maximum heart rate. Those performed in sessions 2 and 5 provided baseline values for maximal aerobic performance and those in sessions 4 and 7 were to assess 24 h recovery in maximal performance following the HIIT under the CON and DEP conditions. Participants used their own bicycles mounted on a cycle ergometer (Computrainer Pro 3D, RacerMate, Seattle, USA) and set-up was the same for each session. To warm up, cyclists completed the Lamberts and Lambert Submaximal Cycling Test (Lamberts et al. 2011). This 15 min test comprises three stages: 6 min at 60% of maximum heart rate max (HR_{max}), 6 min at 80% HR_{max} and 3 min at 90% HR_{max} . Following this, the ramp format PPO test began at 2.5 W kg^{-1} body weight

Table 2 Usual ($n=16$), CON ($n=16$) and DEP ($n=16$) sleep characteristics

	Usual	CON	DEP	p_1	p_2	p_3
Bed time (h:min)	22:50±0:45	22:42±0:40	00:33±0:19	0.368	<0.001	<0.001
Wake-up time (h:min)	06:24±0:37	6:16±0:25	4:23±0:22	0.109	<0.001	<0.001
Time-in-bed (h)	7.58±0.69	7.56±0.63	3.83±0.33	0.919	<0.001	<0.001
Sleep length (h)	6.81±0.50	6.50±0.56	3.38±0.41	0.126	<0.001	<0.001
Efficiency (%)	90.2±3.5	86.4±8.0	88.3±8.1	0.059	0.386	0.246
Latency (min)	6.0 (3.4)	7.5 (0.6)	1.4 (0.4)	0.309	0.522	0.032
WASO (min)	26.9±11.2	32.4±17.7	11.9±7.5	0.156	<0.001	0.002
No. of awakenings	46±14	45±17	20±12	0.609	<0.001	0.002

Data are presented as mean ± SD or median (interquartile range)

CON control sleep condition, DEP partial sleep deprivation condition, USUAL habitual sleep characteristics, WASO wake after sleep onset time

Significance was determined using either a paired t test or Wilcoxon signed-rank test. p_1 : USUAL vs. CON; p_2 : USUAL vs. DEP; p_3 : CON vs. DEP

for 1 min. Power then increased by 20 W min⁻¹ until participants were unable to maintain a minimum cadence of 70 rpm or reached volitional exhaustion. During the test, HR (bpm), power (W), oxygen uptake and carbon dioxide production (ml kg⁻¹ min⁻¹) were measured continuously and rating of perceived exertion (RPE) at the end of every minute. HR was monitored using a Suunto t6d heart rate monitor (v2.1.0.3, Suunto, Oy, Vantaa, Finland), RPE using the 20-point Borg scale (Borg 1998), and respiratory variables using an online breath-by-breath gas analyser and pneumotach (Oxycon, Viasis, Hoechberg, Germany).

HIIT session

This 54 min session took place 5 min after the baseline PPO test in sessions 2 and 5, and simulated a hard training bout from which recovery was measured. Cyclists completed 18×1 min intervals at PPO, with 2 min of active recovery (50 W) between each interval. During the session they had ad libitum access to water and a 7% carbohydrate drink (CarboFuel, Cadence Nutrition, Cape Town, South Africa). The volume of carbohydrate drink consumed

during the HIIT was similar between conditions (CON 360±231 ml, DEP 358±187 ml, $p=0.983$). The same was true for the volume of water drunk (CON 272±257 ml, DEP 222±165 ml, $p=0.523$).

Meals

Participants ingested a recovery drink (200 ml low fat chocolate-flavoured milk; 482 kJ, 6 g protein, 18 g carbohydrate and 4 g fat per serving) within 30 min of the HIIT session, and ate dinner (beef lasagne) within 90 min. Portion sizes were standardised so that each cyclist consumed 0.42 g carbohydrate, 0.62 g protein and 0.35 g fat per kg body mass. A standardised breakfast consisting of oats, milk, fruit and caffeine-free tea was provided.

Usual sleep habits and sleep intervention

Participants wore a wrist-worn actigraph (Actiwatch AW2, Philips Respironics, Bend, OR, USA) and kept a sleep diary for seven consecutive days between sessions 1 and 2 to establish their usual sleep habits. Data for all 7 days were

Table 3 Twelve-hour responses to the HIIT session under the CON and DEP conditions

	n	CON	DEP	d	p value
ΔSleepiness	16	-1 (3) (-9 to 1)	4 (6) (-3 to 14)	1.88	0.001
ΔMotivation to train	15	-0±1 (-2 to 2)	-1±1 (-4 to 0)	-0.97	0.018
ΔMuscle soreness	16	3±3 (-5 to 8)	3±3 (-2 to 8)	0.03	0.882
ΔMuscle tiredness	16	4±4 (-4 to 11)	4±3 (0 to 12)	0.02	0.834
ΔResting HR (bpm)	16	-1±6 (-10 to 12)	-1±8 (-12 to 12)	-0.10	0.734
ΔResting SBP (mmHg)	16	-10±6 (-19 to -1)	-4±8 (-19 to 10)	0.96	0.012
ΔResting DPB (mmHg)	16	-2±8 (-14 to 18)	-3±6 (-14 to 7)	-0.05	0.845

