European Journal of Applied Physiology
https://doi.org/10.1007/s00421-025-05909-9

ORIGINAL ARTICLE

Acute impairments in glucose tolerance following one night of partial sleep restriction are not rescued by moderate-intensity walking in young men

Che-Wei Hsu[1] · Chieh-Sheng Tseng[1] · Richard S. Metcalfe[2] · Yung-Chih Chen[1]

Received: 27 April 2025 / Accepted: 5 July 2025

Abstract

Background High-intensity interval exercise ameliorates the impairment of postprandial metabolic health (e.g., glucose control) that is observed after a night of inadequate sleep. It is unknown whether moderate-intensity walking can elicit similar effects.

Methods Eleven healthy active males (age: 26 ± 2 yr; BMI: 22.8 ± 2.6 kg/m^2) took part in a randomized and repeated-measures cross-over study with: (1) normal sleep (NS; 8 h sleep opportunity); (2) sleep restriction (SR; 3 h sleep opportunity); and (3) sleep restriction + exercise (SRE; 3 h sleep opportunity followed by 30 min brisk walking at 50%$\dot{V}O_{2max}$). This was followed by a 2-h 75 g oral glucose tolerance test (OGTT), with plasma samples collected at baseline, immediately post-exercise (or sedentary), and at regular intervals during OGTT.

Results Total glucose area under the curve (tAUC) was lower in NS trial (924 [95%CI 865, 982] mmol/L) compared to both SR (1012 [95% CI 945, 1080] mmol/L, $p = 0.018$) and SRE trials (1006 [933, 1080] mmol/L, $p = 0.002$) and there was no difference between SR and SRE ($p = 1.00$). Insulin tAUC did not differ between trials ($p = 0.472$). There were no differences in fasting cortisol, c-reactive protein, and non-esterified fatty acids, and fasting and postprandial triacylglycerols, cholesterol, aspartate aminotransferase, and alanine aminotransferase concentrations between trials (all, $p > 0.05$).

Conclusion In healthy physically active young males, a single night of partial sleep restriction results in a decrease in glucose tolerance during a 2-h OGTT performed the following morning and this impaired response is not rescued by 30 min of brisk walking immediately prior to the OGTT.

Keywords Partial sleep deprivation · Brisk walking · Glycemic control · Metabolic health

Introduction

The American Academy of Sleep Medicine and the Sleep Research Society recommend that adults typically require at least 7 to 9 h of sleep per night (Watson et al. 2015). However, sleep surveys consistently find that around one-third of the general population, across different countries and races, regularly does not accumulate sufficient sleep to meet these recommendations (Wang et al. 2023). This is an important public health issue, because long term insufficient sleep has emerged as a strong and independent risk factor for the development of both type 2 diabetes and cardiovascular disease (Shan et al. 2015; Anothaisintawee et al. 2016; Spiegel et al. 2009). This appears to be attributable to impairments in metabolic regulation across multiple tissues following insufficient sleep. For example, reductions in hepatic and peripheral insulin sensitivity, whole-body glucose tolerance, and skeletal muscle mitochondrial function, and elevation of plasma non-esterified fatty acids (NEFAs) and inflammatory markers have been observed even following as little as between one and seven nights of sleep loss (Spiegel et al. 2009; Buxton et al. 2010; Reynolds et al. 2012; Sweeney

Communicated by Ting-Heng Chou .

✉ Yung-Chih Chen
yc.chen@ntnu.edu.tw

[1] Department of Physical Education and Sport Sciences, National Taiwan Normal University, 162, Section 1, Heping E. Rd, Taipei, Taiwan

[2] Applied Sports Technology, Exercise and Medicine Research Centre (A-STEM), Swansea University, Bay Campus, Fabian Way, Swansea, UK

Published online: 21 August 2025

et al. 2017; Saner et al. 2021; Broussard et al. 2016; Donga et al. 2010; Rao et al. 2015).

For many people, insufficient sleep is an inevitable consequence of occupational, social/lifestyle, and/or medical factors (Rosa et al. 2019; Korompeli et al. 2013; Coelho et al. 2023; Linton et al. 2015). As such, it is important to determine what interventions can be recommended to offset the negative effects of insufficient sleep on cardiometabolic health. Exercise is one potential strategy, given that improved insulin sensitivity and glycemic control is a well-described effect of exercise, both in the acute post-exercise period (Frampton et al. 2021; Hong et al. 2024) and in response to chronic exercise training (Munan et al. 2020; Houmard et al. 2004; Henriksen 2002). However, to date, only a limited number of studies have investigated the interactive effects of exercise and sleep restriction on metabolic health. An early study reported that daily aerobic physical activity partially prevented the detrimental changes in glucose tolerance observed following 60-h of total sleep restriction (TSR) (VanHelder et al. 1993). As 60 h of TSR is a relatively extreme example of sleep loss, several more recent studies have investigated the effects of exercise within sleep loss scenarios that are more likely to be encountered by the general population on a regular basis (Saner et al. 2021; Souza et al. 2017; Porter et al. 2021; Sweeney et al. 2020). These studies have provided evidence that exercise can prevent at least some of the detrimental effects of sleep restriction on insulin sensitivity, glycemic control, and/or metabolic regulation, when performed regularly for 2 weeks prior to a single night of TSR (Souza et al. 2017), when performed regularly during a 5-day period of partial sleep restriction (PSR) (Saner et al. 2021; Porter et al. 2021), and when performed in the morning immediately following a single night of PSR (Sweeney et al. 2020).

Although these findings are promising, it is notable that the majority of studies have applied high-intensity interval exercise (HIIE) (Saner et al. 2021; Souza et al. 2017; Sweeney et al. 2020), and many people may be unwilling or unable to engage in HIIE (Hardcastle et al. 2014), particularly during periods of inadequate sleep. Therefore, it is important to determine whether similar beneficial effects are observed with low-to-moderate-intensity continuous bouts of exercise that are more commensurate with current physical activity recommendations (Newsom et al. 2013; Manders et al. 2010; Bajpeyi et al. 2009). In particular, brisk walking is an accessible and equitable form of moderate-intensity physical activity/exercise that requires minimal equipment or planning to perform (Strain et al. 2024; Chen et al. 2024, 2018), and may represent a more feasible, safer, and more widely applicable intervention to promote during (unplanned) periods of inadequate sleep. Therefore, the primary aim of this study was to test the hypothesis that an acute bout of brisk walking would counteract the impairment in glycemic control following a single night of partial sleep restriction. A secondary aim was to investigate the effects of sleep restriction with and without brisk walking on a variety of other systemic biomarkers of metabolic health.

