High-intensity interval exercise induces greater acute changes in sleep, appetite-related hormones, and free-living energy intake than does moderate-intensity continuous exercise

Penelope Larsen, Frank Marino, Kerri Melehan, Kym J. Guelfi, Rob Duffield, and Melissa Skein

Abstract: The aim of this study was to compare the effect of high-intensity interval exercise (HIIE) and moderate-intensity continuous exercise (MICE) on sleep characteristics, appetite-related hormones, and eating behaviour. Eleven overweight, inactive men completed 2 consecutive nights of sleep assessments to determine baseline (BASE) sleep stages and arousals recorded by polysomnography (PSG). On separate afternoons (1400–1600 h), participants completed a 30-min exercise bout: either (i) MICE (60% peak oxygen consumption) or (ii) HIIE (60 s of work at 100% peak oxygen consumption: 240 s of rest at 50% peak oxygen consumption), in a randomised order. Measures included appetite-related hormones (acylated ghrelin, leptin, and peptide tyrosine tyrosine) and glucose before exercise, 30 min after exercise, and the next morning after exercise; PSG sleep stages; and actigraphy (sleep quantity and quality); in addition, self-reported sleep and food diaries were recorded until 48 h after exercise. There were no between-trial differences for time in bed (p = 0.19) or total sleep time (p = 0.99). After HIIE, stage N3 sleep was greater (21% ± 7%) compared with BASE (18% ± 7%; p = 0.02). In addition, the number of arousals during rapid eye movement sleep were lower after HIIE (7 ± 5) compared with BASE (11 ± 7; p = 0.05). Wake after sleep onset was lower following MICE (41 min) compared with BASE (56 min; p = 0.02). Acylated ghrelin was lower and glucose was higher at 30 min after HIIE when compared with MICE (p ≤ 0.05). There were no significant differences between conditions in terms of total energy intake (p ≥ 0.05). HIIE appears to be 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.

Key words: acute exercise, high-intensity interval exercise, sleep stages, polysomnography, appetite regulation, appetite behaviour.

Résumé : Cette étude a pour objectif de comparer l’effet de l’exercice par intervalle d’intensité élevée (« HIIE ») et d’intensité modérée en continu (« MICE ») sur les caractéristiques du sommeil, les hormones associées à l’appétit et le comportement alimentaire. Onze hommes en surpoids et inactifs se sont soumis durant deux nuits consécutives à une évaluation initiale (« BASE ») pour la détermination des phases du sommeil et des rêves au moyen d’un polysomnographe (« PSG »). Au cours d’après-midi distincts (14 h – 16 h), les sujets participent à une séance de 30 min d’exercice : (i) MICE (60 % consommation du pointe d’oxygène) ou (ii) HIIE (60 s d’effort sollicitant 100 % de la consommation du pointe d’oxygène) ou (ii) HIIE (60 s d’effort sollicitant 100 % de la consommation du pointe d’oxygène) ou (ii) HIIE (60 s d’effort sollicitant 100 % de la consommation du pointe d’oxygène) ou (ii) HIIE (60 s d’effort sollicitant 100 % de la consommation du pointe d’oxygène) ou (ii) HIIE (60 s d’effort sollicitant 100 % de la consommation du pointe d’oxygène) ou (ii) HIIE (60 s d’effort sollicitant 100 % de la consommation du pointe d’oxygène) ou (ii) HIIE (60 s d’effort sollicitant 100 % de la consommation du pointe d’oxygène) ou (ii) HIIE (60 s d’effort sollicitant 100 % de la consommation du pointe d’oxygène). Les variables mesurées incluent les suivantes : les hormones associées à l’appétit (ghréline acylée, leptine, peptide tyrosine tyrosine), glucose préexercice, 30 min postexercice et le lendemain matin ; phases du sommeil PSG, actigraphie (quantité et qualité du sommeil), carnet autorapporté de sommeil et d’alimentation jusqu’à 48 h postexercice. Il n’y a pas de différences entre les conditions concernant le temps au lit (p = 0.19) et le temps total de sommeil (p = 0.99). Dans la condition HIIE, le stade N3 du sommeil est plus long (21 ± 7 %) comparativement à BASE (18 ± 7 %; p = 0.02). De plus, le nombre d’éveils au cours du sommeil rapide est plus faible dans la condition HIIE (7 ± 5) comparativement à BASE (11 ± 7; p = 0.05). Les interruptions de sommeil sont inférieures dans la condition MICE (41 min) comparativement à BASE (56 min, p = 0.02). La concentration de ghréline acylée est plus faible et la glycémie plus élevée à 30 min postexercice dans la condition HIIE comparativement à la condition MICE (p ≤ 0.05). Il n’y a pas de différence significative d’apport total en énergie entre les deux conditions (p ≥ 0.05). HIIE semble plus profitable que MICE pour améliorer la qualité du sommeil et pour induire des modifications transitoires favorables des hormones associées à l’appétit chez des hommes en surpoids et inactifs. Néanmoins, l’intensité de l’exercice ne modifie pas l’apport énergétique. [Traduit par la Rédaction]