Changes in actual values are presented as mean ± SD or median (IQR), with the range in parentheses

CON control sleep condition, DEP partial sleep deprivation condition, HIIT high-intensity interval training session, HR heart rate, SBP systolic blood pressure, DPB diastolic blood pressure

The p value indicates significance determined using either a paired t test or Wilcoxon signed-rank test. Cohen's d indicates effect size

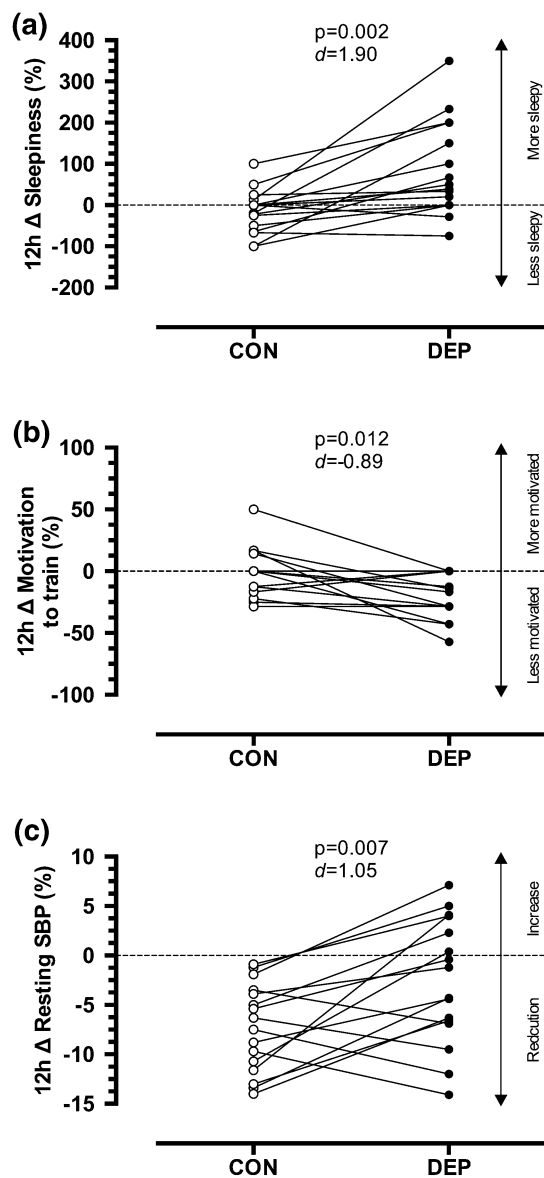


Fig. 2 Individual changes in sleepiness (a), motivation to train (b) and resting systolic blood pressure (c) from Pre to 12 h after the HIIT session under the CON and DEP conditions. Each data point represents an individual's response to the HIIT session (12 h minus Pre), expressed as percentage change. *Open* and *closed circles* represent the normal sleep (CON) and partial sleep deprivation (DEP) conditions, respectively. The *dotted line* at $y=0$ represents no change in the variable measured before and 12 h after the HIIT session (i.e. 0% change). The slope of the connecting line indicates the extent to which the change in a variable after HIIT was different in the DEP compared to the CON condition, in one individual. *SBP* systolic blood pressure, *Pre* prior to the HIIT session, *12 h* 12 h after the HIIT session. Significance was determined using a paired *t* test. Cohen's *d* indicates effect size

averaged and the following outcome variables obtained: bed time, wake-up time, time-in-bed (TIB, h), total sleep time (TST, h), sleep onset latency (SOL, min), sleep

efficiency (SE, %), wake-after sleep onset time (WASO, min) and number of awakenings (count). These data (USUAL) are presented in Table 2.

Participants slept in a sound- and light-proof sleep laboratory following the HIIT sessions. Between the end of the HIIT session and bed time in both the DEP and CON conditions, participants showered, ate dinner ($\pm 20:00$) and were then allowed to either read, watch TV or make use of their laptops in the sleep laboratory lounge. They were not permitted to recline or rest in their beds, and were in the company of the investigator at all times to ensure they did not fall asleep. Participants undertook these same activities in the period between wake-up time and the start of sessions 3 or 6 in the DEP condition.

Bed time and wake-up time during the control night (CON) replicated their USUAL sleep habits. Participants wore the wrist actigraph during the CON and DEP nights to confirm sleep timing and length. There were no differences in any of the sleep variables between the USUAL and CON nights (Table 2).

