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Impaired Insulin Profiles Following a Single Night of Sleep Restriction: The Impact of Acute Sprint Interval Exercise

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Running head: Exercise and insulin sensitivity following sleep restriction

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Abstract

Experimental sleep restriction has demonstrated reduced insulin sensitivity in healthy individuals. Exercise is well-known to be beneficial for metabolic health. A single bout of exercise has the capacity to increase insulin sensitivity for up to two days. Therefore, the current study aimed to determine if sprint interval exercise could attenuate the impairment in insulin sensitivity after one night of sleep restriction in healthy males. Nineteen males were recruited for this randomised crossover study which consisted of four conditions – control (CON), sleep restriction (SR), control plus exercise (CE), and sleep restriction plus exercise (SRE). Time in bed was 8 h (2300 – 0700) in the control conditions and 4 h (0300 – 0700) in the sleep restriction conditions. Conditions were separated by a 1 wk entraining period. Participants slept at home and compliance was assessed using wrist actigraphy. Following the night of experimental sleep, participants either conducted sprint interval exercise or rested for the equivalent duration. An oral glucose tolerance test was then conducted. Blood samples were obtained at regular intervals for measurement of glucose and insulin. Insulin concentrations were higher in SR than CON ($P = 0.022$). Late-phase insulin AUC was significantly lower in SRE than SR (862 ± 589 and 1267 ± 558 ; $P = 0.004$). Glucose AUC was not different between conditions ($P = 0.207$). These findings suggest that exercise improves the late post-prandial response following a single night of sleep restriction.

Key Words: Glucose metabolism, sleep loss, high intensity exercise

Introduction

Short sleep durations are becoming increasingly common, with almost 75% of adults in Great Britain sleeping outside the recommended 7-9 hours each night (Hirshkowitz et al., 2015). Chronic short sleep is associated with increased risk of developing many diseases, including type 2 diabetes (Yaggi et al., 2006). Experimental studies demonstrate impaired glucose control following sleep restriction (Knutson et al., 2007), with a single night of restriction reducing whole-body insulin sensitivity by 20% (Donga et al., 2010).

The proposed mechanisms underlying impaired glucose regulation following sleep restriction include altered peripheral insulin sensitivity (Broussard et al., 2012; Rao et al., 2015) and changes to the metabolic profile, favouring fatty acid transportation (Davies et al., 2014) which may interfere with insulin signalling. However, while the detrimental effects of sleep restriction are clear, few studies have focussed on strategies to counter the impairment.

Exercise may have the potential to alter these proposed underlying mechanisms (Saner et al., 2018). A single bout of exercise positively impacts glucose regulation for up to 24 hours (Koopman et al., 2005). This improvement in glucose regulation is apparent with various types of exercise (Breen et al., 2011; Gillen et al., 2012); although high intensity exercise may be superior to moderate intensity exercise for improving insulin sensitivity (Rynders et al., 2014; Ortega et al., 2015). Sprint interval exercise has been shown to produce improvements in insulin area under the curve (AUC) and the insulin sensitivity index when measured 30 min post cessation of the exercise bout in healthy

males (Ortega et al., 2015). Whether or not improvements such as these would occur in sleep-restricted individuals remains unknown.

Exercise appears to be a promising intervention to alleviate the impairment in insulin sensitivity following total sleep deprivation. Two weeks of high intensity exercise training has been shown to attenuate the insulin response to an oral glucose tolerance test after one night of total sleep deprivation (de Souza et al., 2017). However, it remains unclear if a single bout of exercise performed after partial sleep restriction can produce similar outcomes.

Consequently, this study aimed to investigate the effect of a single bout of sprint interval exercise on whole-body insulin sensitivity, following a single night of sleep restriction. We hypothesised that insulin sensitivity would be reduced following sleep restriction, and that this effect would be attenuated when exercise was performed.

