

Title: High-intensity interval exercise induces greater acute changes in sleep, appetite-related hormones and free-living energy intake compared to moderate-intensity continuous exercise

Authors: Penelope Larsen¹, Frank Marino¹, Kerri Melehan^{2,3}, Kym J Guelfi⁴, Rob Duffield⁵, Melissa Skein¹

Affiliation: ¹School of Exercise Science, Sport and Health, Charles Sturt University, Bathurst, Australia;

²Royal Prince Alfred Hospital, Sydney;

³Discipline of Sleep Medicine, University of Sydney, Sydney, Australia;

⁴School of Human Sciences, University of Western Australia, Perth, Australia;

⁵Sport and Exercise Discipline Group, University of Technology Sydney, Australia.

Correspondence: Penelope Larsen

School of Exercise Science, Sport & Health

Charles Sturt University

Panorama Avenue, Bathurst, Australia, 2795

Phone: +61 2 6338 6101

Email: plarsen@csu.edu.au

Email Address: F Marino: fmarino@csu.edu.au

K Melehan: kerri.melehan@sydney.edu.au

K Guelfi: kym.guelfi@uwa.edu.au

R Duffield: Rob.Duffield@uts.edu.au

M Skein: mskein@csu.edu.au

Abstract

Compare the effect of high-intensity interval and moderate-intensity continuous exercise on sleep characteristics, appetite-related hormones and eating behaviour. 11 overweight, inactive men completed two consecutive nights of sleep assessments to determine baseline (BASE) sleep stages and arousals recorded by polysomnography (PSG). On separate afternoons (1400-1600h), participants completed a 30min exercise bout: 1) moderate-intensity continuous exercise (MICE; 60% $\dot{V}O_{2peak}$) or 2) high-intensity interval exercise (HIIE; 60s work at 100% $\dot{V}O_{2peak}$; 240s rest at 50% $\dot{V}O_{2peak}$), in a randomised order. Measures included appetite-related hormones (acylated ghrelin, leptin, peptide tyrosine tyrosine) and glucose pre-exercise, 30min post-exercise, and the next morning post-exercise; PSG sleep stages, actigraphy (sleep quantity and quality), and self-reported sleep and food diaries were recorded until 48h post-exercise. There was no between-trial differences for time in bed ($p=0.19$) or total sleep time ($p=0.99$). For HIIE, stage N3 sleep was greater ($21 \pm 7\%$) compared to BASE ($18 \pm 7\%$; $p=0.02$). Also, number of arousals during rapid eye movement sleep were lower for HIIE (7 ± 5) compared to BASE (11 ± 7 ; $p=0.05$). Wake after sleep onset was lower following MICE (41min) compared to BASE (56min; $p=0.02$). Acylated ghrelin was lower and glucose higher at 30min post-exercise for HIIE compared to MICE ($p \leq 0.05$). There were no significant differences in total energy intake between conditions ($p \geq 0.05$). HIIE appears more beneficial than MICE for improving sleep quality and inducing favourable transient changes in appetite-related hormones in overweight, inactive men. However, energy intake was not altered regardless of exercise intensity.

Keywords: Acute exercise, high-intensity intermittent exercise, sleep stages, polysomnography, appetite regulation, appetite behaviour

Introduction

Sleep is an essential physiological occurrence required for optimal cognitive performance and metabolic functioning (Spiegel et al. 2004; Alhola and Polo-Kantola 2007). Nevertheless, at least one third of adults do not achieve sleep recommendations (i.e. 7-9 h per night) (Hirshkowitz et al. 2015), in part due to increasing work demands and domestic responsibilities (Rajaratnam and Arendt 2001; Bei et al. 2016). These chronic reductions in sleep quantity are associated with alterations in the circadian rhythms of key regulatory hormones resulting in increased body mass, impaired metabolism, altered calorie intake, and perception of appetite (Lauderdale et al. 2006; Nedeltcheva et al. 2009; Watanabe et al. 2010; McNeil et al. 2017). Furthermore, sleep quality as determined by the proportion of stage N3 sleep (i.e. deep sleep), rapid eye movement (REM) sleep, and sleep continuity (Copinschi et al. 2014; Adams et al. 2017) appears to decline with age (Alves et al. 2011). Given that exercise is believed to promote sleep quality, it is plausible that age-associated effects on sleep may be dampened following exercise; however, much of the previous literature has recruited young adults with minimal sleep complaints (Youngstedt 2005). Therefore, the effects of exercise on sleep patterns in middle-aged to older adults remains unclear.