For the partial sleep deprivation (DEP) night participants were allowed only half of their usual time-in-bed. Thus mean required TIB during the DEP night was calculated to be 3.77 ± 0.35 h and mean TST 3.38 ± 0.23 h. The precise bed time and wake-up time for each participant was determined individually based on his USUAL TIB, and midpoint of sleep during the DEP night was matched to that of USUAL sleep by delaying bed time and advancing wake-up time. The actigraphy data for the DEP night (Table 2) indicate that actual TIB ($p=0.602$) and TST ($p=0.623$) were not different to the required times. Furthermore, SE ($p=0.386$) and SOL ($p=0.522$) were also similar between the USUAL and DEP nights, while the cyclists had reduced WASO time ($p<0.001$) and fewer awakenings ($p<0.001$) on the DEP night. Together these data suggest that the participants obtained the required sleep durations on the CON and DEP nights and that the quality was similar to their usual sleep.

Data and statistical analyses

Data are expressed as mean \pm standard deviation or median with interquartile range. Time data are represented as h:min. Normality was confirmed using the Shapiro–Wilks test and homogeneity of variance using Levene's test. Paired *t* tests or Wilcoxon signed-rank tests were used to compare single variables between the DEP and CON conditions. A two-way analysis of variance with repeated measures was used to compare differences between conditions in variables measured before and 24 h after the HIIT session. Effect sizes were calculated using Cohen's *d*, where $d<0.2$ was considered trivial, 0.20–0.39 small, 0.40–0.79 moderate and >0.80 large. 12 and 24 h measurements

Table 4 Twenty-four-hour responses to the HIIT session under the CON and DEP conditions

	<i>n</i>	CON		DEP		<i>d</i>	<i>p</i> value
		Pre	24 h	Pre	24 h		
Sleepiness	16	5 ± 4 (0–14)	7 ± 3 (2–12)	6 ± 3 (2–13)	12 ± 6 (2–23)	1.41	0.004
Motivation to train	15	7 ± 2 (3–10)	6 ± 2 (2–10)	7 ± 1 (6–10)	5 ± 2 (2–8)	0.74	0.128
Muscle soreness	16	4 ± 3 (0–11)	7 ± 5 (0–19)	4 ± 3 (0–11)	7 ± 5 (0–15)	0.15	0.873
Muscle tiredness	16	5 ± 4 (1–13)	10 ± 5 (3–21)	5 ± 4 (0–14)	11 ± 5 (3–21)	0.29	0.344
Resting HR (bpm)	16	59 ± 6 (46–70)	58 ± 8 (46–78)	59 ± 7 (51–70)	60 ± 7 (50–74)	0.27	0.395
Resting SBP (mmHg)	16	128 ± 8 (106–138)	121 ± 8 (112–141)	127 ± 11 (100–146)	127 ± 9 (113–145)	0.75	0.043
Resting DBP (mmHg)	16	72 ± 9 (54–87)	70 ± 8 (59–88)	73 ± 7 (59–83)	71 ± 8 (58–83)	0.04	0.906
Absolute PPO (W)	16	352 ± 44 (291–424)	347 ± 40 (285–413)	355 ± 38 (295–423)	337 ± 36 (280–411)	–1.98	0.004

Data are presented as mean ± SD, (range)

CON control sleep condition, DEP partial sleep deprivation condition, HIIT high-intensity interval training, Pre prior to the HIIT session, 24 h 24 h after the HIIT session, HR heart rate, SBP systolic blood pressure, DBP diastolic blood pressure, PPO peak power output

The *p* value represents the time-by-condition interaction effect as determined using a two-way ANOVA with repeated measures. Cohen's *d* indicates effect size

were analysed separately to minimise misinterpretation of data due to circadian variation in physiology. Measurements taken 12 h after the HIIT session were obtained in the morning ($\pm 06:30$) and therefore were not directly comparable to those measured immediately before the HIIT session ($\pm 17:30$). Therefore, 12 h data are expressed as changes from baseline (pre HIIT), and these changes are compared between conditions. Since the pre HIIT and 24 h variables were taken at the same time-of-day ($\pm 17:30$), they were compared directly. Correlations were performed using Pearson's product moment test and Spearman's rank correlation tests. Taking into account that (1) the typical error of measurement of the PPO test is 1% (Lamberts 2009), (2) the expected relative PPO in trained athletes is 5 W kg⁻¹ (Capostagno et al. 2014), and (3) sleep deprivation may reduce aerobic performance by 3% (Oliver et al. 2009), sample size was estimated based on an expected difference in change of peak power output between the two conditions of 4% (0.2 ± 0.1 W kg⁻¹). For an α level of 0.05 and a power of 95%, 15 participants were required. Data were analysed using Stata (v12, StataCorp, TX, USA). Significance was accepted when $p \leq 0.05$.