Methods

Participants

Eleven young, healthy, and recreationally active male participants were recruited via posters, social media, and by word-of-mouth, from Taipei city and the surrounding areas. Participants were aged between 23 and 31 years, with fasting glucose concentrations between 4.2 and 5.2 mmol/L, and self-reported that they had taken part in more than three sessions/week of structured exercise lasting at least 30 min over the preceding three months. Other inclusion criteria included: (1) absence of any cardiovascular or metabolic disease; (2) self-reporting no sleep issues for at least 3 months [defined as a Pittsburgh Sleep Quality Index (PSQI), score ≤ 5] (Buysse et al. 1989); (3) self-reporting a typical sleep time of between 7 and 9 h, with the habitual bedtime between 2200 and 0100 and a habitual wake time between 0600 and 0900, for at least the last month; (4) classified as an intermediate chronotype as assessed by the Morningness–Eveningness Questionnaire (Horne and Ostberg 1976); (5) weight stable for at least the last 3 months (less than 3% fluctuation of weight) (Stevens et al. 2006); (6) no smoking and drinking habits; (7) not taking any medications and/or supplementations known to impact metabolic responses, appetite, or sleep; (8) not a shift worker (at least 6 months) and no cross-time zone travel in the last month.

A summary of the participant characteristics is shown in Table 1. The study protocol was approved through National Taiwan Normal University Research Ethics Committee (ID: 202203HM022). All participants provided written informed consent prior to participation.

Experimental design

Following an initial eligibility screening check, and an assessment of baseline characteristics (e.g., maximal oxygen uptake, $\dot{V}O_{2max}$) and lifestyle patterns (e.g., sleep patterns), participants took part in a randomized and counterbalanced acute cross-over study with three experimental conditions: (1) regular sleep (NS; 8 h sleep window); (2) partial sleep restriction (SR; 3 h sleep window); and (3) partial sleep restriction (3 h sleep window) with morning moderate-intensity exercise (SRE). Sleep restriction was set to 3 h as this has previously been shown to impair blood glucose regulation (Saner et al. 2021). A 2-h oral glucose tolerance test (OGTT) was performed the next morning. Each of the trials

Table 1 Participant characteristics (mean ± SD)

Characteristic	Participants ($n = 11$)
Age (years)	26 ± 2
Height (m)	1.76 ± 0.07
Body mass (kg)	71 ± 12
Body mass index (kg/m^2)	22.8 ± 2.6
Total fat mass (%)	16.1 ± 4.8
Waist circumference (cm)	82 ± 9
Hip circumference (cm)	99 ± 6
Waist-to-hip ratio	0.83 ± 0.05
VO_{2max} (ml/kg/min)	48 ± 5
Fasting glucose concentrations (mmol/L)	4.9 ± 0.3
Average sleep duration (min)	490 ± 38
Sleep efficiency (%)	88 ± 2
Morningness–Eveningness questionnaire (score)	51 ± 4
The Pittsburgh Sleep Quality Index (score)	2.8 ± 0.6

were separated by a 1–3-week washout period. A schematic of the experimental protocol is shown in Fig. 1.

Preliminary assessments

Prior to taking part, participants underwent a 1-week assessment of habitual sleep patterns (duration and quality) and physical activity [24-h energy expenditure and step counts) using a commercially available wearable activity tracker (Fitbit Charge 4, Fitbit Inc., USA) (Dong et al. 2022) and a pedometer (3D TriSport, China) (Kuo et al. 2024). The former was worn on the non-dominant hand and the latter on the waist for 24 h/day except for during showering/bathing/swimming. Participants then attended the laboratory for assessment of baseline anthropometrics, including height to the nearest cm, body mass to the nearest kg (JENIX DS-102, Jen An Technology Co., Ltd), waist and hip circumference (following WHO standard operating procedures), and body composition via post-void bioimpedance

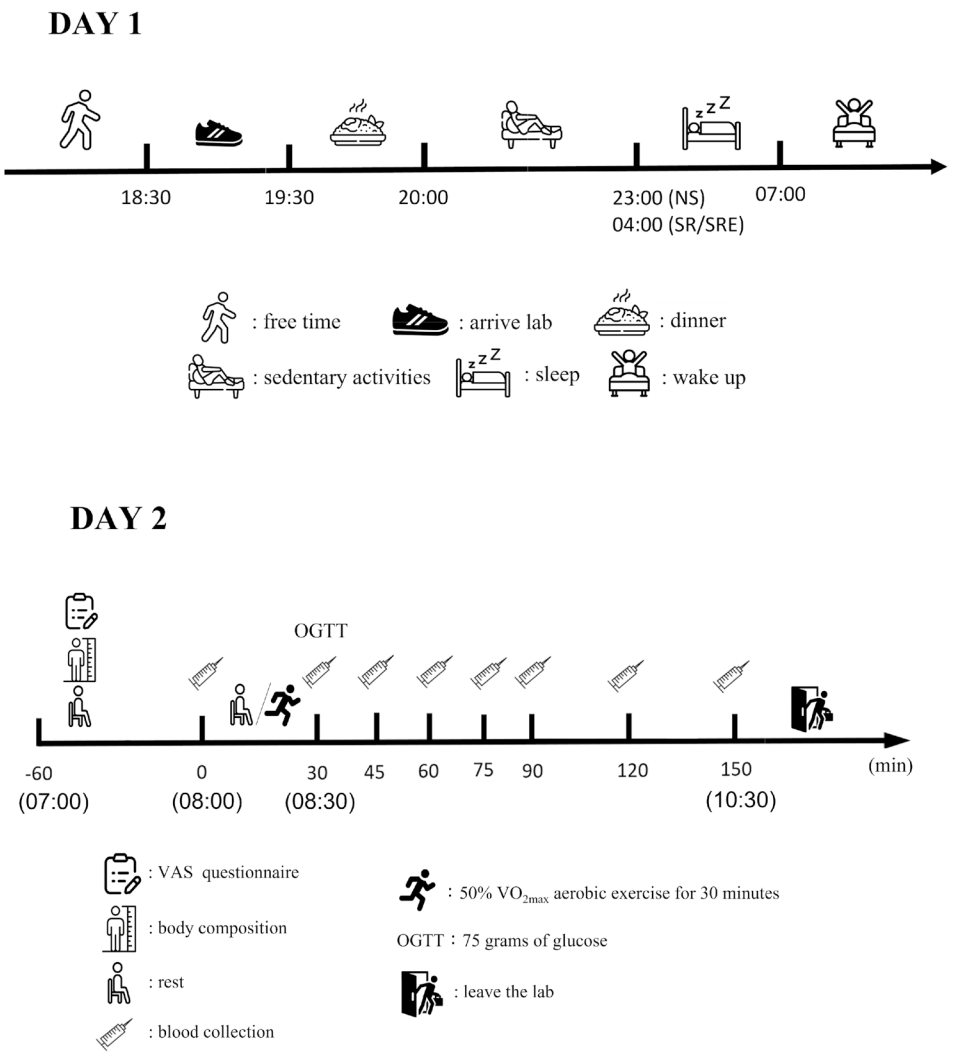

Fig. 1 Experimental procedure on day 1 (sleep manipulation with and without sleep restriction) and day 2 (exercise or sedentary followed by a 2-h 75 g OGTT)

(Inbody 720, Biospace Co. Ltd, Seoul, Korea). Participants then performed an incremental exercise test on a treadmill (h/p/cosmos mercury® med, Germany) to determine $\dot{V}O_{2max}$ and to calculate the prescribed exercise intensity for the SRE trial. The test followed the protocol adapted from Taylor et al. (1955) and involved submaximal $\dot{V}O_2$ test followed by $\dot{V}O_{2max}$ test. Expired air samples were collected at the end of each stage using Douglas bags (Hans Rudolph, Shawnee, KS) and were subsequently analyzed for relative expired fractions of oxygen and carbon dioxide (Servomex, Crowborough, UK) and total expired volume (Dry gas meter, Shinagawa Co., Ltd, Japan) to determine oxygen uptake ($\dot{V}O_2$) (Jeukendrup and Wallis 2005). Ratings of perceived exertion (RPE) and heart rate were also measured in the final minute of each stage and at the point of volitional fatigue, defined as when the participant indicated that only 1 min remained until fatigue.