Methods

Participants

Nineteen healthy males (mean \pm SD; age 25 ± 8 y; body mass 81.4 ± 12.0 kg; stature 180 ± 7 cm) participated in this study. Exclusion criteria included shift workers, regular travel across time zones (>3 times a year) or in the past 4 weeks, presence of any disorders which may influence glycaemic control (such as diabetes) or sleep (such as obstructive sleep apnea), current or previous medication in the past year which may have impacted on glucose metabolism or sleep, alteration of sleep, dietary, or physical activity patterns in the previous 3 months, a history of drug or alcohol abuse or eating

disorders, following a specific diet which may influence the results, such as intermittent fasting, a habitual bedtime before 2200 or after 0100, or a habitual wake time before 0600 or after 0900. Individuals were also excluded if they had a contraindication to exercise, poor sleep quality (defined as a Pittsburgh Sleep Quality Index [PSQI] (Buysse et al., 1989) score of above 5), or were classed as extreme morning or evening types, assessed by the morningness-eveningness questionnaire [MEQ] (Horne & Ostberg, 1976). The study protocol was approved through the Northumbria University Ethical Approval system. All participants provided written informed consent prior to participation.

Study Design

This randomised crossover trial consisted of a familiarisation visit and four one-night experimental trials. The experimental conditions were control (CON), control plus exercise (CE), sleep restriction (SR) and sleep restriction plus exercise (SRE). Each condition involved a single night of either 8 h time in bed from 2300 to 0700 (CON and CE) or 4 h time in bed from 0300 to 0700 (SR and SRE). Four hours sleep has been shown to reduce insulin sensitivity in previous research (Donga et al., 2010). The morning following the control or restricted sleep, participants either rested (CON and SR) or undertook a bout of sprint interval exercise (CE and SRE). All experimental conditions were separated by at least 1 week to prevent carryover effects (van Leeuwen et al., 2010), but no more than 3 weeks.

Throughout the study participants slept at home. Experimental trials were preceded by a one-week entraining period, in which participants were asked to keep a consistent bed and wake time. Wrist actigraphy (GeneActiv, Activinsights Ltd, UK) was used in

conjunction with time-stamped text messages to ensure compliance. Participants sent hourly messages to the researcher between 2300 and 0300 in SR and SRE conditions. Participants were informed which condition they were on the day before each laboratory visit.

Diet was provided for the day preceding each experimental trial and was replicated across conditions. Diet was individualised from food diaries issued during the familiarisation session and total energy, carbohydrates, fat, and protein were kept within 10% of habitual intake. Participants were instructed to eat only the foods provided and to avoid consumption of any caffeine or alcohol, however were permitted to drink water ad libitum. Participants were also asked to refrain from exercise and napping during this time.

Study protocol

Familiarisation

One week prior to the first experimental visit participants attended the laboratory for a screening and familiarisation visit. Upon arrival participants were briefed on the study protocol and given the opportunity to ask questions before completing written informed consent. They completed several screening questionnaires – a physical activity readiness questionnaire, the PSQI (Buysse et al., 1989), and the MEQ (Horne & Ostberg, 1976). Following satisfactory completion of the screening questionnaires, they were issued with a 3-day food diary, actigraphy watch, and 7-day sleep diary.

Body mass and stature were measured using balance scales (SECA, UK) and a free-standing stadiometer (SECA, UK). Stature was measured to the nearest 0.1 cm and body mass to the nearest 0.1 kg.

Participants then underwent an exercise familiarisation, which was a reduced version of the study exercise protocol. The familiarisation exercise bout consisted of a 5-minute warm-up at 70 W on a cycle ergometer (Monark, Sweden), followed by two all-out 30-second sprints against 7.5% of their body mass. Sprints were separated by 4.5 minutes of active recovery at a self-selected pace. A 5-minute cool down was then completed.