Mots-clés : exercice ponctuel, exercice par intervalle d’intensité élevée, stades du sommeil, polysomnographie, régulation de l’appétit, comportement alimentaire.

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 because of 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, and 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 on this topic has recruited young adults with minimal sleep complaints (Youngstedt 2005). Therefore, the effects of exercise on sleep patterns in middle-aged to older adults remain 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 in 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 a reduction in sleep-onset latency (SOL) and wake after sleep onset (WASO), primarily in adolescents and young adults (Kredlow et al. 2015), whereas the effect in middle-aged populations is largely unexplored. In terms of appetite, high-intensity exercise, unlike moderate-intensity exercise, has been associated with the down-regulation of orexigenic signals (e.g., acylated ghrelin) and the up-regulation of anorexigenic signals (e.g., leptin, peptide tyrosine tyrosine, and glucose), which may lead to reduced perceived hunger and energy intake in overweight, inactive men for up to 24 h (Sim et al. 2014). However, the effects 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 h 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).

Previous research 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 now to investigate the effect of exercise on sleep and appetite simultaneously by considering the potential interaction between these 3 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 with a resting baseline (BASE), but that 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 with MICE.

Materials and methods

Participants

Eleven overweight, inactive men (mean ± SD: age, 49 ± 5 years; body mass index (BMI), 28 ± 3 kg·m−2; waist-to-hip ratio (WHR), 0.92 ± 0.05; peak oxygen consumption (VO2peak), 34 ± 8 mL·kg−1·min−1) completed this study. Initially, 13 men volunteered to participate in the study; however, 1 participant was excluded because of signs of sleep apnoea, and 1 participant withdrew because of illness unrelated to the study. Inclusion/exclusion criteria included being a nonsmoker, participating in <150 min of moderate-intensity exercise per week, and having no previous or current diagnosis of sleep or metabolic disorders and no medical conditions or medications that affect sleep quality or quantity. Sleep was assessed initially by the STOP-BANG questionnaire (Chung et al. 2008) and the Epworth Sleepiness Scale (Johns 1991). Risk of sleep apnoea was further assessed by 2 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 that no underlying conditions would be exacerbated by vigorous exercise. The study was approved by the Charles Sturt University Human Ethics Committee, and written informed consent was obtained from all participants prior to data collection.

Experimental overview

Participants came to the laboratory for an initial familiarisation session and BASE assessments of anthropometry and VO2peak. Habitual sleep and eating patterns were also documented for 7 days and nights (Fig. 1). During this time, 2 consecutive nights of PSG sleep assessments were conducted to exclude sleep apnoea and to determine BASE sleep staging and arousals. Following BASE assessment, participants completed 2 experimental trials (4 days in duration each) in a randomised order. The experimental trials included either 30 min of MICE (60% VO2peak) or 30 min of HIIE (60 s at 100% VO2peak: 240 s at 50% VO2peak). 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 postexercise sleep quality and quantity, changes in plasma concentrations of appetite-related hormones, ratings of perceived appetite, and postexercise free-living energy intake.

Familiarisation and BASE testing

The familiarisation session involved assessment of height and body mass to calculate BMI and waist and hip girths to calculate WHR. In addition, VO2peak 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. VO2peak test commenced at 50 W for the first 2 min and increased by 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, Tex., USA).

BASE at-home testing was completed for a total of 7 days and nights, after which participants were fitted with a wrist actigraph (Actiware 2, Philips Respironics, Andover, Mass., USA) and documented sleep and food intake in a diary provided for the duration of BASE testing. During this time, participants were instructed to maintain their usual bedtime, 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 2 PSG sleep studies using a level II, take-home PSG device were conducted...
Fig. 1. Overview of the experimental procedures. HIIE, high-intensity interval exercise; MICE, moderate-intensity continuous exercise; PSG, polysomnography; VO2peak, peak oxygen consumption.