Results

Efficacy of the HIIT session

Participants reported similar amounts of sleep (CON 7.59 ± 1.10 h, DEP 7.88 ± 0.98 h, $p = 0.244$) and had similar weights (CON 77.4 ± 9.5 kg, DEP 78.3 ± 9.4 kg, $p = 0.369$) prior to the HIIT sessions. Twenty-four-hours after the HIIT session under the CON condition, cyclists felt less motivated to train ($p < 0.034$, $d = 2.07$), reported

more muscle soreness ($p = 0.014$, $d = 3.07$) and tiredness ($p < 0.001$, $d = 5.09$) and had lower resting SBP ($p = 0.011$, $d = 0.85$), absolute PPO ($p = 0.012$, $d = 0.44$), and HR_{max} ($p < 0.001$, $d = 2.00$) values. Collectively these data suggest that the HIIT session disturbed the cyclists' physiology, producing a stimulus from which they needed to recover.

Effects of partial sleep deprivation on recovery from exercise

Twelve hours after the HIIT session, participants were sleepier ($p = 0.001$), felt less motivated to train ($p = 0.018$) and demonstrated a blunted reduction in SBP ($p = 0.012$) under the DEP condition compared to the CON condition (Table 3). Figure 2 shows the individual 12 h responses, where data are expressed as percentage change (i.e. 12 h value minus Pre value) for the CON (open circles) and DEP (closed circles) conditions. Compared to before the HIIT session, all but two participants reported feeling sleepier 12 h after the HIIT session in the DEP condition compared to the CON condition (Fig. 2a). Ten participants felt less motivated to train after the HIIT under the DEP condition compared to the CON condition; for two cyclists the change in motivation to train after the HIIT was the same between conditions; and another two felt more motivated to train 12 h after the HIIT in the DEP condition compared to the CON condition (Fig. 2b). Twelve participants experienced a smaller reduction or even increase in SBP 12 h after the HIIT under the DEP condition, while four showed larger reductions in SBP under the DEP condition (Fig. 2c).

Twenty-four hour responses to the HIIT session are presented in Table 4 and Figs. 3 and 4. Differences between the conditions in sleepiness ($p = 0.004$) and SBP ($p = 0.043$) persisted 24 h after the HIIT session, accompanied by