Experimental trials

Participants went to bed at 2300 or 0300, depending on the condition. After getting up at 0700, participants arrived at the laboratory by public transport in a rested state at 0800, following a 10-hour overnight fast. Upon arrival, participants either completed a bout of sprint interval exercise (CE and SRE) or rested in a seated position for the equivalent duration (CON and SR). In all conditions participants were given a 30-minute recovery period, resting in a seated position. After the recovery period a 2-hour oral glucose tolerance test (OGTT) was conducted, and blood samples were drawn at regular intervals. The OGTT consisted of 82.5 g dextrose (MyProtein, UK) mixed with 300 ml water, consumed within 5 minutes. During the OGTT participants remained seated in the laboratory but were permitted to complete sedentary tasks. Participants left the laboratory at approximately 1130.

Exercise

The exercise protocol used in this study was based on previous research by Ortega et al (2015) who demonstrated a 142% increase in IVGTT-derived insulin sensitivity measured 30 minutes following sprint interval exercise in healthy males. All exercise was performed on a cycle ergometer (Monark Ergonomic 894E, Sweden), preceded and proceeded with a 5-minute warm-up and cool-down at 70 W.

The sprint interval exercise consisted of four all-out 30-second sprints against 7.5% of body mass, interspersed with 4.5 min of recovery. During the active recovery period, participants cycled at a self-selected pace against a resistance of 1 kg, which was the lowest permitted by the ergometer. Verbal encouragement was given throughout each sprint by the same researcher.

Blood collection and processing

Blood was collected at baseline (0), 15, 30, 45, 60, 90 and 120 minutes during the OGTT using the cannulation technique. For each sample, 8 ml was drawn into a syringe and transferred to a 10 ml serum vacutainer (Becton Dickinson, Sweden). Vacutainers were inverted to ensure thorough mixing. The samples were left to clot at room temperature for 30 minutes before being centrifuged at 3500 rpm at 4°C for 15 minutes. Serum was aliquoted into microtubes. A small amount of serum from each sample was taken up into a capillary tube and placed in microtubes containing 1 ml hemolysing solution (EKF Diagnostics, UK) for determination of glucose concentration. The remaining serum was frozen at -80°C until further analysis.

Serum glucose was measured using the Biosen C-line automatic glucose analyser (EKF Diagnostics, Germany). Serum insulin was measured using commercially available ELISA kits (Merckodia, Sweden), conducted according to manufacturers instructions. Intra- and inter-assay coefficients of variation were 6% and 14%, respectively.

Data analysis

Sample size calculation

Sample size was calculated using G*Power version 3.1.9.2 (Faul et al., 2007). Based on previous work showing a difference of 26.9 mg/dL in 2-hour glucose values during an OGTT between control and exercise conditions (Rynders et al., 2014), 18 participants were required to achieve 90% power.

Statistical analysis

Data are presented as mean \pm SD. Area under the curve (AUC) for glucose and insulin were calculated using the trapezoidal rule. AUC was calculated for total (2 hours), early-phase (0-60 minutes) and late-phase (60-120 minutes) during the OGTT. HOMA-IR (Matthews et al., 1985) and Matsuda Index (Matsuda & DeFronzo, 1999) were calculated to estimate insulin resistance and whole-body insulin sensitivity.

Data were analysed using SPSS Statistics version 22 (IBM, UK). Shapiro-Wilk tests were used to check for normality and any data which violated the assumption of normality were transformed using a natural logarithm transformation. Linear mixed modelling was used to compare glucose and insulin between conditions. $P < 0.05$ was used to indicate significance.

Results

Sleep

Average time to bed, wake time, time in bed (TIB), and total sleep time (TST) in each condition are presented in Table 1. Differences in TST were observed between CON and SR (mean difference 142 min; $P < 0.001$), CE and SRE (mean difference 182 min; $P < 0.001$), CE and SRE (mean difference 181 min; $P < 0.001$) and CON and SRE (mean difference 141 min; $P < 0.001$). No differences were observed between CON and CE ($P = 0.581$) or SR and SRE ($P = 1.000$).

Exercise

Peak power output (PPO) during each of the 30-second sprints is outlined in Table 2. PPO did not differ between conditions ($P = 0.644$), but a difference was observed over time ($P < 0.001$), with PPO significantly higher in the first sprint compared to the third (mean difference 106 W; $P = 0.009$) and fourth (mean difference 118 W; $P = 0.007$), and higher in the second compared to the fourth (mean difference 60 W; $P = 0.041$). Total work done was similar between conditions (63666 ± 12029 J in CE and 65045 ± 11294 J in SRE; $P = 0.340$).