Pre-experimental controls

To avoid the potential influence of the unfamiliar environment on sleep duration and quality, participants slept one night in the Sleeping Laboratory at least 7 days before the first experimental trial. Furthermore, in the 72 h before each main trial, participants were asked to sleep for at least 7 h (NS: 487 ± 28 min vs. SR: 491 ± 29 min vs. SRE: 479 ± 26 min; $p = 0.313$, confirmed by self-report) and refrain from performing any vigorous-intensity exercise. In the 48 h before each main trial, participants wore a pedometer and an activity tracker (Germini et al. 2022) to monitor daily step counts, and they were asked to try to replicate these step counts prior to subsequent trials to eliminate any confounding effect of other acute physical activity on glucose tolerance. Step counts were similar in the days prior to each trial (NS: 6350 [95% CI 5414, 7286], SR: 6510 [95% CI 5460, 7560], SRE: 6821 [95% CI 5953, 7688] steps; $p = 0.247$). Alcohol and caffeine intake were restricted for 24 h prior to each trial, and participants completed a dietary record up until 15:00 on day of the first main trial and were then asked to replicate this diet before the second and third trials.

Main experimental trial days

Each of the main experimental trials took place over 2 days (Fig. 1). On day 1, participants were under free-living conditions (i.e., self-selected breakfast and lunch, but no food allowed after 15:00 h) before 18:30 h, and they arrived at the Metabolic Health Laboratory of National Taiwan Normal University between 18:30 and 19:30 h. There were no significant differences in physical activity levels prior to attending the lab on day 1 between the trials (NS: 5950 [95% CI 4653, 7246], SR: 5429 [95% CI 4385, 6473], SRE: 5758 [95% CI 4229, 7287] steps; $p = 0.334$). Participants consumed a standardized dinner (12.25 kcal per kg of body weight with 59% carbohydrate, 13% protein and 28% fat) including sausage fried rice (I-MEI Foods Co., Ltd) and sugar free soy milk (Uni-President Enterprises Corp) over a 30 min period, before then participating in sedentary activities (e.g., reading, watching movies and/or using their laptop) for the remainder of the evening until the prescribed sleeping time. The sleeping time was set at 23:00 h in the NS trial, and 04:00 h in both SR and SRE trials. Once participants reported to the laboratory, a researcher always accompanied participants to ensure compliance with the study protocol. Participants slept in a completely dark room and wore an eye mask and earplugs to minimize any potential environmental distractions.

On day 2, participants were awoken at 07:00 h which allowed a sleeping window of 8 h for the NS trial and 3 h for the SR and SRE trials. Upon waking, anthropometric measurements (e.g., waist circumference, hip circumference, height, and body composition) and subjective assessments of sleep characteristics were collected. A cannula (Becton Dickinson, San Jose, CA, USA) was then inserted into an antecubital forearm vein and a 5-mL baseline venous blood sample was collected into tubes with ethylenediaminetetraacetic acid (EDTA) (BD Vacutainers®, Plymouth, UK). Blood samples were immediately centrifuged at 4000 rpm at 4 °C for 10 min to separate the plasma, which was then stored at –80° Celsius prior to future analyses.

At 08:00 h, participants continued sitting for further 30 min in the NS and SR trials or performed 5 min warm-up by walking at 5 km/h followed by 30 min of walking at an intensity corresponding to ~ 50% $\dot{V}O_{2max}$ in the SRE trial. During exercise, a 1-min expired air sample was collected every 10 min and the intensity was adjusted if required; meanwhile heart rate and ratings of perceived exertion were recorded at regular intervals throughout. Another blood sample was collected immediately following exercise. Thereafter, a 2-h OGTT was performed: participants consumed a drink containing 75 g glucose (Xindong-Meidayan Injection), and then, blood samples were collected at 15, 30, 45, 60, 90, and 120 min following glucose ingestion.

Biochemical analyses

Blood samples were collected for measurement of glucose using hexokinase G-6-PDH method (Beckman Coulter), insulin and cortisol using electrochemiluminescence immunoassay (ECLIA) (cobas® e immunoassay analyzers), triacylglycerols (Beckman Coulter), cholesterol extract using enzymatic reagent combined by the esterase and oxidase (Beckman Coulter), non-esterified fatty acids (NEFA) using enzymatic endpoint method on photometric systems (Diagnostic Systems), C-reactive protein (CRP) using latex

immunoturbidimetric assay (Beckman Coulter), aspartate aminotransferase (AST), and alanine aminotransferase (ALT) utilizing a modification of the methodology recommended by the International Federation of Clinical Chemistry (IFCC) (Beckman Coulter).

Statistics analysis

Descriptive data are presented in text and tables as means ± standard deviation (SD), while data on figures are presented as means with 95% confidence intervals (CIs) unless indicated otherwise. The primary outcomes in this study were the glucose and insulin response during the OGTT. To simplify the statistical analysis of a complex time series data set for these outcomes, the total area under the curve (tAUC) as a summary variable of the response was calculated using the trapezoid method (Wolever and Jenkins 1986). The tAUCs were then compared using a one-way repeated-measures ANOVA, with Greenhouse–Geisser corrections applied where intra-individual contrasts where $\varepsilon < 0.75$ and the Huynh–Feldt correction when there was less severe asphericity (Atkinson 2001). Where significant main effects were observed, post hoc comparisons between conditions were made using the *Bonferroni* correction. The same statistical analysis approach was performed for secondary outcomes. Statistical analysis was performed in SPSS version 23 (IBM, Armonk, NY, USA). To estimate the effect size, partial eta-squared (η_p^2) was calculated for significant main effects and interactions, and Cohen's d ($d = (M_2 - M_1) / SD_{\text{pooled}}$ was calculated for post hoc comparisons. Statistical significance was set at $p \leq 0.05$.

