**Title**: Effect of exercise on sleep and bi-directional associations with accelerometer-assessed physical activity in men with obesity

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**Abstract**

This study examined the effect of exercise training on sleep duration and quality and bidirectional day-to-day relationships between physical activity (PA) and sleep. Fourteen inactive men with obesity (49.2±7.9 years, BMI 34.9±2.8 kg/m²) completed a baseline visit, eight-week aerobic exercise intervention, and one-month post-intervention follow-up. PA and sleep were assessed continuously throughout the study duration using wrist-worn accelerometry. Generalised estimating equations (GEE) were used to examine associations between PA and sleep. Sleep duration increased from 5.2h at baseline to 6.6h during the intervention period and 6.5h at one-month post-intervention follow-up (p<0.001). Bi-directional associations showed that higher overall activity volume and moderate-to-vigorous physical activity (MVPA) were associated with earlier sleep onset time (p<0.05). Later timing of sleep onset was associated with lower overall volume of activity, most active continuous 30 minutes (M30CONT), and MVPA (p<0.05). Higher overall activity volume, M30CONT, and MVPA predicted more wake after sleep onset (WASO) (p<0.001), whereas greater WASO was associated with higher overall volume of activity, M30CONT, and MVPA (p<0.001). An aerobic exercise intervention increased usual sleep duration. Day-to-day, more PA predicted earlier sleep onset, but worse sleep quality and vice versa.

Novelty:

* Greater levels of physical activity in the day were associated with an earlier sleep onset time that night, whereas a later timing of sleep onset was associated with lower physical activity the next day in men with obesity
* Higher physical activity levels were associated with worse sleep quality, and vice versa

**Keywords:** moderate-to-vigorous physical activity; exercise; device-based; sleep duration; sleep quality; raw acceleration

# Introduction

Achieving adequate levels of physical activity (PA) and sleep is key for maintaining optimal health. The consequences of low PA levels and poor sleep include increased risk of cardiovascular disease (Lee et al. 2012), metabolic disorder (Reutrakul and Van Cauter 2018), obesity (Reutrakul and Van Cauter 2018), depression (Mammen and Faulkner 2013), and premature mortality (Arem et al. 2015). The wealth of evidence examining the link between PA and sleep suggests that PA can promote sleep (Kredlow et al. 2015). Cross-sectional research has consistently demonstrated an association between higher PA and better sleep duration (McClain et al. 2014) and quality (Loprinzi and Cardinal. 2011), and systematic reviews of exercise interventions on sleep have demonstrated improved sleep duration and quality from engaging in acute and regular exercise (Dolezal et al. 2017). There is also evidence that induced short sleep (<6h) can lead to a significant decrease of PA levels and lower intensity of PA the following day (Schmid et al. 2009; Bromley et al. 2012), and that PA and sleep are linked in a bidirectional manner (Chennaoui et al. 2015). Exercise interventions in overweight and obese adult populations have previously been found to be beneficial for sleep, demonstrating significant improvements in subjective sleep quality (Kline et al. 2012). When measured using device-based methods, exercise training resulted in increased sleep duration (Kjeldsen et al. 2013), reduced sleep onset latency and frequency of difficulty initiating sleep, shorter wake after sleep onset, and higher sleep efficiency (Tan et al. 2016) compared to baseline.

More recently, there has been growing interest in how daily fluctuations in PA and sleep are inter-related in free-living environments, however, studies that investigated day-to-day bidirectional associations between PA and sleep have produced equivocal findings. While some studies investigating bidirectional day-to-day associations between device-based PA and sleep report a bidirectional association between PA and sleep duration (Kishida and Elavsky 2016; Gabriel et al. 2017), some report a positive (Best et al. 2018), or negative (Lambiase et al. 2013) association of PA and subsequent sleep duration or no association at all (Mitchell et al. 2016). Further, there appears to be no evidence of bidirectional associations between PA and sleep quality (e.g. sleep efficiency, wake after sleep onset) (Mitchell et al. 2016; Kishida and Elavsky 2016; Lambiase et al. 2013; McGlinchey et al. 2014) with only a few studies demonstrating that improved sleep quality leads to higher PA the next day (McGlinchey et al. 2014; Lambiase et al. 2013).

Existing studies on the effects of regular exercise on sleep indices in overweight and obese individuals have mainly used aggregate estimates of sleep before and after an exercise intervention by averaging the measurements. This precludes examination of any acute effects exercise might have on sleep. Furthermore, studies investigating bidirectional day-to-day associations between PA and sleep have predominantly focused on women, (Gabriel et al. 2017; Lambiase et al. 2013; Mitchell et al. 2016; Kishida and Elavsky 2016; Baron et al. 2013) and although some studies deployed device-based measures of PA and sleep, PA and sleep characteristics were derived from accelerometer data using a limited time span (i.e. 7 days) (Lambiase et al. 2013; Gabriel et al. 2017; Mitchell et al. 2016).