Glucose and insulin

Glucose and insulin concentrations during the OGTT are displayed in Figure 1. Total, early- and late-phase AUC for glucose and insulin are shown in Figure 2.

Glucose concentrations did not show evidence of an effect of condition ($P = 0.216$) or interaction effect ($P = 0.146$). However, there was a significant effect of time during the OGTT ($P < 0.001$). No significant difference between conditions was found for peak glucose ($P = 0.158$). There was no evidence of an effect of condition ($P = 0.207$) for total glucose AUC. Likewise, late- and early-phase glucose AUC did not show any significant differences between conditions ($P = 0.264$ and $P = 0.122$, respectively).

Insulin concentrations demonstrated an overall difference between conditions ($P = 0.019$), time points ($P < 0.001$), and an interaction between conditions and time points ($P = 0.014$). Post-hoc analyses on these overall differences revealed significantly higher insulin concentrations in SR compared to CON ($P = 0.022$), with SR showing higher concentrations than CON at 30 min (40.04 ± 23.17 and 25.79 ± 13.94 $\mu\text{IU/ml}$; $P = 0.004$). Insulin concentrations were higher in SR compared to SRE at 60 minutes (31.40 ± 13.99 and 21.03 ± 10.67 $\mu\text{IU/ml}$; $P = 0.042$), 90 minutes (19.47 ± 11.11 and 15.52 ± 15.63 $\mu\text{IU/ml}$; $P = 0.002$), and 120 minutes (14.12 ± 14.84 and 5.99 ± 5.27 $\mu\text{IU/ml}$; $P = 0.003$). Total insulin AUC displayed a trend for an effect of condition ($P = 0.075$), with SR tending to be higher than CON ($P = 0.064$). Early- and late-phase insulin AUC also showed significant main effects of condition ($P = 0.010$ and $P < 0.001$, respectively).

The early-phase insulin AUC was higher in SR than CON (1472 ± 811 and 2044 ± 1129 ; $P = 0.048$). Late-phase displayed a lower AUC in SRE than SR (1267 ± 558 and 862 ± 589 ; $P = 0.004$).

HOMA-IR showed a main effect of condition ($P = 0.019$), with SR higher than CON (0.87 ± 0.99 and 1.64 ± 2.60 ; $P = 0.029$). Matsuda index was significantly different

between conditions ($P = 0.003$), with SR displaying a lower index than CON (25.31 ± 20.80 and 12.11 ± 6.38 ; $P = 0.020$).

Discussion

The present study demonstrated that one night of sleep restriction impaired insulin sensitivity, and that performing sprint interval exercise after sleep restriction may improve the late post-prandial response.

Participants displayed reduced insulin sensitivity after sleep restriction, indicated by increased insulin concentrations during the OGTT and decreased Matsuda index in SR compared to CON. This is consistent with previous research that has demonstrated reduced whole-body insulin sensitivity after a single night (Donga et al., 2010), and multiple nights of sleep restriction (Sweeney et al., 2017; Wang et al., 2016). Sleep restriction may impair whole-body insulin sensitivity through alteration of peripheral insulin signalling, with five nights of sleep restriction reducing peripheral, but not hepatic, insulin sensitivity (Rao et al., 2015). Furthermore, sleep restriction reduces Akt phosphorylation, which plays a key role in the insulin signalling pathway in peripheral tissues (Broussard et al., 2012).