Results

Sleep and exercise characteristics

The average sleep duration was 471 ± 26 min in NS and was lower in SR (189 ± 8 min, $p < 0.01$) and in SRE (182 ± 15 min, $p < 0.01$), with no difference between SR and SRE ($p = 0.126$). Sleep efficiency tended to be higher in both SR (93 ± 2%, $p = 0.078$) and SRE (92 ± 3%, $p = 0.089$) compared to NS (89 ± 4%). Subjective perceptions of sleep adequacy were higher in the NS trial (75 ± 13) compared with the SR (17 ± 15, $p < 0.01$) and the SRE trials (13 ± 8, $p < 0.01$), with no difference between SR and SRE ($p > 0.999$).

In SRE, the average exercise intensity and total energy expenditure were 52 ± 2% $\dot{V}O_{2max}$ and 256 ± 37 kcal, respectively, with an average RER of 0.89 ± 0.04, an average heart rate of 130 ± 13 bpm, and an average RPE (Borg, 6 − 20) of 11 ± 2 (i.e., fairly light).

Glucose and insulin responses during the OGTT

The glucose and insulin responses during the OGTT are shown in Fig. 2. There was a significant main effect for glucose tAUC during the OGTT ($p < 0.001$, $\eta_p^2 = 0.489$): post hoc analysis revealed that tAUC for glucose was lower in the NS trial (924 [95% CI 865, 982] mmol/L) compared to both the SR (1012 [95% CI 945, 1080] mmol/L, $p = 0.018$, $d = 0.94$) and the SRE trials (1006 [933, 1080] mmol/L, $p = 0.002$, $d = 0.82$, Fig. 2B). There was no significant difference in glucose tAUC between SR and SRE ($p = 1.00$, $d = 0.06$, Fig. 2B). There were no significant differences in insulin tAUC between the trials ($p = 0.472$, Fig. 2D). There were no interaction effects for both the glucose and insulin responses between the trials ($p = 0.149$ & $p = 0.773$, Fig. 2A, C). A trial effect was observed only in the glucose responses ($p < 0.01$, $\eta_p^2 = 0.444$, Fig. 2A), indicating that glucose concentrations during the OGTT were lower in NS compared with both SR and SRE (both, $p < 0.01$, $d = 0.92$ & $d = 0.86$, respectively). No trial effect was observed for insulin ($p = 0.675$). Time effects were shown for both glucose and insulin responses (both, $p < 0.01$, Fig. 2A, C).

Fasting and postprandial TAGs, CHOL, ALT, and AST response

As for the lipid profiles, no differences between trials were observed for tAUCs for triacylglycerols ($p = 0.921$) and total cholesterol ($p = 0.27$). In terms of temporal patterns, an interaction effect (time × trial) and time effects were observed for plasma TAGs (both, $p < 0.01$), but no differences were observed in the *post hoc* analysis for either condition (all, $p > 0.05$, Fig. 3A). Trial effect was not observed for TAGs between trials ($p = 0.91$, Fig. 3A). Neither an interaction effect ($p = 0.562$) nor a trial effect ($p = 0.26$) were discovered for total cholesterol (Fig. 3B). Time effects were observed for total cholesterol ($p < 0.01$, Fig. 3B), showing that the concentrations of total cholesterol decreased over time.

As for the liver biomarkers, fasting and temporal changes in AST and ALT concentrations did not differ between NS, SR, and SRE (all, $p > 0.05$, Fig. 3C, D). Only ALT demonstrated a time effect ($p = 0.038$, Fig. 3C).

Fasting cortisol, CRP, and NEFA

Fasting plasma cortisol, CRP, and NEFA concentrations were not significantly different between trials ($p = 0.119$, $p = 0.666$, and $p = 0.158$, respectively; Fig. 4).

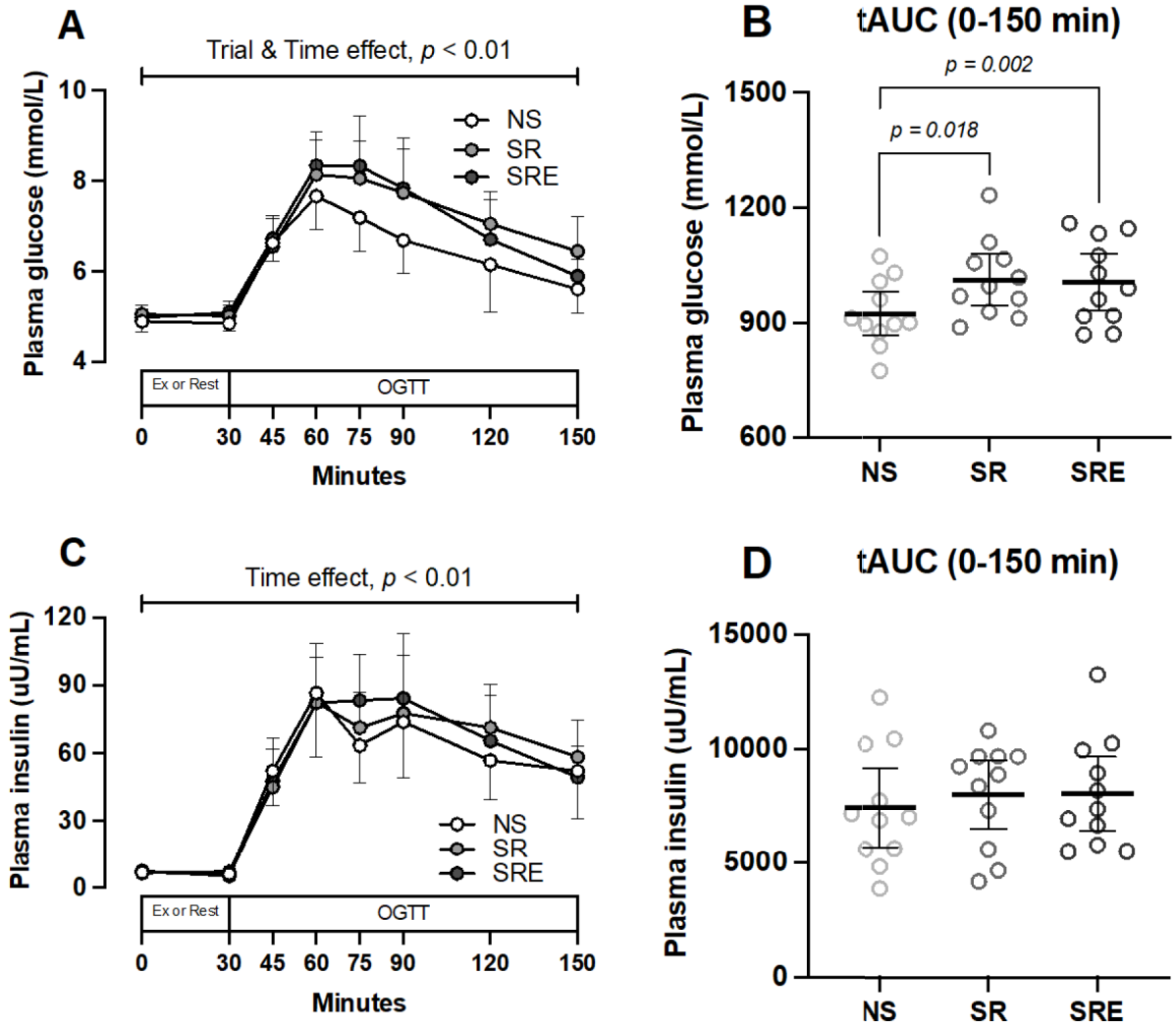

Fig. 2 Plasma glucose (**A**), insulin (**C**) concentrations in the NS, SR, and SRE trials. tAUC for the plasma glucose (**B**), insulin (**D**) between the NS, SR, and SRE trials