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<td>2 consecutive nights PSG</td>
<td>Anthropometry</td>
<td>VO2peak test</td>
<td>7 day actigraphy, sleep and food record</td>
<td>MICE and HIIE: 4-day actigraphy, sleep and food record</td>
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<td>Perceived appetite</td>
<td>Blood sampling</td>
<td>30 min exercise protocol</td>
<td>Perceived appetite</td>
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<td>Blood sampling</td>
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Sleep studies during experimental trials were assessed for sleep staging and arousals only. 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 (sleep duration/wake time × 100), SOL (time from lights out to the first epoch of sleep), REM-onset latency, WASO, percent of time spent in each sleep stage (N1: stage 1; N2: stage 2; N3: stage 3; non-REM sleep (NREM); and REM), and number of arousals (NREM, REM, and total arousals).

**Actigraphy**

Actigraphy was recorded in 1-min epochs (Esliger and Tremblay 2006) and was analysed using Actiware software, version 5.70 (Philips Respironics). Variables obtained included bedtime, wake-up time, time in bed (period between bedtime and wake time), TST (time asleep during time in bed), SOL (period between bedtime and sleep onset), sleep efficiency (percent of time in bed spent sleeping), WASO, and number of awakenings (Knutson et al. 2007).

**Appetite perception and hormones**

Perceived hunger and fullness were assessed using a VAS composed of straight lines (100 mm) accompanied by a question anchored with words representing opposing extreme states of hunger or fullness. VASes were chosen because of their role in hunger and satiety signals (Broom et al. 2009; Balaguera-Cortes et al. 2011), and association with sleep changes (Spiegel et al. 2011). For acylated ghrelin, leptin, and total peptide tyrosine tyrosine (PYYtotal), according to manufacturer’s instructions, using a commercially available assay kit (Milliplex, MilliporeSigma, Mass., USA). These hormones were chosen because of 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 PYYtotal, the intra- and interassay coefficients of variation were <10% and <15%, respectively.

**Sleep and energy intake records**

A diary for reporting sleep and food and drink intake was provided to each participant. Sleep records were used to confirm bedtime and wake-up time for actigraphy data. For food records,

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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 intakes were calculated using commercially available software (Foodworks, Xyris Software, Kenmore Hills, QLD, Australia). In addition, absolute (grams) and relative data (percentages) 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 performed, and it indicated no significant difference between trial 1 and trial 2 for PST TST or WASO (p ≤ 0.14); however, the difference in sleep efficiency was significant (p = 0.006). A repeated-measures (trial x time interaction) ANOVA with Tukey’s least significant difference post hoc test was used to compare physiological and perceptual measures, perceived appetite, appetite-related hormones and glucose, with energy intake, PSG, and actigraphy variables between trials. PSG data were further separated to analyse the initial 180 min after sleep onset, because the first 1 to 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, 2 analyses were conducted to compare the difference between MICE and HIIE immediately after exercise (acute: energy intake for the remainder of the day following exercise) and to compare differences among BASE, MICE, and HIIE (over the total 48-h period of monitoring). Analyses were performed using Statistical Package for Social Sciences (SPSS, version 20.0, IBM Corp., Armonk, N.Y., USA). Data are reported as means ± SD, and statistical significance was accepted at p ≤ 0.05.

Results
Exercise characteristics
The mean HRs for MICE and HIIE were 126 ± 10 bpm and 132 ± 10 bpm, respectively. HR responses during HIIE were higher than those during MICE at 1–6 min, 10–11 min, 15–16 min, 20–21 min, 25–26 min, and 30 min (p ≤ 0.04; Fig. 2A). Mean RPE was significantly lower for MICE (3 ± 1) than for HIIE (7 ± 2; p = 0.001; Fig. 2B). Higher RPEs were reported following all HIIE sprint intervals compared with those reported for the corresponding times for MICE (p ≤ 0.001; Fig. 2B).