Accelerometers have been extensively used for PA assessment in free-living conditions. Traditionally, hip-worn accelerometers have been most widely used for PA measurement (Ainsworth et al. 2015); however, recently, wrist-worn accelerometers have become increasingly used to assess PA while also providing valid measures of sleep (van Hees et al. 2018). The use of wrist-worn accelerometers allows continuous measurement of PA and sleep over a period of time in free-living environments with a single device with minimal participant burden whilst being more accurate than self-report (Lauderdale et al. 2008, Prince et al. 2008). Additionally, raw accelerometer data provides population-independent estimates of overall volume of PA as well as intensity (Rowlands et al. 2019). Thus, by using accelerometry data generated from an eight-week exercise training study, the first aim of this study was to describe whether exercise training had an impact on sleep duration and quality in men with obesity. Second, by conducting post hoc analyses, this study aimed to investigate the bidirectional day-to-day associations between accelerometer-determined PA and sleep characteristics. Specifically, i) to examine whether daytime PA affects subsequent sleep characteristics, and ii) to examine whether daytime PA is affected by the previous night’s sleep.

# Methods

This study was a secondary data analysis of an exercise intervention where the primary objective was to determine the effect of exercise on the gut microbiota of men with obesity (the TEAM GB study: The Exercise and Gut Bacteria study) recruited between September 2016 and December 2017 (ISRCTN32774). The study was a one-group pre-post non-randomised clinical trial. All participants participated in an eight-week training protocol of aerobic exercise. After completing a baseline visit, during which participants underwent a full medical history assessment, anthropometric measurements, biochemistry, and a fitness assessment (VO₂max test), participants began an exercise protocol which consisted of three sessions/week of treadmill walking supervised by trained personnel in the exercise laboratory. Participants could choose to exercise either in the morning (10:00-11:30) or evening (17:00-18:30). Exercise sessions were prescribed based on the initial VO₂max test results at a heart rate of 60% of VO2max. The level of exertion was measured by heart rate monitor and the Borg scale of perceived exertion (Borg 1998), with perceived exercise intensity kept between somewhat hard and hard. Duration and load (treadmill incline and speed) were recorded and reviewed each week and progressions made accordingly. Typically, the exercise duration during week one lasted 25 minutes. This was increased by 5 minutes each week with participants’ progressing to 50 minutes duration of continuous exercise by week six, with five-minute warm-up and cool-down. Adverse events related to the exercise programme were recorded. Alterations to exercise mode from treadmill to cycle ergometer were considered as appropriate to minimise the impact of musculoskeletal adverse events. During the intervention period, participants were instructed not to change their dietary and activity habits other than the prescribed exercise that they undertook as a part of the study. After eight weeks of the exercise intervention, participants returned for a repeat of the baseline visit. One month following the end of the intervention, participants attended the final follow-up visit. During the post-intervention follow-up period, participants were instructed to return to their pre-intervention PA levels. The results presented here are from participants who completed the whole study (12 weeks on average).

## Participants

The study was approved by West Midlands Research Ethics Committee, and before any measurements took place, participants signed a written informed consent. The main eligibility criteria included: males of white European ethnicity aged 25-60 years with a BMI of 30-40kg/m2 inclusive, HbA1c less than 6.5%, and a successfully completed VO₂max test. Because gut bacteria may be influenced by many factors (including sex, age, smoking status, etc.), the TEAM GB study aimed to recruit as a homogenous sample of individuals as possible. During the participant identification process from primary care, information about ethnicity was obtained from participants’ medical records or it was self-reported by participants who were recruited through advertisements. The main exclusion criteria included: smokers, dieters, and highly active individuals or individuals meeting current PA guidelines (self-report ≥2.5 hours of moderate or ≥75 minutes vigorous intensity PA per week) (World Health Organization, 2010).

## PA and sleep measurement

Participants’ PA levels and sleep were monitored with the GENEActiv accelerometer (GENEActiv, Activinsights, Kimbolton, UK). The GENEActiv has been previously validated for assessment of PA (Esliger et al. 2011) and sleep (van Hees, et al. 2018). Participants wore the device on their non-dominant wrist 24 hours/day for the entirety of the study phases and completed a daily sleep log. Recording frequency was set at 50 Hz allowing data collection of a maximum of 15 days; consequently, the monitors were changed fortnightly. To determine habitual PA levels, participants wore the monitor for at least seven days before starting the exercise intervention.

## Data processing and outcome measures

All accelerometer files were processed using GGIR package in R version 1.5-21 (<https://cran.r-project.org/web/packages/GGIR/>) (Van Hees et al. 2013; van Hees et al. 2014) and sleep detection algorithm developed by van Hees et al. (van Hees et al. 2018) was applied to derive sleep outcomes. Sleep logs were used to guide the algorithm to identify the sleep window. Signal processing in GGIR includes autocalibration using local gravity as a reference, detection of sustained abnormally high values, detection of non-wear, calculation of the average magnitude of dynamic acceleration (i.e. the vector magnitude of acceleration corrected for gravity (Euclidean Norm minus 1 *g*) in milli-gravitational units (m*g*) averaged over 5-second epochs (van Hees et al. 2014). Valid days were defined as days with more than 16 hours of valid wear time (van Hees et al. 2013). Accelerometer files were excluded if the post-calibration error was greater than 0.01 *g* (10 m*g*) (van Hees et al. 2014), or wear data were not present for each 15 minutes period of the 24-h cycle. Detection of non-wear has been described in detail previously (Van Hees et al. 2013). Briefly, non-wear is estimated based on the standard deviation and value range of each axis, calculated for 60-minute windows with a 15-minutes sliding window. The window is classified as non-wear if, for at least two out of the three axes the standard deviation is less than three m*g* or the value range is less than 50 m*g*. The default non-wear setting was used, i.e. invalid data were imputed by the average at similar time-points on different days of the week (van Hees et. 2013); therefore, the outcome variables were based on the complete 24-h cycle (1440 minutes) for all participants.