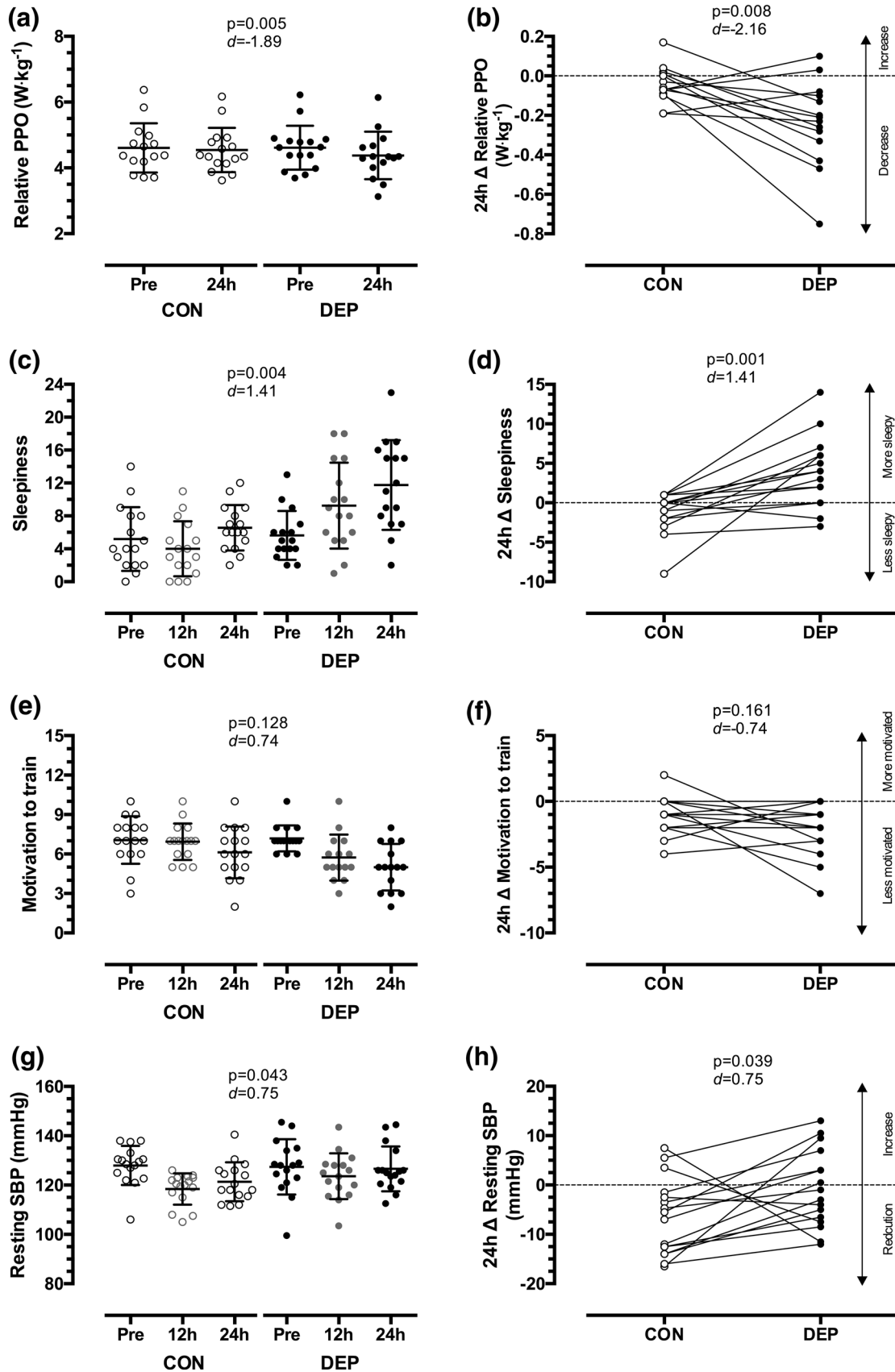


Fig. 3 Individual peak power output (**a, b**), sleepiness (**c, d**), motivation to train (**e, f**) and resting systolic blood pressure (**g, h**) responses to the HIIT session under the CON and DEP conditions. In the *left panel* (**a, c, e, g**), individual raw data are plotted for the pre HIIT, 12 and 24 h time points. The 12 h data are for visual purposes, and are not included in the statistical analysis. The *right panel* (**b, d, f, h**) compares individual responses to each condition, where each data point represents an individual's response to the HIIT session (24 h minus Pre), expressed as percentage change. *Open and closed circles* represent the normal sleep (CON) and partial sleep deprivation (DEP) conditions, respectively. The *dotted line* at $y=0$ (*right panel*) represents no change in the variable measured before and 24 h after the HIIT session (i.e. 0% change). The slope of the connecting line indicates the extent to which the change in a variable after HIIT was different in the DEP compared to the CON condition, in one individual. *PPO* peak power output, *DBP* systolic blood pressure, *Pre* prior to the HIIT session, *12 h* 12 h after the HIIT session, *24 h* 24 h after the HIIT session. Significance was determined using a two-way ANOVA with repeated measures (*left panel*) and a dependent *t* test (*right panel*). Cohen's *d* indicates effect size

a time-by-condition interaction effect for absolute PPO ($p=0.004$, $d=-1.98$, Table 4). Individual responses for relative PPO, sleepiness, motivation to train and SBP are plotted in Fig. 3. The left hand panel shows the absolute data for the Pre, 12 h and 24 h time points, and the right hand panel shows the 24 h percentage change in the variable (i.e. 24 h value minus Pre value) for the CON (open circles) and DEP (closed circles) conditions.

Relative PPO was lower 24 h after the HIIT session under the DEP condition compared to the CON condition ($p=0.005$, $d=-1.89$, Fig. 3a). Relative PPO was lower 24 h after the HIIT session under the DEP condition compared to the CON condition ($p=0.005$, $d=-1.89$, Fig. 3a). As indicated by the slope of the connecting line in Fig. 3b, 12 cyclists had greater reductions in relative PPO 24 h after the HIIT session under the DEP condition compared to the CON condition, three had greater reductions under the CON compared to the DEP condition and for one the reduction in PPO was not different between the conditions (Fig. 3b). As indicated by the slope of the connecting line in Fig. 3d, 15 cyclists were sleepier 24 h after the HIIT session under the DEP compared to the CON condition, while one was less sleepy 24 h after the DEP compared to the CON condition (Fig. 3d). All but three cyclists had smaller reductions or even increases in SBP 24 h after the HIIT session under the DEP condition compared to the CON condition (Fig. 3h). Motivation to train was no longer different between the groups ($d=0.74$, Table 4). Sleep duration was correlated with change in relative PPO, but was not significant for either condition (CON: $r=-0.21$, $p=0.457$, $n=16$; DEP: $r=-0.42$, $p=0.119$, $n=16$).