PSG and actigraphy
Whole-night and initial 180-min PSG data are presented in Table 1. There were no significant differences in terms of time in bed, TST, sleep efficiency, SOL, or N1, N2, NREM, and REM sleep among BASE, MICE, and HIIE (p > 0.05). However, there was a significant decrease in WASO following MICE compared with BASE (p = 0.02). In addition, the proportion of N3 sleep was higher (p = 0.02), whereas the number of arousals was lower during REM sleep (p = 0.05) after HIIE when compared with BASE. There were no differences among BASE, MICE, or HIIE in terms of NREM arousals (p = 0.59) or total arousals (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 with BASE (p = 0.02). The number of arousals during REM sleep was also decreased after HIIE compared with BASE (p = 0.03). There were no differences in terms of NREM arousals (p = 0.21) or total arousals (p = 0.36) between BASE and both exercise trials. There were no between-trial differences in all other sleep parameters assessed (p > 0.05).

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

Perceived appetite and appetite-related hormones
There was no trial x time interaction for perceived hunger (p = 0.29; Fig. 3A) or fullness (p = 0.73; Fig. 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 ≤ 0.02).

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

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

Energy and macronutrient intake for BASE, and 2 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 than for MICE (p = 0.03). Absolute carbohydrate intake after MICE on the day of exercise was higher compared with BASE (p = 0.04), but lower at 2 days after exercise compared with BASE (p = 0.05). Moreover, relative carbohydrate intake for the day after MICE was higher compared with BASE (p = 0.03), whereas for 2 days after exercise, intake was higher after MICE than after HIIE (p = 0.03). Absolute protein intake was higher on the day of MICE compared with BASE (p = 0.04), although 1 day following MICE, intake was lower compared with BASE (p = 0.04). On the day of MICE and HIIE, and 2 days after HIIE, sodium intake was higher compared with BASE (p ≤ 0.03). There were no further trial x time interactions for energy intake (p = 0.61), macronutrient intake (p ≥ 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 2 days following exercise (p = 0.02).

Discussion
This study investigated the effect of HIIE compared with traditional MICE 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 2 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 with BASE, whereas only lower WASO compared with BASE was observed following MICE. In addition, circulating acylated ghrelin was lower and glucose concentrations were higher transiently after HIIE compared with

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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 HIIE may have a greater positive influence on sleep and appetite-related hormones than does MICE. However, the alterations in acute sleep and appetite regulation may not be reflected in changes in perceptual measures (hunger and satiety) or behaviours (sleep hygiene and dietary choices).

The observed changes in sleep following exercise are consistent with the results of previous research that has examined various populations. Specifically, greater stage N3 sleep and improved sleep efficiency have been observed, in conjunction with decreased SOL, WASO, 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 HIIE, a greater proportion of REM sleep was experienced later in the night, compared with BASE sleep. Robey and colleagues (2013) observed similar changes in REM sleep following vigorous evening exercise in highly trained cyclists. Although the cause of the change in distribution warrants further investigation, Netzer and colleagues (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. The authors reported that an extension of REM-onset latency and a 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

Fig. 2. (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). Data are presented as means ± SD. b, Significant trial × time interaction between MICE and HIIE (p ≤ 0.04).
### Table 1. Whole-night and initial 180-min sleep data for baseline (BASE) and after moderate-intensity continuous exercise (MICE) and high-intensity interval exercise (HIIE) trials (n = 11).

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<th></th>
<th>BASE</th>
<th>MICE</th>
<th>HIIE</th>
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<tr>
<td><strong>BASE</strong></td>
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<tr>
<td><strong>Whole night</strong></td>
<td>484.6±39.8</td>
<td>473.2±31.2</td>
<td>461.7±34.9</td>
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<td><strong>Initial 180 min</strong></td>
<td>405.7±54.4</td>
<td>163.7±14.3</td>
<td>407.1±40.7</td>
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<tr>
<td><strong>Whole night</strong></td>
<td>83.7±6.9</td>
<td>90.8±7.9</td>
<td>85.7±6.9</td>
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<tr>
<td><strong>Initial 180 min</strong></td>
<td>85.7±6.9</td>
<td>93.4±4.5</td>
<td>88.2±5.6</td>
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<tr>
<td><strong>Rapid eye movement latency (min)</strong></td>
<td>23.1±16.2</td>
<td>27.4±28.2</td>
<td>18.4±15.2</td>
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<td><strong>Wake after sleep onset (min)</strong></td>
<td>84.2±21.0</td>
<td>107.8±70.6</td>
<td>109.5±34.6</td>
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<td><strong>Sleep efficiency (%)</strong></td>
<td>8.4±4.0</td>
<td>6.9±3.4</td>
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<td><strong>Stage N1 sleep (%)</strong></td>
<td>53.9±5.9</td>
<td>52.8±7.9</td>
<td>54.4±8.9</td>
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<td><strong>Stage N2 sleep (%)</strong></td>
<td>8.0±7.2</td>
<td>27.7±10.6</td>
<td>20.7±6.9</td>
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<td><strong>Non-rapid eye movement (%)</strong></td>
<td>80.3±3.9</td>
<td>87.3±5.4</td>
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<td><strong>Rapid eye movement (%)</strong></td>
<td>19.7±3.9</td>
<td>12.7±5.4</td>
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<td><strong>Non-rapid eye movement arousals (No.)</strong></td>
<td>53.3±22.4</td>
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<td><strong>Rapid eye movement arousals (No.)</strong></td>
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<td>2.3±1.5</td>
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<td><strong>Total arousals (No.)</strong></td>
<td>83.0±30.8</td>
<td>31.5±12.0</td>
<td>89.5±24.9</td>
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</table>