Discussion

The current study investigated the effects of a single night of partial sleep restriction, with and without a bout of moderate-intensity brisk walking, on the glucose and insulin response to an OGTT. The primary finding was that the postprandial glycemic response during a 2-h OGTT was higher following a single night of partial sleep restriction in healthy physically active young males and, in contrast with our hypothesis, this impaired response was not restored by performing 30 min of brisk walking immediately prior to the OGTT. In addition, our results indicated that a single night of sleep restriction has little effect on a variety of lipid (triacylglycerols, total cholesterol, and NEFA), inflammatory (cortisol and CRP), and liver (AST and ALT) health-related biomarkers.

Impaired postprandial glucose control and/or insulin sensitivity is now a well-established finding following sleep deprivation and has been observed following a 24-h period of TSR (Souza et al. 2017) and following consecutive days of PSR (Buxton et al. 2010; Saner et al. 2021). In addition, Donga et al. (2010) showed that even a single night of PSR (3.7 vs 7.5 h) resulted in an increase in hepatic glucose output (i.e., hepatic insulin resistance) and decrease in the rate of glucose disposal (i.e., peripheral insulin resistance) during a hyperinsulinemic–euglycemic clamp. Our data confirm and extend these findings by showing that the increases in hepatic and peripheral insulin resistance ultimately result in impaired whole-body glucose tolerance. We also specifically

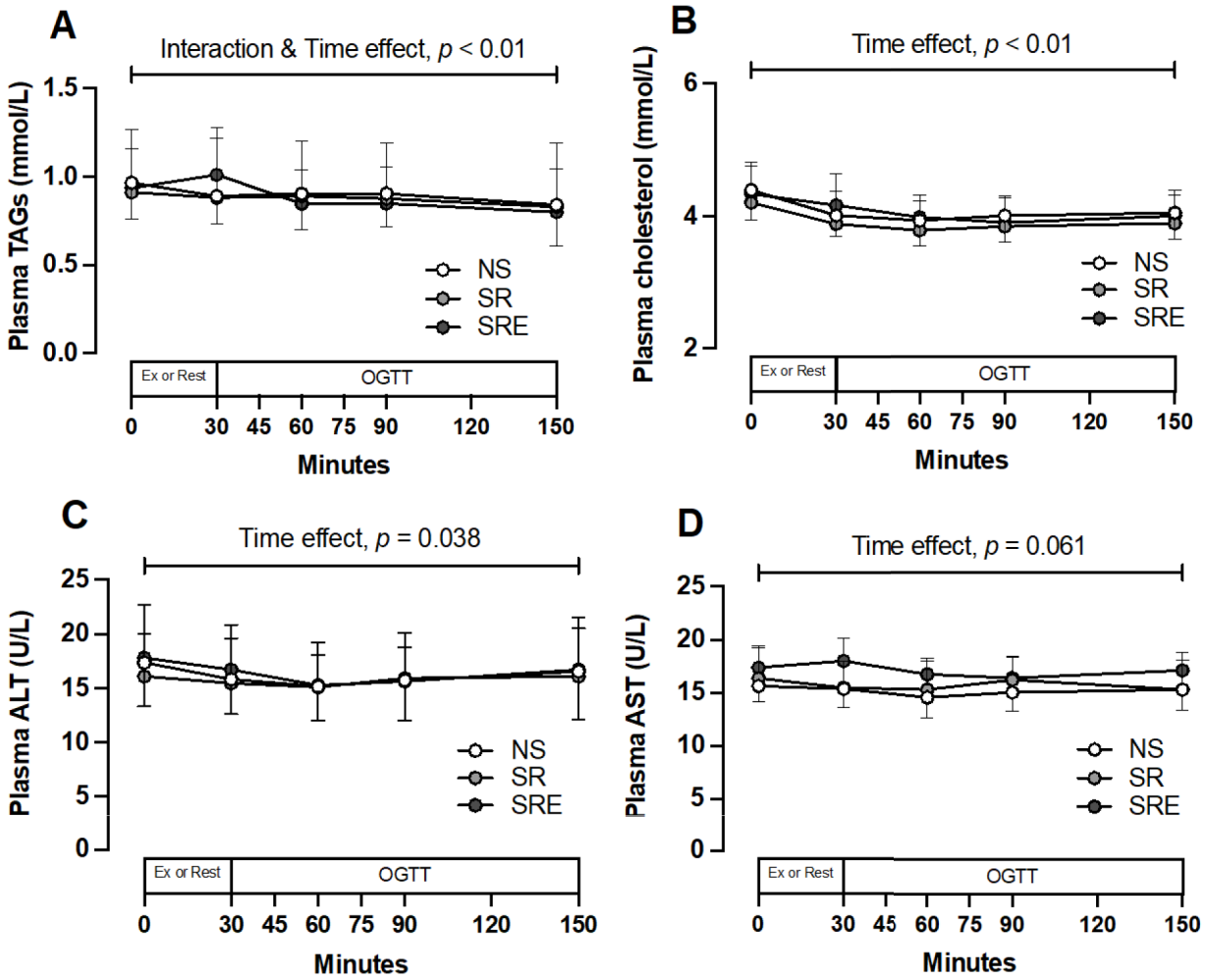

Fig. 3 Plasma triacylglycerols (**A**), cholesterol (**B**), alanine aminotransferase (ALT) (**C**), and aspartate aminotransferase (AST) (**D**) concentrations in the NS, SR, and SRE trials