The following PA characteristics were obtained: average acceleration in m*g* (ACC; a proxy for overall activity volume), timing and average acceleration in m*g* of most active continuous 30 minutes (M30CONT), minutes of MVPA in 1-minute bouts (MVPA; defined as above an acceleration of 100 m*g* (Hildebrand et al. 2014) lasting at least one minute with no upper boundary) and light activity (defined as acceleration between 40-100 m*g*) (Bakrania et al. 2016). M30CONT is a novel accelerometer metric that captures the average magnitude of acceleration during a person’s most active continuous 30 minutes (Rowlands et al. 2019). This metric does not rely on population- and protocol-dependent cut-points to estimate the intensity of PA.

The following sleep characteristics were obtained: sleep duration (total accumulated sleep within the sleep window, discounting any wake time and daytime sleep); wake after sleep onset (WASO; minutes of wake in the sleep window), the timing of sleep onset, and sleep efficiency (SE; the ratio of total sleep time compared to total sleep window duration). WASO and sleep efficiency were used as measures of sleep quality.

The algorithm used to derive sleep outcomes has been described in detail previously (van Hees. et al. 2018). Briefly, the algorithm is based on angular wrist rotation which is derived from the z-axis (the axis positioned perpendicular when the wrist is in the anatomical position). Sustained inactivity is detected as the absence of change in wrist rotation greater than five degrees for five minutes (the definition used in the present study), or user-defined duration. Sleep is defined as sustained inactivity within a sleep period time window (SPT-window). In this study, sleep logs were applied to guide detection of the SPT-window: the time window starting at sleep onset and ending when waking up after the last sleep episode of the night. The timing of sleep onset equalled the start of the first sustained inactivity bout that overlapped or followed the sleep onset time derived from the sleep log. Sleep duration was the sum of all inactivity bouts within the SPT-window. WASO was calculated as the time difference between the SPT-window and sleep duration within the SPT-window. SE was calculated as (sleep duration/SPT-window)\*100.

All valid days from accelerometers were used in the analyses. The first week before the start of the intervention was defined as baseline, the eight weeks of the exercise intervention as intervention period and one month after the eight-week intervention has finished was defined as post-intervention follow-up.

## Descriptive variables

Participants’ height and weight were assessed to the nearest 0.5cm and 0.5kg, respectively. Body mass index (BMI) was calculated as mass (kg)/height (m)2 and percentage body fat was estimated using bioelectric impedance scales (Tanita TBE 611, Tanita, West Drayton, UK). Waist circumference (midway between the lower rib and the iliac crest) was assessed to the nearest 0.5cm. The Balke protocol was followed to determine VO₂max (Balke and Ware 1959). Treadmill speed was set at 5.3 km/hour, and the initially flat gradient was increased to 2% in the second minute and by 1% with each subsequent minute.

## Statistical analyses

Repeated-measures analysis of variance (ANOVA) (one group x three times) was used to explore the changes in physiological outcomes at baseline, intervention outcome visit, and post-intervention follow-up, and paired-sample t-test was conducted to examine changes in VO₂max at baseline and intervention outcome visit. Non-normally distributed outcomes (waist circumference) were transformed by natural logarithm before ANOVA analyses.

To address the first aim, Generalised Estimating Equations (GEE) models were used to examine changes in PA and sleep characteristics during the intervention and post-intervention follow-up compared to baseline and on exercise days vs. non-exercise days during the intervention period. The analysis incorporated all valid days per participant during each study phase, with repeated data per person taken into account using an unstructured correlation matrix. Reported values represent the overall mean levels during each period. Due to normality violations, all PA and sleep characteristics were transformed by natural logarithm for all analyses.

To address the second aim to examine the bidirectional associations between PA and sleep, two datasets were produced. In the first dataset, to assess whether daytime PA was associated with sleep that night, sleep characteristics on night i were aligned with PA characteristics on day i. In the second dataset, to examine if sleep characteristics were associated with PA characteristics the next day, sleep variables on night i were arranged in line with PA variables on day i+1. Using each dataset, GEE models with an unstructured correlation structure were used to assess day-to-day associations between PA and sleep characteristics. The association between each PA outcome and sleep outcome were examined using separate models. All models were adjusted for age, BMI, and the intervention period (baseline, intervention, and post-intervention follow-up). Further, two sensitivity analyses were conducted. First, to assess whether the association of PA with sleep characteristics was modified by the time of day when M30CONT occurred, an interaction term for the M30CONT\*timing of the M30CONT (morning>8h before bedtime; afternoon=4–8h before bedtime; or evening<4h before bedtime) (Buman et al. 2014) was added to models. Second, to explore whether the bidirectional associations between PA and sleep were different during different phases of the study (baseline phase, intervention phase, post-intervention phase), an interaction term for the intervention period\*PA/sleep outcome was added to models and then further by stratifying significant interactions by intervention period and non-intervention periods (baseline and post-intervention periods combined). All analyses were performed using SPSS v. 24. An alpha level of 0.05 was set for all analyses.