Regular exercise, irrespective of exercise intensity or mode, has been shown to modestly increase sleep duration, stage N3 sleep and REM onset latency (Youngstedt 2005). Independently, exercise also enhances appetite regulation (Martins et al. 2008) and sensitivity to the signalling of orexigenic and anorexigenic hormones (Dyck 2005). However, there is ongoing interest regarding the specific exercise intensity that is most beneficial for both sleep and appetite responses (Dworak et al. 2008; Broom et al. 2009; Hayashi et al. 2014; Sim et al. 2014). Examination of high-intensity exercise specifically suggests an increase in stage N3 sleep and reduces sleep onset latency (SOL) and wake after sleep onset (WASO) primarily within adolescents and young adults (Kredlow et al. 2015), while the effect in middle-aged populations is largely unexplored. For appetite, high-intensity exercise has been

associated with the downregulation of orexigenic signals (e.g. acylated ghrelin) and upregulation of anorexigenic signals (e.g. leptin, peptide tyrosine tyrosine: PYY, and glucose) which may lead to reduced perceived hunger and energy intake in overweight, inactive men for up to 24 hours compared to moderate-intensity exercise (Sim et al. 2014). However, the effect of these differing exercise intensities on sleep and appetite have not been examined concurrently. This is important given that shifts in sleep may alter the amplitude and circadian variation of appetite hormones, such as leptin and ghrelin (Spiegel et al. 2004; Copinschi et al. 2014). For instance, acute sleep restriction (e.g. 4 - 5.5 hours per night) has been linked to elevations in circulating ghrelin and reduced levels of leptin leading to increased feelings of hunger and desire for calorie-dense foods (Spiegel et al. 2004) and overall energy intake (Nedeltcheva et al. 2009).

To date, previous literature has focussed on the association between sleep and appetite regulation (Spiegel et al. 2004; Magee et al. 2009; Nedeltcheva et al. 2009; St-Onge et al. 2012); acute exercise effects on sleep quality and quantity (Kredlow et al. 2015); or acute exercise effects on appetite regulation (Sim et al. 2014; Panissa et al. 2016; Holliday and Blannin 2017). Accordingly, it may be important to now investigate the role of exercise on sleep and appetite simultaneously considering the potential interaction between these three major behaviours. Hence, the aim of this study was to compare the effect of high-intensity interval exercise (HIIE) and traditional moderate-intensity continuous exercise (MICE) on sleep characteristics, appetite-related hormones and free-living energy intake in inactive, middle-aged men. It was hypothesised that both exercise intensities would improve sleep duration and quality compared to a resting baseline, but HIIE would be more beneficial to sleep (increased stage N3 sleep and reduced arousals) and appetite parameters (anorexigenic changes in the circulating hormones and reduced energy intake) compared to MICE.

Methods

Participants

Eleven overweight, inactive men (mean \pm SD; age: 49 ± 5 y; BMI: 28 ± 3 kg·m⁻²; waist-to-hip ratio (WHR): 0.92 ± 0.05 ; $\dot{V}O_{2\text{peak}}$: 34 ± 8 ml·kg⁻¹·min⁻¹) completed this study. Initially, 13 men volunteered to participate in the study; however, one participant was excluded due to signs of sleep apnoea and one participant withdrew due to illness unrelated to the study. Inclusion/exclusion criteria included non-smokers, participating in < 150 min of moderate-intensity exercise per week, had no previous or current diagnosis of sleep or metabolic disorders, and no medical conditions or medications that affect sleep quality or quantity. Sleep was initially assessed by the STOP-BANG questionnaire (Chung et al. 2008) and the Epworth Sleepiness Scale (Johns 1991). Risk of sleep apnoea was further assessed by two consecutive nights of polysomnography (PSG) sleep studies. Medical clearance was obtained from a General Practitioner and a Pre-Exercise Medical Health Questionnaire was completed by each participant prior to participating in the study to ensure no underlying conditions would be exacerbated by vigorous exercise. The study was approved by the Institution's Human Ethics Committee and written informed consent was attained from all participants prior to data collection.

Experimental Overview

Participants attended the laboratory for an initial familiarisation session and baseline assessments of anthropometry and peak oxygen consumption ($\dot{V}O_{2\text{peak}}$). Habitual sleep and eating patterns were also documented for 7 days and nights (refer to Figure 1). During this time, the two consecutive nights of PSG sleep assessments were conducted to exclude sleep apnoea and determine baseline sleep staging and arousals. Following baseline, participants completed two experimental trials (4 days in duration each) in a randomised order. The experimental trials included either 30 min of moderate-intensity continuous exercise (MICE; 60% $\dot{V}O_{2\text{peak}}$) or 30 min of high-intensity interval exercise (HIIE; 60 s at

100% $\dot{V}O_{2\text{peak}}$: 240 s at 50% $\dot{V}O_{2\text{peak}}$). The total mechanical work performed for each exercise protocol was matched (Sim et al. 2014). Experimental trials were performed at the same time of day, with a minimum of 5 days between visits. Primary outcome measures included post-exercise sleep quality and quantity, changes in plasma concentrations of appetite-related hormones, ratings of perceived appetite, and post-exercise free-living energy intake.

Familiarisation and Baseline Testing

The familiarisation session involved assessment of height and body mass to calculate body mass index (BMI), and waist and hip girths to calculate waist-to-hip ratio (WHR). In addition, $\dot{V}O_{2\text{peak}}$ was assessed using a ramp protocol (Barstow et al. 2000) on a stationary cycle ergometer (Lode B.V., Excalibur Sport, Groningen, The Netherlands) to calculate workloads for the experimental trials. The $\dot{V}O_{2\text{peak}}$ test commenced at 50 W for the first 2 min and increased 25 W every minute thereafter with cadence maintained at 70 rpm until volitional exhaustion. During the test, heart rate (HR; F1, Polar, Electro-Oy, Kempele, Finland) was monitored every minute and breath-by-breath pulmonary gas exchange was obtained via a mouthpiece connected to a calibrated metabolic gas oxygen analysis system and custom-developed software (LabVIEW; National Instruments, Austin, TX, USA).