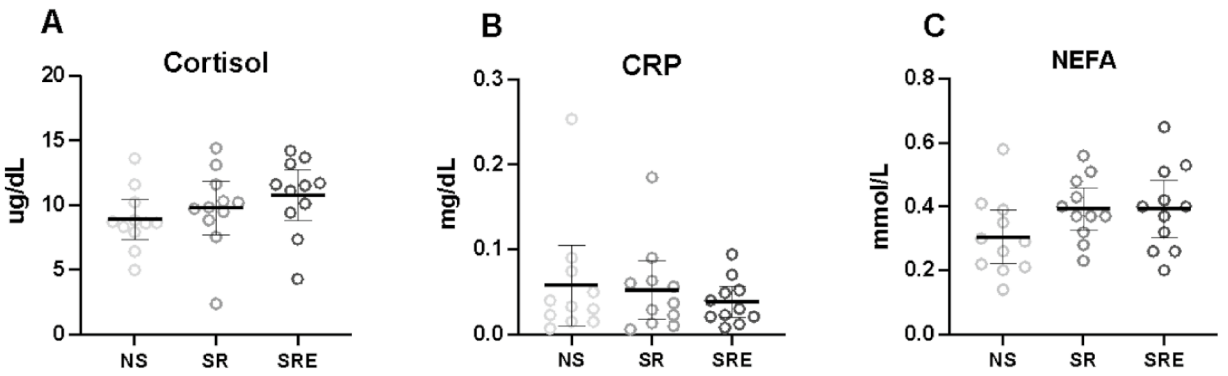

Fig. 4 Fasting cortisol (**A**), CRP (**B**), and NEFA (**C**) concentrations between the NS, SR, and SRE trials

included physically active participants with a high level of cardiorespiratory fitness and, therefore, our data indicate that a higher training and/or physical activity status does not appear to offer protection against the adverse acute effects of sleep restriction. However, this needs to be directly investigated in future work. These acute effects of sleep restriction on glucose tolerance, if repeated on a regular and consistent basis, may ultimately help to explain epidemiological data linking a lack of sleep with an increased risk of developing type 2 diabetes or cardiovascular disease (Spiegel et al. 2009; Antza et al. 2021).

The systemic metabolic response (e.g., blood glucose excursions) to a given meal is in part attributable to the interaction between various modifiable lifestyle exposures in the hours and days prior to the meal (Farah and Gill 2013; Schmid et al. 2011). As such, the negative effects of one exposure (e.g., sleep restriction) could in theory be counteracted by positive effects of other exposures (e.g., physical activity or exercise) over the same time frame (Saner et al. 2018; Maloney and Kanaley 2024). In this study, we found that a single bout of moderate-intensity walking, performed in the morning after a night of PSR and immediately prior to the OGTT, did not prevent the detrimental effects of the sleep restriction on the postprandial glucose response. This finding contrasts with several previous studies that have concluded that exercise may counteract the negative effects of sleep restriction on aspects of glucose tolerance (Saner et al. 2021; Souza et al. 2017; Porter et al. 2021). de Souza et al. (2017) found that participating in 2 weeks of HIIT prior to a 24-h TSR could reverse the impaired postprandial insulin sensitivity in healthy, active young males. Similarly, during 5 nights of PSR, Saner et al. (2021) and Porter et al. (2021) observed that 3 days of HIIT or 5 days of moderate-intensity exercise prevented any impairment in postprandial glucose regulation. One potential explanation for the different observations is that their findings reflect a combination of both the acute and chronic effects of exercise, in the context of different sleep interventions, on glucose homeostasis, rather than just the (potential) acute effects that were being investigated in the present study.

To date, the only study to apply a comparable design is Sweeney et al. (2020) who, in young healthy men, found that an acute bout of sprint interval exercise (4×30-s all-out sprints) that was performed after a single night of PSR partially protected against changes in the postprandial insulin response that were observed after sleep restriction alone. In comparison with the current data, it can be speculated that the intensity of acute exercise after PSR may be important for mitigating changes in glucose control and/or insulin sensitivity. That would make sense mechanistically given that skeletal muscle glycogen breakdown is thought to be a major driver of acute changes in glycemic control with exercise (Johnson-Bonson et al. 2021; Taylor et al. 2018) and glycogen breakdown is much greater following sprint interval exercise compared with moderate-intensity walking (Metcalfe et al. 2015; Parolin et al. 2000). However, this hypothesis will need to be directly tested in future research. Under normal sleep conditions, low-to-moderate exercise intensity has been shown to enhance glucose regulation and insulin sensitivity (Frampton et al. 2021; Hong et al. 2024). Therefore, it is tempting to conclude that the adverse effects of PSR on glucose tolerance may overshadow the beneficial effects of moderate-intensity exercise. Nonetheless, moderate-intensity aerobic exercise performed after a meal or glucose challenge (i.e., postprandial exercise) may be more effective for lowering glucose and insulin concentrations compared to when exercise is performed immediately prior to the meal (i.e., fasted exercise) (Solomon et al. 2020). In our study, participants walked on a treadmill and the exercise was performed after an overnight fast and immediately prior to the OGTT and this may, in part, explain the lack of observed effect on glucose tolerance. Additionally, since we only measured glucose and insulin concentrations during a 2-h window immediately post-exercise, it remains uncertain whether extending the measurement period to capture responses to subsequent meals would have revealed an effect of exercise on lowering postprandial glucose concentrations. Future research should investigate the effects of postprandial walking on glucose tolerance after sleep restriction and include an extended measurement period perhaps using continuous glucose monitoring.

Cortisol is the hormone associated with circadian rhythms and stress. It is released via the hypothalamic–pituitary–adrenal (HPA) axis and is known to be involved in regulating several aspects of glucose homeostasis (Kuo et al. 2015). The current study found no effect of one night of PSR on morning cortisol concentrations measured in the fasted state. Similar findings have been reported following a single night of total sleep deprivation (Souza et al. 2017). Wilms et al. (2019a) reported an altered trajectory of cortisol concentrations in the morning following a night where sleep was restricted specifically in the late phase of the night (i.e., an early wake up), but, in agreement with our findings, no effect was observed when sleep was restricted in the early phase of the night (i.e., a late bedtime). An impairment in glucose homeostasis has been reported in all these scenarios; thus, taken together, it appears that alterations in circulating cortisol are not involved in the disruption of glucose homeostasis following a single night of sleep restriction. C-reactive protein, mainly secreted by the liver, plays a direct role in acute (pro)inflammation (Sproston and Ashworth 2018) and elevated CRP levels have been shown to impair fasting and postprandial glycemia and insulinemia (D'Alessandris et al. 2007; Kato et al. 2018). In the current study, one night of PSD did not lead to elevated CRP concentrations, indicating that the impairment in postprandial glycemia is not

explained by alterations in the acute phase of inflammation in the liver. We also found that fasting and postprandial concentrations of AST and ALT, which are representative of liver function, did not differ between the partial sleep restriction and normal sleep conditions. Taken together, we found that liver function and liver-related inflammatory markers remained unchanged in the physically active males following single day of PSR in the current study.

The concentrations of NEFAs are associated with adipose tissue lipolysis, which partially reflects the metabolic processes occurring within adipose tissue. Elevation of plasma NEFAs has been observed after acute (~20%) and short-term cumulative sleep deprivation (~60%) (Donga et al. 2010; Rao et al. 2015; Souza et al. 2017; Broussard et al. 2015) and the experimental elevation of plasma NEFA via infusion has been shown to impair skeletal muscle insulin signaling and glucose metabolism (Belfort et al. 2005). In our study, fasting NEFAs' concentrations were non-significantly higher (~29% in both trials) in both the SR (0.39 [95% CI 0.33, 0.46] mmol/L, Cohen's $d = 0.79$) and SRE (0.39 [95% CI 0.3, 0.48] mmol/L, Cohen's $d = 0.68$) trials compared to the NS trial (0.31 [95% CI 0.22, 0.39] mmol/L). These observations may be of physiological relevance for the changes in glycemic control observed following sleep restriction in our study, given previously observed changes in insulin signaling and inflammation that have been observed after acute sleep restriction in both adipose tissue (Wilms et al. 2019b; Cedernaes et al. 2018; and skeletal muscle (Cedernaes et al. 2018); We observed no significant differences in fasting or postprandial triacylglycerols and cholesterol responses following a single night of PSR, indicating little change in the circulating lipid profile beyond (possible) elevations of fasting NEFA concentrations. However, it is important to note that we administered a glucose drink subsequent to the sleep intervention, which likely accounts for the lack of observed differences in triacylglycerols and cholesterol during the postprandial phase.