# Results

Fifteen men were recruited into the study (Figure 1). Table 1 presents participants’ descriptive characteristics at baseline. Of the 15 participants who completed the study, 14 were included in the analyses. One participant was excluded due to working shifts. There were no changes in participants’ physiological outcomes (BMI, body fat, and waist circumference) at intervention outcome visit or post-intervention follow-up (all p>0.05) (Table S1). However, there was a significant improvement in fitness level measured via VO₂max test at intervention outcome visit (29.4±5.1 (ml/kg/min), compared to baseline (27.9±4.3 (ml/kg/min), p=0.001)) (Table S1). None of the participants reported being on sleep medication and one was on antidepressants. Three participants switched to a combination of treadmill walking and stationary bike at the beginning of the study due to reporting mild-to-moderate tightness in calves and one switched to stationary bike during week three until the end of the exercise programme due to mild shin pain. One participant reported occasional mild knee and moderate lower back pain but chose to adhere to treadmill walking throughout the exercise programme. All 14 participants returned devices with valid wear. Participants had a mean of 76.7±14.7 of valid accelerometer days; the maximum number of valid days possible per participant was 92 and 48 days minimum. The fewer number of valid days was due to monitor malfunction, not because of non-compliance (which meant the loss of data of 15 consecutive days). Excluded days totalled 190 (8 files=113 days post-calibration error was greater than 0.01 *g*, 77 days with less than 16 hours of data). This resulted in 1074 data points in total. The number of valid days at each intervention period was 8.7±3.8, 48.8±11.3, 23.5±6.1 at baseline, during the intervention, and at post-intervention follow-up, respectively.

## Impact of eight-week exercise intervention on sleep characteristics

Participants’ PA and sleep characteristics at baseline and during the intervention and at one-month post-intervention follow-up are shown in Table 2. There was an increase in all PA characteristics during the intervention and post-intervention follow-up compared to baseline (all p>0.05). Sleep duration was longer during the intervention (06:35 hh:mm, 95%CI: 06:12, 06:44) and post-intervention follow-up (06:32 hh:mm, 95% CI: 06:17, 06:49) compared to baseline (05:12 hh:mm, 95%CI: 04:30, 05:39, p=0.001)). There were no other changes in sleep characteristics from baseline and during the intervention and at post-intervention follow-up.

PA and sleep characteristics on non-exercise and exercise days during the intervention period are presented in Table 3. The mean number of exercise days during the intervention was 21.2 ± 1.7 and non-exercise days 31.4 ± 2.9. All PA characteristics, except for light activity, were significantly higher on exercise days compared to non-exercise days (all p<0.05). However, there were no differences in sleep characteristics on exercise compared to non-exercise days (all p>0.05).

## Bidirectional day-to-day associations between PA and sleep characteristics

### PA characteristics predicting sleepcharacteristics

Predictor estimates, significance levels, and model parameters are presented in Table 4a for the model predicting sleep from PA characteristics. Higher overall activity and MVPA were associated with earlier sleep onset time. Every 10% higher ACC and MVPA were associated with 0.16% (95% CI:(-0.30, -0.02)) and 0.04% (95% CI:(-0.05, 0.03)) earlier sleep onset, respectively. Higher PA predicted more WASO the following night. For each 10% higher ACC, M30CONT and MVPA, WASO was 3.14% (95% CI:(0.30, 5.05)), 1.70% (95% CI:(0.71, 2.70)) and 0.55% (95% CI:(0.27, 0.85)) higher, respectively. There were no other associations between PA with sleep characteristics.

These results are also presented as standardised (per SD) regression values in Supplementary Table S2a.

### Sleep characteristics predicting PA characteristics

Predictor estimates, significance levels and model parameters are shown in Table 4b for the model predicting PA from sleep characteristics. Later timing of sleep onset was associated with less PA the next day. A sleep onset that was 10% later was associated with a 7.57% (95% CI:(-12.59, -2.26)), 14.02% (95% CI:(-22.04, -5.19)) and 14.28% (95% CI:(-25.85, -0.89)) lower ACC, M30CONT and MVPA, respectively. Longer sleep duration predicted less MVPA and light activity, such that a 10% increase in sleep duration was associated with 8.71% (95% CI:(-12.78, -4.44)) lower MVPA and 3.09% (95% CI:(-4.64, -1.50)) lower light activity. Higher WASO predicted more PA, whereas greater SE predicted less PA the next day. Each 10% higher WASO was associated with a 1.04% (95% CI:(0.72, 1.36)) higher ACC, 1.29% (CI:(95% 0.76, 1.82)) higher M30CONT and 4.39% (95% CI:(2.78, 6.03)) higher MVPA. A 10% greater SE was associated with a 5.74% (95% CI:(-7.65, -3.80)), 9.52% (95% CI:(-12.02, -6.95)) and 22.26% (95% CI:(-27.10, -17.15)) lower ACC, M30CONT and MVPA, respectively.

These results are also presented as standardised (per SD) regression values in Supplementary Table S2b.