**Note:** Data are presented as means ± SD. a, Significant difference compared with BASE (p ≤ 0.05).

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**Fig. 3.** Mean ± SD (A) Perceived hunger and (B) perceived fullness on the day of exercise (day 0) before exercise and 30 min after exercise, and the morning after exercise (day 1 morning) for moderate-intensity continuous exercise (MICE) and high-intensity interval exercise (HIIE) (n = 11). Data are presented as means ± SD. *, Main effect of time for both trials (p ≤ 0.05).
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 with 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 HR, a release of metabolic hormones (e.g., growth hormone) and lactate, and depletion of adenosine triphosphate, creatine phosphate, and glycogen stores (Weinstein et al. 1998; Tomlin and Wenger 2001; Trapp et al. 2007; Boucher 2011). Consequently, recovery may be extended, and oxygen consump-

Fig. 4. Mean ± SD (A) Acylated ghrelin, (B) leptin, (C) total peptide tyrosine tyrosine (PYYtotal), and (D) glucose on the day of exercise (day 0) before exercise and 30 min after 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, Significant trial × time interaction between MICE and HIIE (p ≤ 0.03); *, main effect of time for both trials (p < 0.05).

Table 2. Total energy and macronutrient intake for baseline (BASE), day of moderate-intensity continuous exercise (MICE-0), 1 day after MICE (MICE+1), 2 days after MICE (MICE+2), day of high-intensity interval exercise (HIIE−0), 1 day after HIIE (HIIE+1), and 2 days after HIIE (HIIE+2) (n = 11).

<table>
<thead>
<tr>
<th></th>
<th>BASE</th>
<th>MICE-0</th>
<th>MICE+1</th>
<th>MICE+2</th>
<th>HIIE-0</th>
<th>HIIE+1</th>
<th>HIIE+2</th>
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<tbody>
<tr>
<td>Total energy intake (kJ)</td>
<td>8501±3248</td>
<td>9471±4039</td>
<td>7229±4468</td>
<td>8454±5367</td>
<td>8395±2217</td>
<td>7215±3266</td>
<td>7813±3544</td>
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<tr>
<td>Carbohydrates</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>g</td>
<td>204±84</td>
<td>265±129</td>
<td>193±119</td>
<td>140±62</td>
<td>220±93</td>
<td>179±72</td>
<td>190±83</td>
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<tr>
<td>%</td>
<td>41±7</td>
<td>47±13</td>
<td>45±8</td>
<td>32±15b</td>
<td>43±11</td>
<td>43±6</td>
<td>42±9b</td>
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<td>Fats</td>
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<td>g</td>
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<td>66±43</td>
<td>61±34</td>
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<tr>
<td>%</td>
<td>34±6</td>
<td>31±9b</td>
<td>33±9</td>
<td>36±11</td>
<td>37±27b</td>
<td>32±10</td>
<td>30±8</td>
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<tr>
<td>Protein</td>
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<tr>
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<td>97±37</td>
<td>115±45a</td>
<td>67±38a</td>
<td>82±43</td>
<td>93±39</td>
<td>79±38</td>
<td>80±45</td>
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<td>%</td>
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<td>16±3</td>
<td>18±7</td>
<td>18±5</td>
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<td>16±4</td>
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<tr>
<td>Sodium (mg)</td>
<td>2078±349</td>
<td>3357±1789</td>
<td>2327±1544</td>
<td>1620±858</td>
<td>2658±752a</td>
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<td>Sugar (g)</td>
<td>81±41</td>
<td>114±59</td>
<td>82±66*</td>
<td>63±21*</td>
<td>85±49</td>
<td>66±46*</td>
<td>73±49*</td>
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<tr>
<td>Caffeine (mg)</td>
<td>146±76</td>
<td>120±62</td>
<td>102±92</td>
<td>151±101</td>
<td>100±78</td>
<td>100±78</td>
<td>125±78</td>
</tr>
</tbody>
</table>