Baseline at-home testing was completed for a total of 7 days and nights at which time participants were fitted with a wrist actigraph (Actiware 2, Philips Respironics, Andover, MA), and documented sleep and food intake in a diary provided for the duration of baseline testing. During this time, participants were instructed to maintain usual bed time, wake-up time, and diet. These data were obtained to provide a representation of habitual sleep quantity and diet given the day-to-day variations associated with these factors (Champagne et al. 2013; Bei et al. 2016). The two PSG sleep

studies using a level II, take home PSG device were conducted during the 7 night baseline period to exclude sleep disorders and record baseline sleep stages and arousals.

Experimental Trials

During each experimental trial, participants did not engage in physical activity and documented all food and drink consumption 24 hours prior to exercise. On the day of exercise, participants abstained from alcohol and caffeine; and fasted for 3 hours before arriving to the laboratory between 1400-1600 h. Upon arrival, participants were asked to indicate perceived hunger and fullness on validated Visual Analogue Scales (VAS) (Flint et al. 2000) and a capillary blood sample was obtained from the fingertip for the assessment of appetite-related hormones and glucose. Participants then performed the 30 min MICE protocol or HIIE protocol. Exercise was performed on a stationary cycle ergometer (Wattbike Trainer, Wattbike Ltd, Nottingham, UK) and intensity was monitored via power output (PO) and HR (F1, Polar, Electro-Oy, Kempele, Finland) responses every minute. Participants also reported rating of perceived exertion (RPE; 1-10 scale) (Borg 1982) every 5 min. Immediately post-exercise, participants were instructed to sit quietly for 30 min after which time a second blood sample was obtained and perceived appetite was recorded to assess the acute exercise effects on appetite variables. Following exercise, nocturnal sleep was recorded using a level II, take home PSG device and scored for sleep stages and arousals (details below). Participants returned to the laboratory the following morning (within 60 min after waking), for a fasted capillary blood sample and reported perceived appetite to examine appetite variables in relation to the preceding night's sleep. Actigraphy, and sleep and food records were maintained for 3 days during each trial, including the day of exercise, one day after exercise, and two days after exercise (refer to Figure 1). Data were examined for sleep quantity and energy intake up to 48 hours post-exercise. Following exercise, participants were free to choose bed times, wake-up times, and food intake to observe sleep and eating responses to the respective trials.

Polysomnography

Polysomnography was performed using recommended electrode and sensor placements (Berry et al. 2016), connected to an Alice PDx system (Philips Respironics, Pittsburg, USA) and analysed using Sleepware G3 software version 3.7.4 (Philips Respironics, Pittsburg, USA). Electrode and sensor placements included: three electroencephalogram (EEG; F3-A2, C4-A1, and O1-A2) electrodes, unilateral electrooculogram (EOG), chin electromyography (EMG), electrocardiography (ECG; lead I), oxygen saturation via pulse oximetry, thoracic and abdominal respiratory effort via belts, and nasal airflow via pressure transducer. The baseline sleep studies were scored to exclude sleep disorders and data were used for the baseline sleep staging and arousal parameters. Sleep studies during experimental trials were only assessed for sleep staging and arousals. All sleep studies were scored using standard guidelines (Berry et al. 2016) by an experienced sleep technician who was blinded to the experimental trials. Sleep parameters assessed included time in bed, total sleep time (TST), sleep efficiency (SE) $[(\text{sleep duration} - \text{wake time}) / \text{sleep duration}] \times 100$, sleep onset latency (SOL: time from lights out to the first epoch of sleep), rapid eye movement (REM) onset latency, wake after sleep onset (WASO: total time awake after sleep onset), percent of time spent in each sleep stage (N1: stage 1; N2: stage 2; N3: stage 3; NREM: non-rapid eye movement sleep; REM), and number of arousals (NREM, REM and total arousals).

Actigraphy

Actigraphy was recorded in 1 min epochs (Esliger and Tremblay 2006) and analysed using Actiware v5.70 software (Philips Respironics, Pittsburgh, USA). Variables obtained included bed time, wake-up time, time in bed (period between bed time and wake time), TST (time asleep during time in bed), SOL (period between bed time and sleep onset), SE (percent of time in bed spent sleeping), WASO (total time awake after sleep onset), and number of awakenings (Knutson et al. 2007).