## Sensitivity analyses

Sensitivity analyses revealed that there was a main effect of timing of M30CONT on sleep onset and WASO (p<0.05) and an interaction of the timing and magnitude of M30CONT with sleep onset and WASO (p<0.05 for interactions) (Table 5). Every 10% higher M30CONT in the afternoon (4-8h before sleep onset) was associated with 0.09% (95% CI:(-0.17, -0.01)) earlier sleep onset, whereas every 10% higher M30CONT in the evening (less 4 hours before sleep onset) was associated with a 0.48% (95% CI:(0.18, 0.77)) later sleep onset. Additionally, M30CONT occurring in the morning and afternoon was associated with greater WASO (p=0.001), whereas no association was seen with higher M30CONT in the evening (p>0.05).

These results are also presented as standardised (per SD) regression values in Supplementary Table S3.

In sensitivity analyses exploring whether the bidirectional associations between PA and sleep were different during different phases of the study, there were no interactions (p>0.05) between the intervention period\*PA/sleep outcomes for any of the significant main effects, apart from one (sleep onset predicting MVPA) where no association was observed during the intervention period.

# Discussion

The current study provides new insights into the associations between device-assessed PA and sleep over an extended period of time in men with obesity. First, sleep duration significantly increased during the intervention period and at post-intervention follow-up, however, there were no changes in outcomes related to sleep quality. Second, when examining the bidirectional associations between PA and sleep, there were no day-to-day associations between PA and sleep duration during the following night, but longer sleep duration was associated with fewer minutes of MVPA and light activity the next day. Higher PA predicted earlier timing of sleep onset that night. In turn, later sleep onset was associated with a decrease in PA the following day. Additionally, higher PA was associated with more time awake after sleep onset the following night, but not with sleep duration, perhaps reflecting more rest with greater time spent in bed, but not more time asleep. The opposite association was also true, with greater time awake after sleep onset, but not sleep duration, associated with greater physical activity the next day. Finally, the time of day and magnitude of most active continuous 30 minutes was associated with earlier sleep onset if it occurred in the afternoon but later sleep onset if it occurred in the evening, and with higher WASO when it occurred in the morning and afternoon but not in the evening.

## Impact of eight-week exercise training on sleep

In the present study sleep duration was longer during the intervention period and at post-intervention follow-up compared to baseline. Similar increases in sleep duration (~80 min) were also observed in a 13-week exercise intervention study in overweight men (mean BMI 28 kg/m2), however, it should be highlighted that the intervention consisted of daily high-intensity exercise training (~ 60 minutes/day or 600 kcal/d-1) (Kjeldsen et al. 2013). Another study that investigated the effects of regular exercise on sleep in overweight and obese men (mean BMI 29.2 kg/m2) found that while sleep duration did not change after six months of an aerobic exercise training programme, there was an improvement in sleep quality outcomes (e.g. reduced WASO and higher sleep efficiency) in the exercise group compared to baseline (Tan et al. 2016). However, this study included participants with chronic insomnia symptoms. It was previously reported that individuals with pre-existing sleep complaints may experience greater effects of exercise on sleep (Driver and Taylor 2000). In the current study, although sleep duration increased during the intervention and at post-intervention follow-up compared to baseline, despite an increase in all PA outcomes on exercise days there was no change in sleep duration on exercise days compared to non-exercise days, suggesting that PA had a chronic but not an acute day-to-day effect on sleep duration.

It is worth noting that information on sleep disorder symptoms of the participants was not collected at baseline because sleep was not the primary outcome of interest for the TEAM GB study. However, none of the participants reported taking sleep-related medications. Sleep-related disorders such as insomnia symptoms were previously reported by 28% in the middle-aged population in the UK (Kyle et al. 2017) and sleep apnoea reported to be highly prevalent among men with obesity (Heinzer et al. 2015). Therefore, the magnitude of the effect of the exercise intervention on sleep may be different in participants with different baseline sleep symptomology.

The increase in sleep duration on the initiation of exercise training in this cohort is likely to be clinically significant. Participants’ baseline sleep duration was only 5.2 hours which is under the recommended 7-8 hours (Dolezal et al. 2017), which increased to 6.6 hours during the intervention period. Short sleep duration has been associated with a host of adverse cardiometabolic outcomes, such as overall and central obesity, insulin resistance, hypertension, dyslipidemia, and atherosclerosis (Reutrakul and Van Cauter 2018). However, the increase in sleep duration observed during the exercise intervention to approximately 6.5 hours is reflective of population averages when sleep is measured using device-based methods (Lauderdale et al. 2006) and is likely to attenuate these risks. For instance, previous research has shown that an increase in the risk of mortality and morbidity due to short sleep is largely only evident below six hours of sleep duration (Brady et al. 2018).

Although not statistically significant, sleep efficiency also appeared to improve by approximately 6% during the intervention supporting the evidence that exercise may enhance sleep quality (Kredlow et al. 2015). Interestingly, the impact of the intervention extended into the period after the intervention stopped where sleep duration remained at 6.5 hours. However, PA levels also remained elevated, suggesting the intervention led to an adoption of a more physically active lifestyle which continued to support a healthier sleep profile.