Individual responses for HR_{max} , VO_{2max} and RER_{max} reached during the PPO are plotted in Fig. 4. As for Fig. 3, the left hand panel shows the absolute data and the right hand panel shows the 24 h percentage change in the

variable. The extent to which HR_{max} was reduced at 24 h was not different between conditions (Fig. 4a), ($d=-0.81$). For all but three of the cyclists HR_{max} was lower during the PPO completed 24 h after the HIIT compared to before (Pre) under the DEP condition compared to the CON condition (Fig. 4b). Neither VO_{2max} nor RER_{max} changed differently in the DEP compared to the CON condition (Fig. 4c, e, respectively), and the individual responses were varied (Fig. 4d, f, respectively).

The cyclists had similar changes in CK activity, sIgA secretion rate and leukocyte cell counts at 12 and 24 h after the HIIT session under both sleep conditions (Online Resource 1, Figures S1 and S2, Tables S1 and S2, respectively). Time effects indicated that WBC ($p=0.020$) and neutrophil ($p<0.001$) counts were lower and basophil counts ($p<0.001$) higher 24 h after the HIIT session.

Discussion

Athletes have identified sleep as an important recovery strategy (Venter 2014; Kölling et al. 2016; Fullagar et al. 2016). This study provides some support for this concept. The main finding was that peak power output was reduced to a larger extent 24 h after the HIIT session when cyclists were partially sleep deprived compared to having had a normal night of sleep. In addition, the cyclists reported higher levels of sleepiness and less motivation to train, and the HIIT-induced reduction in resting systolic blood pressure the following day was blunted in the partial sleep deprivation condition. Collectively these data suggest that recovery from a HIIT session is compromised when followed by a single night of partial sleep deprivation, and that a night of normal sleep facilitates near full recovery in maximal performance capacity.

The extent to which relative peak power was decreased following the HIIT session in the DEP condition was 5%, compared to a reduction of only 1% in the CON condition. Alternatively, a normal night of sleep following HIIT may facilitate up to 99% of recovery from the training stimulus, while one night of partial sleep deprivation may limit this recovery to approximately 95%. Laboratory measurement of peak power output is routinely used to assess performance in cycling since it is well correlated with time-trial performance (Hawley and Noakes 1992). In terms of performance amongst elite cyclists, where a 1% alteration in performance is considered to be important (Currell and Jeukendrup 2008), the observed 5% reduction appears to be meaningful.

The variation in the extent to which peak power was affected in the recovery phase of the HIIT session under the partial sleep deprivation condition demonstrates individual sensitivity of physical performance to sleep loss. Twelve of

the 16 cyclists had greater reductions in peak power when partially sleep deprived, one experienced similar levels of power reduction between the two conditions, and one showed a smaller power decrement after partial sleep deprivation and two actually showed improvements in power after the sleep deprivation intervention.

Possible explanations for differences in susceptibility to sleep deprivation and the ensuing influence on maximal performance may relate to chronotype or habitual bed times or sleep lengths (Taillard et al. 2003). For example, evening-types may cope better with sleep deprivation compared to morning-types (Taillard et al. 2011; Barclay and Myachykov 2016). Individuals with a large sleep debt may appear to be less susceptible to the effects of one night of partial sleep deprivation, as their baseline performance may be impaired by their sleep debt. Change in relative peak power under the CON and DEP conditions were correlated against chronotype score, usual bed time and total sleep duration, however, no significant relationships were observed (data not shown). One explanation for this may be that these cyclists were more morning-oriented than the general South African population (Kunoroza et al. 2012; Henst et al. 2015) and lacked any evening-types. Thus the potentially moderating role of chronotype on sleep deprivation and the ensuing recovery could not be assessed. Future work would need to be done in this area to test this concept.

The 24 h reduction in peak power observed in both conditions occurred in the absence of any change in VO_{2max} . This suggests a dissociation between VO_{2max} and peak power, and potentially a lower gross efficiency (Noordhof et al. 2010) 24 h after the HIIT session; an effect which was not altered by recovery sleep length. Neither did sleep length appear to moderate the reduction in maximum RER observed 24 h after the HIIT session. Together these data suggest that any perturbances to the metabolic system induced by the HIIT were able to recover similarly regardless of sleep length.

An acute, exhausting bout of exercise, such as HIIT, is understood to both increase blood volume (Convertino 1991) and alter the sympathovagal balance in favour of the parasympathetic system (Bellenger et al. 2016). Thus when physically tired or unrecovered individuals exercise maximally, they typically reach lower maximum heart rates (Le Meur et al. 2013), indicative of reduced sympathetic drive. The reductions of resting SBP and maximum heart observed in the HIIT recovery phase following normal sleep in the present study are understood to reflect these changes, and have been shown previously (Tochikubo et al. 1996).