Appetite Perception and Hormones

Perceived hunger and fullness were assessed using a VAS comprised of straight lines (100 mm) accompanied by a question anchored with words representing opposing extreme states of hunger and fullness at either end (Flint et al. 2000). A 600 µl blood sample was obtained from a fingertip using a sterile lancet. To assist vasodilation, the hand was submerged in a bowl of warm water for 5 min prior to blood draw. Blood glucose concentration was measured directly from the fingertip using an Accu-Chek Performa (Roche, Mannheim, Germany). The remaining blood was immediately aliquoted into pre-chilled EDTA tubes (Becton Dickinson, Sydney, Australia) treated with serine protease inhibitor (25 µl per 600 µl of blood; Pefabloc® SC, Sigma-Aldrich, St. Louis, USA) then centrifuged at 3000 rpm for 10 min. Plasma obtained was stored at -80°C and later analysed according to manufacturer's instructions for acylated ghrelin, leptin and peptide tyrosine tyrosine (PYY_{total}) using a commercially available assay kit (Milliplex, Millipore Corporation, MA, USA). These hormones were chosen due to their role in hunger and satiety signalling, responsiveness to exercise (Broom et al. 2009; Balaguera-Cortes et al. 2011) and association with sleep changes (Spiegel et al. 2011). For acylated ghrelin, leptin and PYY_{total} the intra- and inter-assay coefficient of variations were < 10% and < 15%.

Sleep and Energy Intake Records

A self-reported diary for sleep, and food and drink intake, was provided to participants. Sleep records were used to confirm bed time and wake-up time for actigraphy data. For food records, instructions on the use (including a 1 day example), and the necessity for accurate (i.e. food and drink brands and quantities) and detailed recordings of energy intake immediately after consumption were emphasised. Total energy and macronutrient intake were calculated using commercially available

software (Foodworks; Xyris Software, Kenmore Hills, QLD, Australia). Also, absolute (g) and relative data (%) were calculated for carbohydrate, fat and protein intake.

Statistical Analysis

A *priori* sample size calculations for a repeated measures ANOVA was performed using G*Power (version 3.1.9.2) which confirmed that a sample size of 8 would provide an actual power of 94%, therefore the final sample size of 11 was adequate for the input parameters (Nilius et al. 2017). Order effect analysis was also completed and indicated no significant difference between trial 1 and trial 2 for PSG total sleep time or wake after sleep onset ($p \leq 0.14$); however, sleep efficiency was significant ($p = 0.006$). A repeated measures (trial \times time interaction) ANOVA with Tukey's LSD *post hoc* were used to compare physiological and perceptual measures, perceived appetite, appetite-related hormones and glucose, total and macronutrient energy intake, PSG and actigraphy variables between trials. PSG data were further separated to analyse the initial 180 min after sleep onset as the first 1-2 sleep cycles have been shown to be altered by acute stimuli including high-intensity exercise (Netzer et al. 2001; Myllymäki et al. 2012). For total and macronutrient energy intake, two analyses were conducted to compare the difference between MICE and HIIE immediate post-exercise (i.e. acute: energy intake for the remainder of the day following exercise) and to compare differences between BASE, MICE and HIIE (i.e. over the total 48 h period of monitoring). Analyses were performed using Statistical Package for Social Sciences (SPSS v 20.0, Chicago, USA). Data are reported as mean \pm standard deviation (SD) and statistical significance was accepted at $p \leq 0.05$.

Results

Exercise Characteristics

The mean heart rate for MICE and HIIE were 126 ± 10 bpm and 132 ± 10 bpm, respectively. Heart rate responses during HIIE were higher compared to MICE at 1-6, 10-11 min, 15-16 min, 20-21 min, 25-26 min, and 30 min ($p \leq 0.04$; Figure 2A). Mean RPE was significantly lower for MICE (3 ± 1) compared to HIIE (7 ± 2 ; $p = 0.001$; Figure 2B). Higher RPE was reported following all HIIE sprint intervals compared to the corresponding times for MICE ($p \leq 0.001$; Figure 2B).

Polysomnography and Actigraphy

Whole night and initial 180 min polysomnography data are presented in Table 1. There were no significant differences for time in bed, total sleep time, sleep efficiency, sleep onset latency, or N1, N2, NREM and REM sleep between BASE, MICE and HIIE ($p > 0.05$). However, there was a significant decrease in wake after sleep onset following MICE compared to BASE ($p = 0.02$). Also, the proportion of N3 sleep was higher ($p = 0.02$), while the number of arousals were lower during REM sleep ($p = 0.05$) for HIIE compared to BASE. There were no differences for NREM arousals ($p = 0.59$) or total arousals between BASE, MICE or HIIE ($p = 0.64$). When the initial 180 min PSG was considered, the proportion of NREM sleep was higher and REM sleep was lower after HIIE compared to BASE ($p = 0.02$). The number of arousals during REM sleep was also decreased for HIIE compared to BASE ($p = 0.03$). There were no differences for NREM arousals ($p = 0.21$) or total arousals between BASE and both exercise trials ($p = 0.36$). There were no between-trial differences for all other sleep parameters assessed ($p > 0.05$).

Actigraphy data indicated time in bed was longer the night after MICE compared to HIIE ($p = 0.02$), however, there was no significant differences between trials for any other actigraphy variables ($p > 0.05$).