## Bidirectional day-to-day associations between PA and sleep

One of the main findings was the mutual association between PA and the timing of sleep onset. Consistent with the study by Master and colleagues (2019) who reported that more MVPA was associated with earlier sleep onset in adolescents, higher overall activity and MVPA predicted earlier sleep onset that night in this study, suggesting that a more active day may lead to an earlier bedtime. Additionally, timing and magnitude of most active continuous 30 minutes of the day 4–8h before bedtime predicted earlier sleep onset, whereas, when it occurred in the evening, it was associated with later subsequent sleep onset. This suggests that being active within a 4-hour time frame before bedtime encourages a later sleep onset. Further, later sleep onset was negatively associated with the next day’s PA characteristics, which is consistent with studies in adults (Shechter and St-Onge 2014) and children (Jarrin et al. 2013; McGrath and Drake 2013; Master et al. 2019). Later timing of sleep beyond sleep duration has previously been reported to be associated with unhealthy behaviours including unfavourable PA profile (Knutson and von Schantz 2018). Therefore, the bidirectional association between PA and sleep onset observed in this study suggests that increasing PA could be used to encourage earlier sleep onset as long as it does not occur close to bedtime. In turn, encouraging earlier bedtime may lead to an increase in PA the following day.

In the present study, higher PA was associated with higher WASO and lower sleep efficiency, which contradicts most of the prevailing literature demonstrating a positive association between sleep quality and PA (Loprinzi and Cardinal 2011) and exercise (Kredlow et al. 2015). However, Pesonen et al. (2011) also found that higher PA levels were associated with both shorter and worse sleep that night and vice versa in adolescents. Moreover, some acute trials reported a negative effect of exercise on WASO, however, this effect was moderated by the timing of the exercise (>8h or <4h before sleep) (Chennaoui et al. 2015). In this study, the timing and magnitude of the most active continuous 30 minutes of the day was also associated with WASO. While consistent with some studies reporting increased WASO following exercise session eight hours before sleep (Chennaoui et al. 2015), present findings contradict findings from meta-analyses of acute exercise trials that showed no association between WASO and exercise 3-8 hours before bedtime (Kredlow et al. 2015) and beneficial effects of evening exercise (Stutz et al. 2019). Another potential explanation for increased WASO could be attributed to adverse effects related to musculoskeletal pain from the exercise intervention reported by five participants. However, exercise-related adverse events were of mild-to-moderate severity in the current study, with the exercise mode altered to minimise pain. Previously only strenuous exercise (Driver and Taylor 2000) and overtraining in athletes (Chennaoui et al. 2015) have been linked to disturbed sleep. Possibly, other external determinants e.g. work schedules and family commitments may have a greater effect on sleep-wake cycles than the direct association between PA and sleep (Mitchell et al. 2016). Furthermore, although prone to bias, subjective assessment of sleep quality reflects one’s satisfaction with sleep and can be as important as device-based assessment (Kishida and Elavsky 2016). Thus, future work should incorporate potential moderators of the association between PA and sleep in the analyses and include self-report alongside device-based measures of sleep.

The strengths of this study include the longitudinal study design which enabled exploration of the direction of the associations between PA and sleep. Another strength is the device-based assessment of PA and sleep using non-proprietary PA and sleep metrics. However, accelerometers have limited ability to record static movement and activities such as cycling, and although they provide some key sleep outcomes, it is not possible to assess sleep stages (e.g. slow-wave sleep and rapid eye movement sleep) that have been shown to be affected by exercise (Kredlow et al. 2015). Moreover, WASO estimated using accelerometry indicates more movement during sleep but it does not differentiate between sleep and wakefulness. An important indicator of sleep quality – sleep onset latency that has been found to be associated with subsequent activity levels and vice versa (Baron et al. 2013) was not included in the analyses as the sleep detection algorithm used in the current study does not facilitate detection of sleep onset latency. Another limitation is that the present findings can only be interpreted at group-level. Previous studies have shown that the associations between PA and sleep can be different at intra- or interindividual levels (Kishida and Elavsky 2016). Limitations which impact generalisability of the current study include the small homogenous sample size of white European ethnicity participants; however, it provides a relevant addition to the current evidence on the bidirectional associations between PA and sleep which have been predominantly examined in women (Lambiase et al. 2013; Gabriel et al. 2017; Mitchell et al. 2016; Kishida and Elavsky 2016; Baron et al. 2013). Additionally, the findings of the present study are limited to men with low levels of activity. Evidence suggests that fit and habitually active individuals are typically good sleepers, therefore, the effects of exercise on sleep are limited by the ceiling effects (Buman and King 2009). Several experimental studies including unfit and sedentary participants have demonstrated improvements in sleep indices after participating in an exercise intervention (Buman and King 2009), indicating that the magnitude of the effect of exercise on sleep may be different depending on an individual’s fitness and habitual activity levels. It has also been suggested that time spent outdoors, time of year and week, and employment status that were not addressed in this study can be significant moderators that influence both behaviours (Kishida and Elavsky 2016; Mitchell et al. 2016) and that may have provided context to the present findings. The one-group non-randomised nature of the intervention is also a limitation, with reported effects potentially due to external factors that were not captured in the analysis.