In the partial sleep deprivation condition, however, the HIIT-induced reduction in resting SBP did not occur. In contrast to a fatiguing bout of exercise, acute sleep deprivation is thought to increase sympathetic and decrease

parasympathetic contributions to cardiovascular modulation (Zhong 2005). One of the ways in which this could manifest is via an increase in resting SBP the day after sleep deprivation (Lusardi et al. 1996). Therefore, one might hypothesise that the smaller post-HIIT session reduction in resting SBP pressure observed in this study under the DEP condition suggests that any exercise-induced increase in parasympathetic activity may have been marginalised by the sleep deprivation-induced increase in sympathetic activity. Future research investigating the potential role of the autonomic nervous system on physical recovery in the sleep deprived state is warranted.

Both intense exercise in athletes and sleep deprivation are understood to decrease innate and acquired immunity (Faraut et al. 2012; Walsh and Oliver 2016). Although this study observed some blood leukocyte changes 24 h after the HIIT, likely representative of trafficking (Dhabhar 2002), the extent to which this occurred was not different between the conditions. Therefore perturbation of the immune system does not appear to have a role in recovery from a single bout of HIIT under a condition of partial sleep deprivation.

Although not measured in this study, the longer term consequences of repeated nights of partial sleep deprivation on recovery should be considered. Not only could adaptation to training be compromised, but repeated training sessions with insufficient sleep in the recovery phase may increase the risk for non-functional overreaching and ultimately overtraining (Lehmann et al. 1997). To improve performance, athletes finely balance training stimulus with recovery; since increasing training load elicits a greater adaptation (functional overreaching), while insufficient recovery results in a maladaptive response (non-functional overreaching) (Meeusen et al. 2013). Longer term non-functional overreaching is understood to eventually lead to a state of overtraining, in which accumulated training stresses result in long term performance decrements and fatigue, recovery from which may take several weeks (Meeusen et al. 2013). The data from this study suggest that inadequate sleep following a single high load training session increases the time needed to recover. Therefore, one might speculate that regular short sleep patterns during high load training periods may result in non-functional overreaching. In contrast, athletes with sufficient sleep during such periods may be more likely to remain in the functional overreaching zone, maximising adaptation to training.

A limitation to this study is that neither heart rate variability nor catecholamine levels were measured to assess alterations in autonomic nervous system function. Furthermore, given the relatively small sample size, and fairly large variation in ability of these cyclists, the results need to be interpreted with caution. Another limitation is that two of the participants consumed one caffeine beverage each in