Despite conducting a well-controlled study with methodological rigor, there are still limitations in the current research. First, due to sex differences in sleep and circadian rhythms on metabolic control (Lok et al. 2024; Wong et al. 2015) and the potential influences of menstrual cycle phases on metabolic responses in females, we included only male participants in this study. The current findings may not be directly applicable to females. Furthermore, we aimed to investigate whether acute exercise could counteract the detrimental effects of sleep restriction; therefore, we did not include a normal sleep with exercise trial, as adding a fourth trial could complicate participant recruitment and willingness to participate in this study. Finally, the wearable device used in the current study can only detect sleep efficiency when sleep duration exceeds 3 h. We instructed participants to go to bed an additional 10 min earlier to capture sleep efficiency. This procedure was standardized in both the SR and SRE trials. Since we did not use research-grade devices to collect objective sleep data, the sleep metrics may not be as precise as those obtained from research-grade wearable devices (Dong et al. 2022). However, subjective perceptions of sleep adequacy were higher in the NS trial (75 ± 13) compared to the SR and SRE trials (17 ± 15 and 13 ± 8, respectively), with no difference between the SR and SRE trials. A research-grade device for collecting objective sleep data should be utilized in future studies.

Conclusions

Postprandial glucose tolerance was negatively impacted by a single night of partial sleep restriction in healthy and physically active young males. This impairment in glucose tolerance was not mitigated by performing 30 min of moderate-intensity walking in the morning following sleep restriction and immediately prior to the OGTT.

Acknowledgements The authors thank all the participants for their time and effort to take part in this project. The authors would like to express our gratitude to Lezen Reference Lab for their assistance in conducting the sample analysis.

Author contributions C-WH was responsible for the study design, conducted the experiments, collected the data, authored the initial draft, and revised the manuscript. C-ST performed experiments, data collection, and manuscript revision. RM contributed to critical analysis and interpretation of the data and the drafting and critical revision of the manuscript. Y-CC was responsible for the funding, study design, data analysis and interpretation, statistical analysis, first draft written, manuscript revision, and final version of the manuscript. All authors approved the final version of the manuscript.

Funding Open access funding provided by National Taiwan Normal University. This project was financially supported by the National Science and Technology Council of Taiwan (110–2410-H-003–146-MY2) and was also partially funded by the National Taiwan Normal University (NTNU) within the framework of the Higher Education Sprout Project by the Ministry of Education (MOE) in Taiwan to Y-CC. The funding body did not play a role in the design or conduct of the study.

Data availability Data generated or analyzed during this study are available from the corresponding author upon reasonable request.

Declarations

Conflict of interest The authors declare no competing interests.

References

Anothaisintawee T, Reutrakul S, Van Cauter E, Thakkinstian A (2016) Sleep disturbances compared to traditional risk factors for diabetes development: systematic review and meta-analysis. Sleep Med Rev 30:11–24. https://doi.org/10.1016/j.smrv.2015.10.002

Antza C, Kostopoulos G, Mostafa S, Nirantharakumar K, Tahrani A (2021) The links between sleep duration, obesity and type 2 diabetes mellitus. J Endocrinol 252(2):125–141. https://doi.org/10.1530/JOE-21-0155

Atkinson G (2001) Analysis of repeated measurements in physical therapy research. Phys Ther Sport 2(4):194–208

Bajpeyi S, Tanner CJ, Slentz CA, Duscha BD, McCartney JS, Hickner RC et al (2009) Effect of exercise intensity and volume on persistence of insulin sensitivity during training cessation. J Appl Physiol 106(4):1079–1085. https://doi.org/10.1152/japplphysiol.91262.2008. (**Epub 2009/02/07**)

Belfort R, Mandarino L, Kashyap S, Wirfel K, Pratipanawatr T, Berria R et al (2005) Dose-response effect of elevated plasma free fatty acid on insulin signaling. Diabetes 54(6):1640–1648. https://doi.org/10.2337/diabetes.54.6.1640

Broussard JL, Chapotot F, Abraham V, Day A, Delebecque F, Whitmore HR et al (2015) Sleep restriction increases free fatty acids in healthy men. Diabetologia 58(4):791–798. https://doi.org/10.1007/s00125-015-3500-4

Broussard JL, Wroblewski K, Kilkus JM, Tasali E (2016) Two nights of recovery sleep reverses the effects of short-term sleep restriction on diabetes risk. Diabetes Care 39(3):e40–e41. https://doi.org/10.2337/dc15-2214. (**Epub 2016/01/21**)

Buxton OM, Pavlova M, Reid EW, Wang W, Simonson DC, Adler GK (2010) Sleep restriction for 1 week reduces insulin sensitivity in healthy men. Diabetes 59(9):2126–2133. https://doi.org/10.2337/db09-0699

Buysse DJ, Reynolds CF 3rd, Monk TH, Berman SR, Kupfer DJ (1989) The Pittsburgh sleep quality index: a new instrument for psychiatric practice and research. Psychiatry Res 28(2):193–213. https://doi.org/10.1016/0165-1781(89)90047-4. (**Epub 1989/05/01**)

Cedernaes J, Schonke M, Westholm JO, Mi J, Chibalin A, Voisin S et al (2018) Acute sleep loss results in tissue-specific alterations in genome-wide DNA methylation state and metabolic fuel utilization in humans. Sci Adv 4(8):eaar8590. https://doi.org/10.1126/sciadv.aar8590

Chen YC, Betts JA, Walhin JP, Thompson D (2018) Adipose tissue responses to breaking sitting in men and women with central adiposity. Med Sci Sports Exerc 50(10):2049–2057. https://doi.org/10.1249/MSS.0000000000001654

Chen YC, Tseng CS, Hsu CW (2024) Effects of breaking up sitting on gut hormone responses and subsequent compensatory behaviors in physically inactive adults. Med Sci Sports Exerc 56(10):2049–2058. https://doi.org/10.1249/MSS.0000000000003489

Coelho J, Lucas G, Micoulaud-Franchi JA, Philip P, Boyer L, Fond G (2023) Poor sleep is associated with work environment among 10,087 French healthcare workers: results from a nationwide survey. Psychiatry Res 328:115448. https://doi.org/10.1016/j.psychres.2023.115448