We hypothesised that a bout of sprint interval exercise would attenuate the impairment in insulin sensitivity in sleep-restricted individuals. Whilst total insulin AUC was not significantly altered after the exercise bout, our findings suggest that there was an alteration to the late post-prandial response when comparing the sleep restricted conditions. However, we did not observe an improvement in insulin or glucose profiles

after exercise in the early-phase of the OGTT. Our findings are in contrast to Ortega and colleagues (2015) who demonstrated improved insulin and glucose 60 minutes following high intensity exercise. Methodological differences may explain this discrepancy, as an IVGTT was employed by Ortega and colleagues, whereas we used an OGTT. Gastric emptying plays a role during the OGTT whereas this is bypassed when glucose is injected rather than ingested orally. Gastric emptying rate may be slowed by intermittent high intensity exercise (Leiper et al., 2001), delaying the absorption of the glucose drink and therefore findings may not be comparable between an OGTT and IVGTT. Alternatively, it may be possible that sleep restriction alters the metabolic response to exercise.

Our findings reflect those by Rynders and colleagues (2014), who noted improvements in the late rather than total postprandial response after a bout of exercise. High intensity exercise is known to temporarily increase glucose due to gluconeogenesis and possible carbohydrate sparing for glycogen repletion (Marliss et al., 1992). It may be possible that as glucose regulation was measured 30 minutes after cessation of exercise, the temporary alterations in glucose regulation which occur during exercise were still influencing our measurements. Additionally, although not statistically significant, it appears that insulin concentrations in the early-phase of the OGTT were increased after exercise compared to rest in the current study. An increase in early-phase insulin secretion may influence late-phase responses, potentially through suppression of endogenous glucose production (Del Prato et al., 2002). This early-phase response may therefore contribute to the lower late-phase insulin AUC which was observed in SRE compared to SR. The late-phase of the OGTT has been shown to predict incident

diabetes independent of the early-phase (Lorenzo et al., 2012), suggesting that despite no overall change, a decrease in late-phase insulin AUC may be beneficial.

Whilst the present study suggests there is potential for exercise to positively influence the post-prandial response to an OGTT in sleep-restricted individuals, it was not designed to identify the possible mechanisms. Previous research has suggested that insulin action is improved after exercise through altered phosphorylation of components of the insulin signalling pathway in peripheral tissues (Wojtaszewski & Richter, 2006). As sleep restriction is thought to negatively impact peripheral insulin sensitivity (Broussard et al., 2012; Rao et al., 2015), it can be speculated that exercise improves insulin sensitivity in sleep-restricted individuals through alteration of insulin action in peripheral tissues.

The present study has some limitations which should be noted. Firstly, the study population consisted only of healthy males, meaning it may not be feasible to extrapolate the findings to other populations including females and individuals with metabolic abnormalities. Secondly, an OGTT does not enable measurement of metabolic characteristics such as glucose disposal and uptake, so gives limited information regarding the decrease in insulin sensitivity.

In summary, sprint interval exercise may offer some potential to attenuate the impairments in insulin sensitivity following a single night of reduced sleep. This may have implications for individuals facing sleep curtailment. To our knowledge, this is the first study to explore the effect of acute exercise on insulin sensitivity following sleep restriction. Therefore, future research may investigate whether exercise modality, intensity, duration, or timing influences the change in glucose regulation in sleep-

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restricted individuals. It would also be beneficial to investigate the time course of improved glucose regulation to determine whether the benefit of exercise persists for multiple hours as is the case in non-sleep-restricted individuals.

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Author contributions

The study was designed by ELS, IHW, DP and JGE; data were collected and analysed by ELS, IK, TH, and IHW; data interpretation and preparation of manuscript were conducted by ELS, IHW, and DP; all authors provided comments on the manuscript and approved the final version.

Conflicts of interest

The authors have no conflicts of interest to declare.

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Table 1. Sleep variables for each experimental condition.

	CON	CE	SR	SRE
Bed time (hhmm)	2303	2306	0305	0255
Wake time (hhmm)	0658	0659	0700	0657
TIB (min)	472 ± 27	472 ± 22	236 ± 19	244 ± 15
TST (min)	337 ± 95	377 ± 61	195 ± 43*†	196 ± 37*†

Bed time, wake time, time in bed (TIB) and total sleep time (TST) in control (CON), control plus exercise (CE), sleep restriction (SR) and sleep restriction plus exercise (SRE) condition. Data are mean ± SD. * indicates difference from CON ($P < 0.05$). † indicates difference from CE ($P < 0.05$).