In conclusion, this study suggests that in a cohort of men with obesity, an exercise intervention substantially increased usual sleep duration which was maintained once the intervention ceased. However, the chronic effect on sleep duration was not mirrored acutely where the day-to-day variability in sleep duration was not affected by the proceeding days’ activity profile. Nevertheless, there was a bidirectional day-to-day association between higher PA and earlier timing of sleep onset and between higher PA and more time awake after sleep onset which warrants further investigation. This study further highlights the complex relationships between PA and sleep.

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Table 1. Descriptive characteristics (median (25th -75th percentile)) of the participants at baseline (N = 14).

|  |  |
| --- | --- |
| **Variable** | **Baseline** |
| Age (yrs) | 48.0  (46.0-55.0) |
| BMI (kg/m²) | 35.1  (34.1-36.9) |
| Body fat (%) | 34.0  (32.2-35.1) |
| Waist circumference (cm) | 118.9  (115.2-121.2) |
| VO₂max (ml/kg/min) | 27.4  (24.7-32.9) |

BMI = body mass index

VO₂max = maximum rate of oxygen consumption during incremental exercise

Table 2. Physical activity and sleep characteristics before and during the intervention and at post-intervention follow-up. Number of valid days at baseline = 8.7±3.8, during the intervention = 48.8±11.3, post-intervention follow-up = 23.5±6.1.

|  |  |  |  |
| --- | --- | --- | --- |
|  | **Baseline** | **During intervention** | **Post-intervention follow-up** |
| *Physical activity characteristics* |  |  |  |
| Acceleration (m*g*)a | 18.1 | 38.0\*\* | 37.8\*\* |
|  | (14.1, 23.2) | (34.6, 41.7) | (33.9, 42.2) |
| Most active 30 min (m*g*)b | 103.9 | 127.3\*\* | 130.9\* |
|  | (93.1, 116.0) | (114.8, 141.2) | (106.2, 161.4) |
| MVPA (min)c | 4.9 | 20.0\*\* | 19.4\*\* |
|  | (2.0, 12.1) | (16.2, 24.6) | (15.2, 24.7) |
| Light activity (min)d | 91.7 | 186.2\*\* | 193.0\*\* |
|  | (69.8, 120.4) | (163.0, 212.8) | (167.3, 222.7) |
| *Sleep characteristics* |  |  |  |
| Sleep onset (hh:mm) | 00:05 | 23:38 | 00:04 |
|  | (23:40, 00:31) | (23:14, 00:02) | (23:41, 00:28) |
| Sleep duration (hh:mm)e | 05:12 | 06:35\*\* | 06:32\*\* |
|  | (04:30, 05:39) | (06:12, 06:44) | (06:17, 06:49) |
| Wake after sleep onset (hh:mm) | 00:45 | 00:46 | 00:43 |
|  | (00:37, 00:55) | (00:39, 00:54) | (00:35, 00:53) |
| Sleep efficiency (%) | 81.9 | 87.6 | 85.5 |
|  | (76.2, 87.9) | (86.1, 89.1) | (82.2, 88.9) |

Data are displayed as mean (CI).

Covariates: age, BMI

BMI = body mass index

MVPA = moderate-to-vigorous activity

a Significant increase in acceleration during intervention and post-intervention follow-up compared to baseline

b Significant increase in most active 30 minutes m*g* during intervention and post-intervention follow-up compared to baseline

c Significant increase in MVPA during intervention and post-intervention follow-up compared to baseline

d Significant increase in light activity during intervention and post-intervention follow-up compared to baseline

e Significant increase in sleep duration during intervention and post-intervention follow-up compared to baseline

\**p* < 0.05.

\*\**p* < 0.001.

Table 3. Physical activity and sleep characteristics on non-exercise and exercise days the 8-week intervention period. Number of valid exercise days = 21.2 ± 1.7 and non-exercise n = 31.4 ± 2.9.

|  |  |  |
| --- | --- | --- |
| *Physical activity characteristics* | **Non-exercise day** | **Exercise day** |
| Acceleration (m*g*)**\*\*** | 30.3 | 34.6 |
|  | (28.2, 32.5) | (31.5, 37.9) |
| Most active 30 min (m*g*)**\*\*** | 106.3 | 168.6 |
|  | (96.4, 117.3) | (144.1, 197.2) |
| MVPA (min)**\*\*** | 11.6 | 29.8 |
|  | (9.1, 14.8) | (21.9, 40.4) |
| Light activity (min) | 171.0 | 179.4 |
|  | (154.0, 190.0) | (164.8, 195.3) |
| *Sleep characteristics* | **Non-exercise day** | **Exercise day** |
| Sleep onset (hh:mm) | 23:48 | 23:52 |
|  | (23:28, 00:08) | (23:35, 00:09) |
| Sleep duration (hh:mm) | 06:05 | 06:07 |
|  | (05:52, 06:18) | (05:54, 06:22) |
| Wake after sleep onset (hh:mm) | 00:42 | 00:40 |
|  | (00:35, 00:50) | (00:33, 00:49) |
| Sleep efficiency (%) | 89.4 | 88.6 |
|  | (87.1, 91.6) | (86.3, 91.0) |

Data are displayed as mean (CI).