Note: Data are presented as means ± SD. a, Significant difference compared with BASE (p ≤ 0.05); b, significant trial × time interaction between MICE and HIIE (p = 0.03).

*Main effect of time for MICE and HIIE (p ≤ 0.05).
tion remains elevated after exercise to accelerate the return of metabolic processes to a resting state (La Forgia et al. 2006; Boutcher 2011). Given the vigorous nature of our HIIE protocol, it is plausible that participants experienced greater glycogen depletion and metabolism accumulation, which required a longer recovery compared with the MICE trial. Simultaneously, untrained individuals have been shown to experience slower rates of recovery compared with 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 that the nature of stage N3 sleep is 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 more favourable for reducing energy intake when compared with MICE. More specifically, decreased ghrelin and increased glucose were observed 30 min after exercise in the HIIE trial. The lower ghrelin following HIIE occurred despite significantly higher circulating ghrelin prior to exercise, suggesting that the magnitude of the 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 and energy intake was controlled prior to both trials; therefore, the difference may simply reflect the considerable intra- and inter-individual variation of acylated ghrelin (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 with MICE may also contribute to a potential down-regulation of appetite. The present results are consistent with those of Sim and colleagues (2014), who observed a significant decline in ghrelin and an increase in glucose acutely following high-intensity exercise compared with a bout of traditional moderate-intensity exercise. These responses were observed despite a longer fasting time (overnight 10-h fast) compared with the current study and exercise being performed in the morning instead of the afternoon. As such, these 2 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 and MICE trials. Food records did indicate lower total energy intake for the remainder of the exercise day following HIIE compared with MICE; however, this difference was not statistically significant. Sim and colleagues (2014) observed significant reductions in energy intake for up to 24 h after exercise following a high-intensity protocol compared with a nonexercise control trial and MICE trial. Similarly, Thivel and colleagues (2012) observed suppressed energy intake in obese adolescents following vigorous exercise compared with moderate-intensity exercise for up to 24 h after exercise. However, it is important to note that energy intake at the postexercise meal was assessed under controlled laboratory conditions in these studies by Sim and colleagues (2014) and Thivel and colleagues (2012), whereas the current study utilised self-reported food diaries, which may make the detection of differences in food intake more difficult and may explain the large variation among participants. Although overall energy intake was not altered significantly, there was some evidence of changes in macronutrient intake in the current study, such as the reduction of sugar intake in the 2 days following exercise compared with 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 a response to the acute exercise bouts.

The complex neuroendocrine pathways that link sleep and appetite communicate continuously to maintain energy homeostasis (Wynne et al. 2005). As such, when sleep is altered there are subsequential 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 sleep patterns and appetite (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 with the MICE trial, 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, the duration of stage N3 sleep is expected to decrease dramatically after approximately 35 years of age, 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 BASE sleep. As such, the increase in stage N3 sleep and the 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 BASE, 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, because of the complexity of the neuroendocrine pathways, continued research is required to further assess the link between sleep and appetite following exercise.

The strength and novel aspect of the current study are the examination of the interaction among 3 key areas (i.e., sleep, appetite, and exercise). Nonetheless, there are several limitations that must be addressed and may assist the direction of future research. There were limited time points for the analysis of acylated ghrelin, leptin, PYYtotal, and glucose; however, the 3 designated time points are in alignment with capturing acute and prolonged responses across all hormones. In addition, the accuracy of self-reporting physical activity may have been limited because of 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 the reduced number of arousals during REM sleep were associated with the high energy demands associated with high-intensity exercise and the 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 the increased glucose concentration; however, these changes were not significant and did not continue during the 48 h after 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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Conflict of interest statement
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P.L. and M.S. developed the study concepts. P.L. collected data, performed the data analysis, and prepared the manuscript. K.M. scored all of the sleep studies. All authors provided important insight into data interpretation and contributed to the manuscript.

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