Perceived Appetite and Appetite-Related Hormones

There was no trial \times time interaction for perceived hunger ($p = 0.29$; Figure 3A) or fullness ($p = 0.73$; Figure 3B). However, there was a main effect of time for both trials whereby hunger was higher and fullness was lower the morning after-exercise compared with pre-exercise ratings ($p \leq 0.02$).

The hormone and glucose responses to MICE and HIIE are shown in Figure 4. There was a trial \times time interaction for acylated ghrelin, with post hoc analyses revealing significantly higher acylated ghrelin pre-exercise for HIIE compared to MICE ($p = 0.001$), and lower ghrelin at 30 min post-exercise for HIIE compared to MICE ($p = 0.03$; Figure 4A). There was also a trial \times time interaction for glucose, with higher concentrations at 30 min post-exercise for HIIE compared to MICE ($p = 0.02$; Figure 4D). There was no trial \times time interaction for leptin or PYY_{total} ($p > 0.05$), although there was a main effect of time for leptin with higher concentrations the morning post-exercise compared to 30 min post-exercise ($p = 0.05$; Figure 4B).

Free Living Energy Intake

Energy intake for the remainder of the day after exercise for HIIE (4281 ± 1822 kJ) was lower than MICE (5273 ± 2589 kJ); however, this was not statistically significant ($p = 0.55$). The contribution of carbohydrate (MICE: $39 \pm 12\%$; HIIE: $33 \pm 14\%$; $p = 0.09$) and protein (MICE: $17 \pm 6\%$; HIIE: $13 \pm 5\%$; $p = 0.09$) to energy intake was similar between trials for the remainder of the day following exercise. In addition, there was no difference in sodium (MICE: 1747 ± 1289 mg; HIIE: 1056 ± 918 mg; $p = 0.16$) or sugar intake (MICE: 49 ± 45 g; HIIE: 34 ± 30 g; $p = 0.10$). Likewise, absolute fat intake was similar between trials; however, the proportion of energy intake from fat was higher following HIIE ($42 \pm 7\%$) compared to MICE ($34 \pm 11\%$; $p = 0.04$) for the remainder of the day following exercise.

Energy and macronutrient intake for BASE, and two days after the day of MICE and HIIE (i.e. day+1 and day+2) are presented in Table 2. Relative fat intake for the day following exercise was significantly greater for HIIE compared to MICE ($p = 0.03$). Absolute carbohydrate intake for MICE on the day of exercise was higher compared to BASE ($p = 0.04$); but lower at two days post-exercise compared to BASE ($p = 0.05$). Moreover, relative carbohydrate intake for the day following MICE was higher compared to BASE ($p = 0.03$); while for two days after exercise, MICE was higher compared to HIIE ($p = 0.03$). Absolute protein intake was higher on the day of MICE compared to BASE ($p = 0.04$); although, one day following MICE intake was lower compared to BASE ($p = 0.04$). On the day of MICE and HIIE, and two days after HIIE, sodium intake was higher compared to BASE ($p \leq 0.03$). There were no further trial \times time interactions for energy intake ($p = 0.61$), macronutrient intake ($p \geq 0.07$) or caffeine ingestion ($p = 0.54$). There was a main effect of time for sugar intake, with reduced consumption from the day of exercise until the two days following exercise ($p = 0.02$).

Discussion

This study investigated the effect of high-intensity interval exercise compared to traditional moderate-intensity continuous exercise on sleep characteristics, appetite responses and subsequent free-living energy intake in overweight, inactive men. The novel design of the study allowed for a simultaneous examination of sleep and appetite responses following two popular exercise modalities. It appears that HIIE induced an increased proportion of stage N3 sleep and total NREM sleep, and reduced REM sleep and arousals during REM sleep compared to BASE; while only lower wake after sleep onset was observed following MICE compared to BASE. Also, circulating acylated ghrelin was lower and glucose concentrations were higher transiently after HIIE compared to MICE, suggesting a favourable hormonal milieu for reduced energy intake. However, these changes were not associated with significant alterations in total energy intake either acutely (i.e. for the remainder of the day following

exercise) or chronically (i.e. for the 2 days following exercise). Collectively, these findings indicate that HIIIE may have a greater positive influence on sleep and appetite-related hormones compared to MICE. However, the alterations in acute sleep and appetite regulation may not be reflected by changes in perceptual measures (hunger and satiety) or behaviours (sleep hygiene and dietary choices).

The observed changes in sleep following exercise are consistent with previous research that has examined various populations. Specifically, greater stage N3 sleep and improved sleep efficiency have been observed, in conjunction to decreased sleep onset latency, wake after sleep onset and number of arousals (Bunnell et al. 1983; Horne and Staff 1983; Dworak et al. 2008; Passos et al. 2010; Flausino et al. 2012; Wong et al. 2013; Hayashi et al. 2014). However, several studies have also indicated decreased REM sleep (Passos et al. 2010; Flausino et al. 2012; Wong et al. 2013; Hayashi et al. 2014). In contrast, despite a reduction in REM sleep in the initial 180 min of sleep, there was no difference in REM sleep across the whole night suggesting that after HIIIE, a greater proportion of REM sleep was experienced later in the night, compared to baseline sleep. Robey et al. (2013) observed similar changes in REM sleep following vigorous evening exercise for highly trained cyclists. While the cause of the change in distribution warrants further investigation, Netzer et al. (2001) investigated a potential aminergic effect following intense exercise in highly trained endurance athletes whereby a similar decrease in the proportion of REM sleep in the first half of sleep was observed. Authors reported that an extension of REM onset latency and reduction in REM sleep percentage correlated with an increase of norepinephrine and epinephrine, suggesting that the autonomic nervous system plays a key role in the regulation of REM sleep (Netzer et al. 2001). Nonetheless, further research is required to examine the influence of catecholamine excretion during exercise on subsequent sleep and whether age- and fitness-related factors alter the effects.