Covariates: age, BMI

BMI = body mass index

MVPA = moderate-to-vigorous physical activity

Significant increase in acceleration, most active 30 min m*g*, MVPA on exercise days compared to non-exercise days: **\*\****p* <0.001.

Table 4-a. GEE models for physical activity characteristics predicting sleep characteristics.

|  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
| **Outcome:** | **Sleep onset** | | | **Sleep duration (min)** | | | | **Wake after sleep onset (min)** | | | | **Sleep efficiency (%)** | | | |
|  | b (95% CI) | *p* | | b (95% CI) | | *p* | | b (95% CI) | | | *p* | b (95% CI) | | | *p* |
| **Acceleration (m*g*)** | **-0.162** | **0.027** | | -0.124 | | 0.635 | | **3.136** | | | **0.001** | -0.162 | | | 0.191 |
|  | (-0.295, -0.019) |  | | (-0.637, 0.392) | |  | | (0.295, 5.051) | | |  | (-0.400, 0.076) | | |  |
| **M30CONT (m*g*)** | -0.019 | 0.637 | | -0.209 | | 0.246 | | **1.701** | | | **0.001** | 0.010 | | | 0.751 |
|  | (-0.105, 0.067) |  | | (-0.561, 0.124) | |  | | (0.708, 2.695) | | |  | (-0.029, 0.048) | | |  |
| **MVPA (min)** | **-0.038** | **0.001** | | -0.038 | | 0.345 | | **0.554** | | | **0.001** | 0.038 | | | 0.487 |
|  | (-0.048, 0.029) |  | | (-0.124, 0.048) | |  | | (0.267, 0.852) | | |  | (-0.076, 0.162) | | |  |
| **Light (min)** | 0.067 | 0.477 | | -0.247 | | 0.106 | | -1.382 | | | 0.084 | -0.114 | | | 0.335 |
|  | (-0.124, 0.258) |  | | (-0.542, 0.057) | |  | | (-2.930, 0.191) | | |  | (-0.333, 0.114) | | |  |
| 4-b. GEE models for sleep characteristics predicting physical activity characteristics. | | | | | | | | |  |  | |  |  |  | | |
| **Outcome:** | **Acceleration (m*g*)** | | | | **Most active 30 min (m*g*)** | | | **MVPA (min)** | | | | **Light (min)** | |  | | |
|  | b (95% CI) | | *p* | | b (95% CI) | | *p* | b (95% CI) | | *p* | | b (95% CI) | | *p* | | |
| **Sleep onset** | **-7.571** | | **0.006** | | **-14.021** | | **0.002** | **-14.275** | | **0.037** | | -3.529 | | 0.475 | | |
|  | (-12.592, -2.261) | |  | | (-22.038, -5.188) | |  | (-25.850, -0.892) | |  | | (-12.567, 6.452) | |  | | |
| **Sleep duration (min)** | -1.194 | | 0.169 | | -1.729 | | 0.141 | **-8.709** | | **0.001** | | **-3.087** | | **0.001** | | |
|  | (-11.687, 0.504) | |  | | (-3.988, 0.583) | |  | (-12.783, -4.444) | |  | | (-4.636, -1.504) | |  | | |
| **WASO (min)** | **1.035** | | **0.001** | | **1.285** | | **0.001** | **4.392** | | **0.001** | | -0.133 | | 0.547 | | |
|  | (0.717, 1.363) | |  | | (0.756, 1.818) | |  | (2.783, 6.027) | |  | | (-0.570, 0.305) | |  | | |
| **Sleep efficiency (%)** | **-5.738** | | **0.001** | | **-9.523** | | **0.001** | **-22.261** | | **0.001** | | 2.265 | | 0.118 | | |
|  | (-7.650, -3.796) | |  | | (-12.023, -6.952) | |  | (-27.049, -17.150) | |  | | (-0.570, 4.935) | |  | | |

Data are displayed as beta-coefficients (CI). P < 0.05 taken to indicate significance. BMI = body mass index

Physical activity and sleep variables are log transformed. M30CONT = most active continuous 30 minutes

Beta-coefficients should be interpreted as the percent change in Y for every 10% change in X. MVPA = moderate-to-vigorous activity

Covariates: age, BMI, intervention period WASO = wake after sleep onset

Table 5. GEE models for the interaction between timing and the magnitude of most active 30 minutes (morning>8 h before bedtime; afternoon=4–8 h before bedtime; or evening<4 h before bedtime) during the day predicting sleep characteristics.

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Outcome:** | **Sleep onset** | | **Wake after sleep onset (min)** | |
|  | b (95% CI) | *p* | b (95% CI) | *p* |
| **Morning** | 0.019  (-0.067, 0.114) | 0.595 | **2.226**  (1.323, 3.136) | **0.001** |
| **Afternoon** | **-0.086**  (-0.171, -0.010) | **0.025** | **2.695**  (1.437, 3.432) | **0.001** |
| **Evening** | **0.478**  (0.181, 0.765) | **0.001** | 0.382  (-0.382, 4.521) | 0.852 |

Data are displayed as beta-coefficients (CI). P < 0.05 taken to indicate significance.

Physical activity and sleep variables are log transformed.

Beta-coefficients should be interpreted as the percent change in Y for every 10% change in X.

Covariates: age, BMI

BMI = body mass index