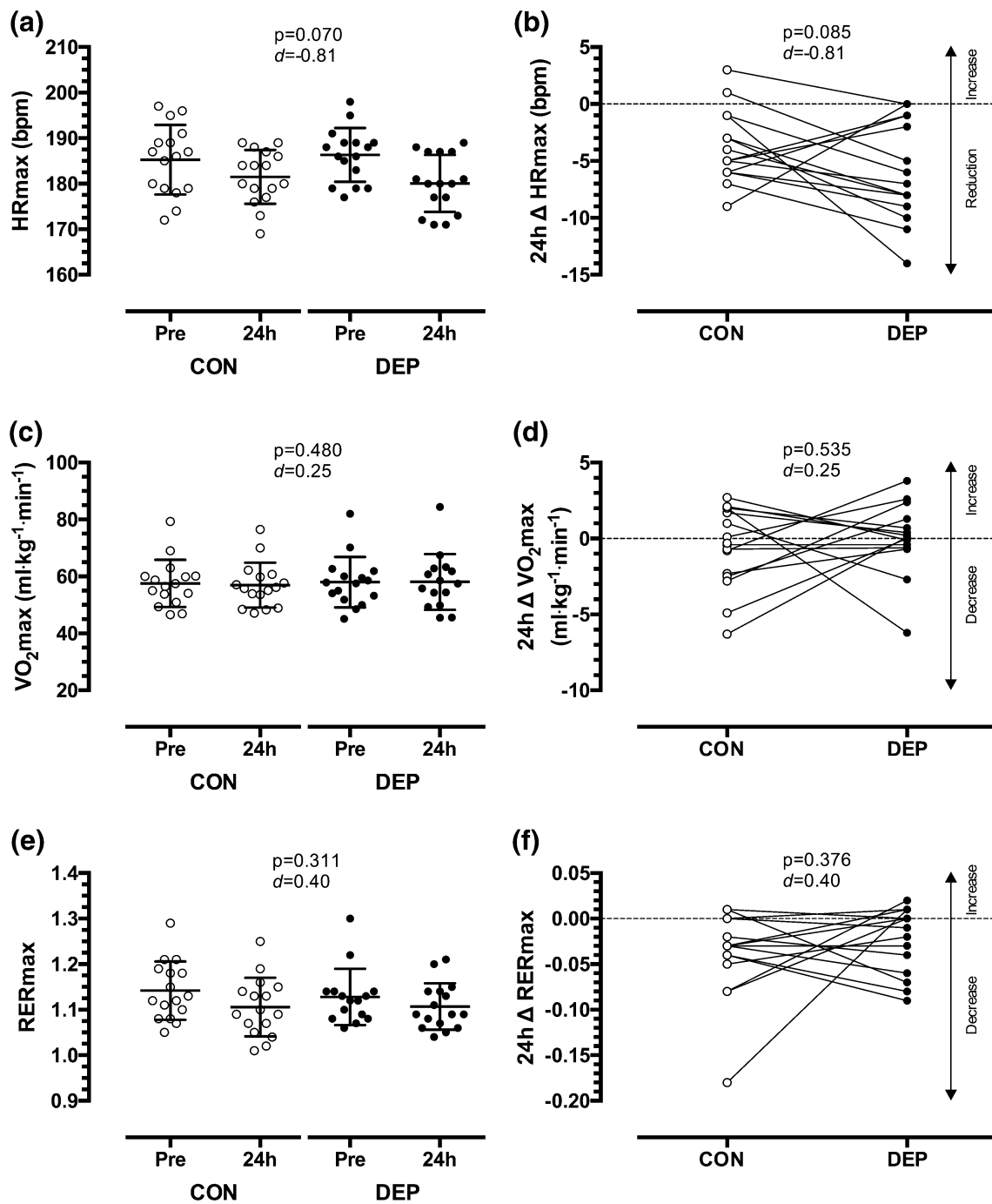


Fig. 4 Individual maximum heart rate (a, b), oxygen uptake (c, d) and respiratory exchange ratio (e, f) responses to the HIIT session under the CON and DEP conditions. In the *left panel* (a, c, e), individual raw data are plotted for the pre HIIT and 24 h time points. The *right panel* (b, d, f) compares individual responses to each condition, where each data point represents an individual’s response to the HIIT session (24 h minus Pre), expressed as percentage change. *Open and closed circles* represent the normal sleep (CON) and partial sleep deprivation (DEP) conditions, respectively. The *dotted line* at $y=0$ (*right panel*) represents no change in the variable measured

before and 24 h after the HIIT session (i.e. 0% change). The slope of the *connecting line* indicates the extent to which the change in a variable after HIIT was different in the DEP compared to the CON condition, in one individual. HR_{max} maximum heart rate, VO_{2max} maximum oxygen uptake volume, RER_{max} maximum respiratory exchange ratio, *Pre* prior to the HIIT session, *24 h* 24 h after the HIIT session. Significance was determined using a two-way ANOVA with repeated measures (*left panel*) and a dependent *t*-test (*right panel*). Cohen’s *d* indicates effect size

the 24 h period prior to their Baseline session, following which they were partially sleep deprived. Ingested caffeine may enhance physical performance, although the effects are varied and depend on factors such as dose, form, individual tolerance to caffeine and type of exercise performed (Rogers and Dinges 2005; Spriet 2014). Therefore the baseline performance of these two athletes may have been artificially raised. The performance analysis for this study was repeated excluding these two individuals, but the results for peak and absolute power remained the same (data not shown). Although individuals with diagnosed sleep disorders were excluded from this study, the WASO and number of awakenings measured using the Actiwatch during the participant's usual sleep are higher than expected. While these data cannot be used diagnostically, it is possible that some of these individuals may have had an undiagnosed sleep disorder. Alternatively, these variables measured with the Actiwatch are not directly comparable to those obtained using polysomnography.

Conclusions

While there is a significant body of work describing the effects of sleep deprivation on physical performance, few studies have investigated the consequences of limited sleep on recovery from exercise. This study confirms a role for sleep in recovery from training and contributes to our understanding of the physiological effects of partial sleep deprivation on recovery. It differs from and is complementary to previous similar studies (McMurray and Brown 1984; Skein et al. 2013) in that it used a model of partial sleep deprivation for only one night and tested endurance sports athletes recovering from a single training session.

From the perspective of athletes and coaches, these data suggest that recovery of the systems needed to produce maximal efforts is incomplete when an athlete does not obtain a full night of sleep following a HIIT session. The consequence of this may be that if a training session is scheduled for the following day, the athlete may not be able to work at a high intensity. In part, this may be reinforced by the athlete's lower level of motivation to train following sleep restriction. In periods of training where intensity is more important than duration, an inability to train at the prescribed intensity may mean that a lower training stimulus is applied, and as a consequence, the level of adaptation induced might be lower.

- Future research in this area could investigate individual sensitivity to sleep deprivation on recovery, paying particular attention to the role of chronotype. Given that there is some evidence to suggest that a number of athletes suffer from poor quality sleep, the effect of frag-

mented sleep on recovery deserves attention. Finally, it remains to be determined whether or not chronic insufficient sleep in the recovery phase of training predisposes an athlete to worsened performance or non-functional overreaching.

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Compliance with ethical standards

Conflict of interest The authors have no conflicts of interest to declare.

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