A possible explanation for the improved sleep following HIIE may be the increased physiological stress associated with high-intensity exercise compared to moderate-intensity exercise (Burgomaster et al. 2005; Helgerud et al. 2007; Wisløff et al. 2007; Crisp et al. 2012). In contrast to MICE, high-intensity exercise induces rapid increases in heart rate, release of metabolic hormones (e.g. growth hormone), lactate, and depletion of adenosine triphosphate, creatine phosphate and glycogen stores (Weinstein et al. 1998; Tomlin and Wenger 2001; Trapp et al. 2007; Boutcher 2010). Consequently, recovery may be extended, and oxygen consumption remains elevated post-exercise to accelerate the return of metabolic processes to a resting state (Laforgia et al. 2006; Boutcher 2010). Given the vigorous nature of our HIIE protocol, it is plausible that participants experienced greater glycogen depletion and metabolite accumulation which required a longer recovery compared to the MICE trial. Simultaneously, untrained individuals have been shown to experience slower rates of recovery compared to trained counterparts (Gore and Withers 1990; Børsheim and Bahr 2003). As such, the deconditioned state of the current cohort may have exacerbated the physiological effects of high-intensity exercise, resulting in an extended recovery time. Given the nature of stage N3 sleep to restore and repair peripheral tissue, it is plausible that the observed increase in stage N3 sleep after HIIE was associated with a greater need for body restoration, and vital growth and repair (Tasali et al. 2008).

In addition to sleep changes, HIIE induced transient changes in appetite-related hormones and glucose that would appear favourable for reducing energy intake compared to MICE. More specifically, decreased ghrelin and increased glucose were observed 30 min after exercise for the HIIE trial. The lower ghrelin following HIIE occurred despite significantly higher circulating ghrelin prior to exercise, suggesting the magnitude of decrease in ghrelin with HIIE was substantial. The reason for the higher ghrelin prior to the HIIE trial is unclear. All participants complied with the fasting requirements of this study, suggesting that the difference may simply reflect the considerable intra- and inter-individual

variation of acylated ghrelin despite controlling for energy intake as reported in the literature (Spiegel et al. 2011). Furthermore, given that increases in circulating glucose stimulate the release of insulin and act centrally to increase satiety and blunt the food reward response (Flint et al. 2007; Page et al. 2013), the higher glucose response to HIIE compared to MICE may also contribute to a potential downregulation of appetite. The present results are consistent with those of Sim et al. (2014) who observed a significant decline in ghrelin and increase in glucose acutely following high-intensity exercise compared with a bout of traditional moderate-intensity exercise. These responses were observed despite a greater fasting time (overnight 10 h fast) compared to the current study, and exercise being performed in the morning instead of the afternoon. As such, these two studies suggest that high-intensity exercise significantly alters appetite-related hormone concentrations independent of exercise time-of-day.

Despite the abovementioned transient alterations in appetite-related hormones and metabolites, the current study did not observe significant differences in energy intake between the HIIE or MICE trials. Food records did indicate lower total energy intake for the remainder of the exercise day following HIIE compared to MICE; however, this difference was not statistically significant. Sim et al. (2014) observed significant reductions in energy intake for up to 24 hours post-exercise following a high-intensity protocol compared to a non-exercise control trial and MICE trial. Similarly, Thivel et al. (2012) observed suppressed energy intake in obese adolescents following vigorous exercise compared to moderate-intensity exercise for up to 24 hours post-exercise. However, it is important to note that energy intake at the post-exercise meal was assessed under controlled laboratory conditions in these studies by Sim et al. (2014) and Thivel et al. (2012); whereas, the present study utilised self-reported food diaries which may make the detection of differences in food intake more difficult and may explain the large variation between participants. While overall energy intake was not significantly altered, there was some evidence of changes in macronutrient intake in the current study, such as the

reduction of sugar intake in the two days following exercise compared to the day of exercise for both MICE and HIIE. However, these observed changes may simply reflect day-to-day variation which is influenced by many other factors including food availability, food diversity, and social engagements (Champagne et al. 2013), rather than in response to the acute exercise bouts.