Table 2. Peak power output and total work done during sprints.

	PPO (W)		Total work (J)	
	CE	SRE	CE	SRE
1	857 ± 189	853 ± 187	18356 ± 2903	18355 ± 3471
2	818 ± 177	804 ± 160	16551 ± 2921	16580 ± 3139
3	762 ± 194	736 ± 172	15488 ± 2842	15424 ± 3237
4	733 ± 179	746 ± 144	14686 ± 2408	15502 ± 2490

Peak power output (PPO) and total work during 4 all-out 30 s sprints performed the morning after a night of 8 h (CE) or 4 h (SRE) time in bed. No significant differences were observed between conditions. Data are presented as mean ± SD.

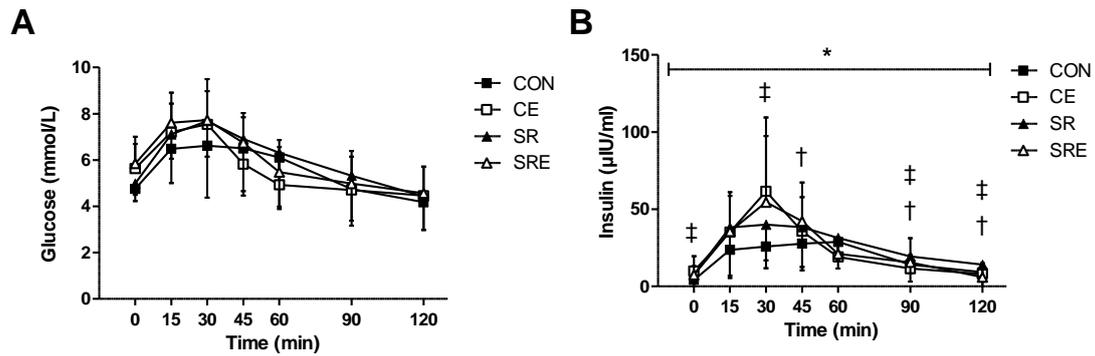


Figure 1. Glucose (A) and insulin (B) concentrations during the OGTT. $P < 0.05$ indicates significance. * indicates main effect of condition, with SR higher than CON. ‡ indicates difference between CON and SR. † indicates difference between SR and SRE.

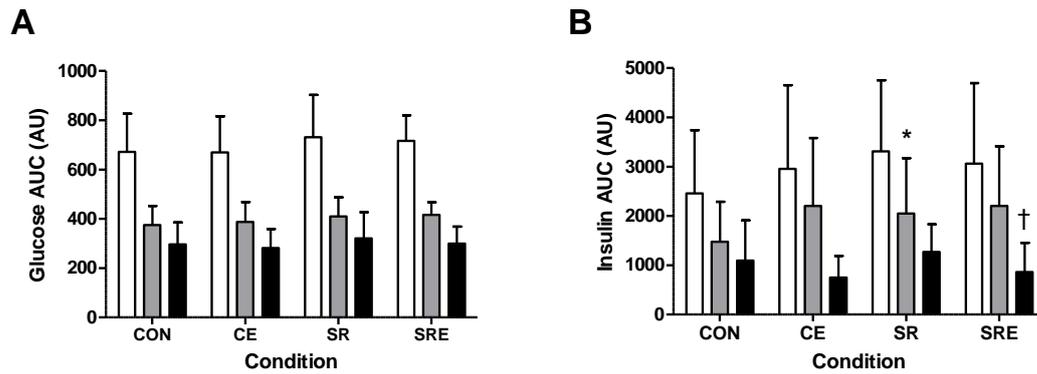


Figure 2. Total (white bars), early (grey shaded bars) and late (black bars) phase AUC for glucose (A) and insulin (B). Data are presented as mean \pm SEM. Main effect of condition for early and late AUC. * indicates significant difference ($P < 0.05$) compared to CON in early phase. † indicates significant difference ($P < 0.05$) compared to SR in late phase.