The complex neuroendocrine pathways which link sleep and appetite continuously communicate to maintain energy homeostasis (Wynne et al. 2005). As such, when sleep is altered there are subsequent changes to the circadian rhythm of appetite-related hormone release which influences dietary and eating behaviour changes (Spiegel et al. 2004; Copinschi et al. 2014). Given this knowledge, it was important to examine sleep and appetite simultaneously following acute exercise which has been shown to influence both sleep patterns (Robey et al. 2013) and appetite (Sim et al. 2014). Although not all sleep measures were significantly different between trials, trends indicate that the HIIE trial had a greater positive impact on sleep quality measures compared to MICE, including a dominance of NREM sleep in the first half of sleep, and a greater proportion of REM sleep in the second half of sleep (Rama et al. 2005; Sharma and Kavuru 2010; Copinschi et al. 2014). However, it is expected that from approximately 35 years of age the duration of stage N3 sleep reduces dramatically followed by a progressive decline in REM sleep (Copinschi et al. 2014). Given the age of the participants, it is plausible that age-related factors were influencing baseline sleep. As such, the increase in stage N3 sleep and redistribution of REM sleep to the latter half of the night after HIIE suggests that vigorous exercise may slow the rate of age-related sleep changes. Even though we did not observe a difference in perceived appetite or energy intake, it is possible that the changes in ghrelin and leptin concentrations over time were positively influenced by sleep regardless of exercise intensity. Although appetite-related hormones were not measured at baseline, the morning after exercise results for ghrelin support the notion that there is a sleep-associated inhibition of the orexigenic signal (Copinschi et al. 2014). Nonetheless, continued research is required to further assess

the link between sleep and appetite following exercise due to the complexity of the neuroendocrine pathways.

The strength and novel aspect of the current study is the examination of the interaction between three key areas that is sleep, appetite and exercise. Nonetheless, there are several limitations which need to be addressed and may assist the direction of future research. There were limited time points for the analysis of acylated ghrelin, leptin, PYY_{total} and glucose; however, the three designated time points are in alignment with capturing acute and prolonged responses across all hormones. Additionally, the accuracy of self-reporting physical activity may have been limited due to the risk of participants under- or over-reporting exercise duration, type and intensity during the study. As such, future research may assess physical activity with a combination of accelerometer activity data and self-reported data.

In summary, the key findings of this study are that HIIE induces positive changes in sleep and appetite which would appear favourable for improved sleep quality and reduced energy intake. We surmise that the increase in stage N3 sleep and reduced number of arousals during REM sleep were associated with the high energy demands associated with high-intensity exercise and subsequent need for body restoration. In conjunction, the minimal reduction in energy intake following HIIE may have been a result of the transient reduction of ghrelin and increased glucose concentration; however, these changes were not significant and did not continue during the 48 hours post-exercise. Taken together, the acute sleep and appetite responses to high-intensity exercise appear small and transient. Nonetheless, compounding these effects may better assist sleep quality, regulation of metabolic hormones, weight management and eating behaviour over an extended time. As such, future studies may profit from investigating these sleep, appetite and exercise associations further under a chronic setting.

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PL and MS developed the study concepts. PL collected data, performed the data analysis and prepared the manuscript. KM scored all of the sleep studies. All authors provided important insight on data interpretation and contributed to the manuscript.

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Table 1. Mean \pm SD whole night and initial 180 min sleep data for baseline (BASE), after moderate-intensity continuous exercise (MICE) and high-intensity interval exercise (HIIE) trials ($n = 11$).

	BASE		MICE		HIIE	
	Whole Night	Initial 180 min	Whole Night	Initial 180 min	Whole Night	Initial 180 min
Time in bed (min)	484.6 \pm 39.8		473.2 \pm 31.2		461.7 \pm 34.9	
Total sleep time (min)	405.7 \pm 54.4	163.7 \pm 14.3	405.1 \pm 38.2	168.5 \pm 8.0	407.1 \pm 40.7	167.5 \pm 11.3
Sleep efficiency (%)	83.7 \pm 6.9	90.8 \pm 7.9	85.7 \pm 6.9	93.4 \pm 4.5	88.2 \pm 5.6	92.8 \pm 6.3
Sleep onset latency (min)	23.1 \pm 16.2		27.4 \pm 28.2		18.4 \pm 15.2	
Rapid eye movement latency (min)	84.2 \pm 21.0	82.9 \pm 21.9	107.8 \pm 70.6	107.8 \pm 70.6	109.5 \pm 34.6	109.5 \pm 34.5
Wake after sleep onset (min)	55.7 \pm 32.6	16.7 \pm 14.2	40.8 \pm 21.9 ^a	11.9 \pm 8.0	36.2 \pm 21.6	13.0 \pm 11.3
Stage N1 sleep (%)	8.4 \pm 4.0	6.9 \pm 3.4	7.3 \pm 1.9	5.2 \pm 2.3	6.3 \pm 2.3	5.6 \pm 3.2
Stage N2 sleep (%)	53.9 \pm 5.9	52.8 \pm 7.9	54.4 \pm 8.9	53.7 \pm 10.7	55.5 \pm 7.7	55.3 \pm 7.9
Stage N3 sleep (%)	18.0 \pm 7.2	27.7 \pm 10.6	20.7 \pm 6.9	31.2 \pm 5.9	21.0 \pm 7.3 ^a	31.9 \pm 8.2
Non-rapid eye movement (%)	80.3 \pm 3.9	87.3 \pm 5.4	82.4 \pm 3.9	90.2 \pm 6.0	82.8 \pm 5.2	92.4 \pm 4.2 ^a
Rapid eye movement (%)	19.7 \pm 3.9	12.7 \pm 5.4	17.6 \pm 3.9	9.8 \pm 6.0	17.2 \pm 5.2	7.6 \pm 4.3 ^a
Non-rapid eye movement arousals (#)	53.3 \pm 22.4	23.2 \pm 9.8	58.5 \pm 21.7	28.5 \pm 10.6	61.3 \pm 28.5	31.2 \pm 16.2
Rapid eye movement arousals (#)	10.8 \pm 6.8	2.3 \pm 1.5	11.2 \pm 7.7	3.1 \pm 2.8	7.4 \pm 4.9 ^a	1.3 \pm 1.2
Total arousals (#)	83.0 \pm 30.8	31.5 \pm 12.0	89.5 \pm 24.9	38.1 \pm 14.4	82.4 \pm 32.9	37.5 \pm 17.5

^a Indicates significant difference compared to BASE ($p \leq 0.05$).

Table 2. Mean \pm SD total energy and macronutrient intake for baseline (BASE), day of moderate-intensity continuous exercise (MICE-0), one day after MICE (MICE+1), two days after MICE (MICE+2), day of high-intensity interval exercise (HIIE-0), one day after HIIE (HIIE+1), two days after HIIE (HIIE+2) ($n = 11$).

	BASE	MICE-0	MICE+1	MICE+2	HIIE-0	HIIE+1	HIIE+2
Total Energy Intake (kJ)	8501 \pm 3248	9471 \pm 4039	7229 \pm 4468	8454 \pm 5367	8395 \pm 2217	7215 \pm 3266	7813 \pm 3544
Carbohydrates (g)	204 \pm 84	265 \pm 129 ^a	193 \pm 119 ^a	140 \pm 62	220 \pm 93	179 \pm 72	190 \pm 83
(%)	41 \pm 7	47 \pm 13	45 \pm 8 ^a	32 \pm 15 ^b	43 \pm 11	43 \pm 6	42 \pm 9 ^b
Fats (g)	78 \pm 37	78 \pm 37	63 \pm 45	74 \pm 32	83 \pm 29	66 \pm 43	61 \pm 34
(%)	34 \pm 6	31 \pm 9 ^b	33 \pm 9	36 \pm 11	37 \pm 7 ^b	32 \pm 10	30 \pm 8
Protein (g)	97 \pm 37	115 \pm 45 ^a	67 \pm 38 ^a	82 \pm 43	93 \pm 39	79 \pm 38	80 \pm 45
(%)	19 \pm 3	21 \pm 7	16 \pm 3	18 \pm 7	18 \pm 5	18 \pm 4	16 \pm 4
Sodium (mg)	2078 \pm 349	3357 \pm 1789 ^a	2327 \pm 1544	1620 \pm 858	2658 \pm 752 ^a	1985 \pm 1372	1680 \pm 670 ^a
Sugar (g)	81 \pm 41	114 \pm 59	82 \pm 66*	63 \pm 21*	85 \pm 49	66 \pm 46*	73 \pm 49*
Caffeine (mg)	146 \pm 76	120 \pm 62	102 \pm 92	151 \pm 101	100 \pm 91	100 \pm 78	125 \pm 78

^a Indicates significant difference compared to BASE ($p \leq 0.05$).

^b Indicates significant trial \times time interaction between MICE and HIIE ($p = 0.03$).

* Indicates a main effect of time for MICE and HIIE ($p \leq 0.05$).

Figure 1. Overview of the experimental procedures. MICE: moderate-intensity continuous exercise; HIIE: high-intensity interval exercise; PSG: polysomnography; $\dot{V}O_{2peak}$: peak oxygen consumption.

Figure 2. Mean \pm SD A. heart rate; and B. rating of perceived exertion during 30 min of moderate-intensity continuous exercise (MICE) and high-intensity interval exercise (HIIE) ($n = 11$).

^b Indicates significant trial \times time interaction between MICE and HIIE ($p \leq 0.04$).

Figure 3. Mean \pm SD A. perceived hunger; and B. perceived fullness on the day of exercise (day 0) at pre-exercise and 30 min post-exercise, and the morning after exercise (day 1 morning) for moderate-intensity continuous exercise (MICE) and high-intensity interval exercise (HIIE) ($n = 11$).

* Indicates a main effect of time for both trials ($p \leq 0.05$).

Figure 4. Mean \pm SD A. acylated ghrelin; B. leptin; C. peptide tyrosine tyrosine (PYY_{total}); and D. glucose on the day of exercise (day 0) at pre-exercise and 30 min post-exercise, and the morning after exercise (day 1 morning) for moderate-intensity continuous exercise (MICE) and high-intensity interval exercise (HIIE) trials ($n = 11$).

^b Indicates significant trial \times time interaction between MICE and HIIE ($p \leq 0.03$).

* Indicates a main effect of time for both trials ($p < 0.05$).