D'Alessandris C, Lauro R, Presta I, Sesti G (2007) C-reactive protein induces phosphorylation of insulin receptor substrate-1 on Ser307 and Ser 612 in L6 myocytes, thereby impairing the insulin signalling pathway that promotes glucose transport. Diabetologia 50(4):840–849. https://doi.org/10.1007/s00125-006-0522-y. (**Epub 2007/02/07**)

de Souza JFT, Dattilo M, de Mello MT, Tufik S, Antunes HKM (2017) High-intensity interval training attenuates insulin resistance induced by sleep deprivation in healthy males. Front Physiol 8:992. https://doi.org/10.3389/fphys.2017.00992

Dong X, Yang S, Guo Y, Lv P, Wang M, Li Y (2022) Validation of Fitbit Charge 4 for assessing sleep in Chinese patients with chronic insomnia: a comparison against polysomnography and actigraphy. PLoS ONE 17(10):e0275287. https://doi.org/10.1371/journal.pone.0275287

Donga E, van Dijk M, van Dijk JG, Biermasz NR, Lammers GJ, van Kralingen KW et al (2010) A single night of partial sleep deprivation induces insulin resistance in multiple metabolic pathways in healthy subjects. J Clin Endocrinol Metab 95(6):2963–2968. https://doi.org/10.1210/jc.2009-2430

Farah NM, Gill JM (2013) Effects of exercise before or after meal ingestion on fat balance and postprandial metabolism in overweight men. Br J Nutr 109(12):2297–2307. https://doi.org/10.1017/S0007114512004448

Frampton J, Cobbold B, Nozdrin M, Oo HTH, Wilson H, Murphy KG et al (2021) The effect of a single bout of continuous aerobic exercise on glucose, insulin and glucagon concentrations compared to resting conditions in healthy adults: a systematic review, meta-analysis and meta-regression. Sports Med 51(9):1949–1966. https://doi.org/10.1007/s40279-021-01473-2

Germini F, Noronha N, Borg Debono V, Abraham Philip B, Pete D, Navarro T et al (2022) Accuracy and acceptability of wrist-wearable activity-tracking devices: systematic review of the literature. J Med Internet Res 24(1):e30791. https://doi.org/10.2196/30791

Hardcastle SJ, Ray H, Beale L, Hagger MS (2014) Why sprint interval training is inappropriate for a largely sedentary population. Front Psychol 5:1505. https://doi.org/10.3389/fpsyg.2014.01505

Henriksen EJ (2002) Invited review: effects of acute exercise and exercise training on insulin resistance. J Appl Physiol 93(2):788–796. https://doi.org/10.1152/japplphysiol.01219.2001

Hong JY, Li YJ, Metcalfe RS, Chen YC (2024) Effects of acute and chronic stair-climbing exercise on metabolic health: a systematic review. J Sports Sci 42(6):498–510. https://doi.org/10.1080/02640414.2024.2345414

Horne JA, Ostberg O (1976) A self-assessment questionnaire to determine morningness-eveningness in human circadian rhythms. Int J Chronobiol 4(2):97–110 (**Epub 1976/01/01**)

Houmard JA, Tanner CJ, Slentz CA, Duscha BD, McCartney JS, Kraus WE (2004) Effect of the volume and intensity of exercise training on insulin sensitivity. J Appl Physiol 96(1):101–106. https://doi.org/10.1152/japplphysiol.00707.2003. (**Epub 2003/09/16**)

Jeukendrup AE, Wallis GA (2005) Measurement of substrate oxidation during exercise by means of gas exchange measurements. Int J Sports Med 26(Suppl 1):S28-37. https://doi.org/10.1055/s-2004-830512

Johnson-Bonson DA, Narang BJ, Davies RG, Hengist A, Smith HA, Watkins JD et al (2021) Interactive effects of acute exercise and carbohydrate-energy replacement on insulin sensitivity in healthy adults. Appl Physiol Nutr Metab 46(10):1207–1215. https://doi.org/10.1139/apnm-2020-1043

Kato K, Otsuka T, Saiki Y, Kobayashi N, Nakamura T, Kon Y et al (2019) Association between elevated C-reactive protein levels and prediabetes in adults, particularly impaired glucose tolerance. Can J Diabetes 43(1):40.e2-45.e2. https://doi.org/10.1016/j.jcjd.2018.03.007. (**Epub 2018/07/22**)

Korompeli A, Chara T, Chrysoula L, Sourtzi P (2013) Sleep disturbance in nursing personnel working shifts. Nurs Forum 48(1):45–53. https://doi.org/10.1111/nuf.12005

Kuo T, McQueen A, Chen TC, Wang JC (2015) Regulation of glucose homeostasis by glucocorticoids. Adv Exp Med Biol 872:99–126. https://doi.org/10.1007/978-1-4939-2895-8_5. (**Epub 2015/07/29**)

Kuo F-C, Lin Y-T, Chueh T-Y, Chang Y-K, Hung T-M, Chen Y-C (2024) Breaking prolonged sitting increases 24-h physical activity and self-perceived energy levels but does not acutely affect cognition in healthy adults. Eur J Appl Physiol 124(2):445–455. https://doi.org/10.1007/s00421-023-05278-1

Linton SJ, Kecklund G, Franklin KA, Leissner LC, Sivertsen B, Lindberg E et al (2015) The effect of the work environment on future sleep disturbances: a systematic review. Sleep Med Rev 23:10–19. https://doi.org/10.1016/j.smrv.2014.10.010

Lok R, Qian J, Chellappa SL (2024) Sex differences in sleep, circadian rhythms, and metabolism: implications for precision medicine. Sleep Med Rev 75:101926. https://doi.org/10.1016/j.smrv.2024.101926

Maloney A, Kanaley JA (2024) Short sleep duration disrupts glucose metabolism: can exercise turn back the clock? Exerc Sport Sci Rev 52(3):77–86. https://doi.org/10.1249/JES.0000000000000339

Manders RJ, Van Dijk JW, van Loon LJ (2010) Low-intensity exercise reduces the prevalence of hyperglycemia in type 2 diabetes. Med Sci Sports Exerc 42(2):219–225. https://doi.org/10.1249/MSS.0b013e3181b3b16d. (**Epub 2009/11/21**)

Metcalfe RS, Koumanov F, Ruffino JS, Stokes KA, Holman GD, Thompson D et al (2015) Physiological and molecular responses to an acute bout of reduced-exertion high-intensity interval training (REHIT). Eur J Appl Physiol 115(11):2321–2334. https://doi.org/10.1007/s00421-015-3217-6

Munan M, Oliveira CLP, Marcotte-Chenard A, Rees JL, Prado CM, Riesco E et al (2020) Acute and chronic effects of exercise on continuous glucose monitoring outcomes in type 2 diabetes: a meta-analysis. Front Endocrinol (Lausanne) 11:495. https://doi.org/10.3389/fendo.2020.00495

Newsom SA, Everett AC, Hinko A, Horowitz JF (2013) A single session of low-intensity exercise is sufficient to enhance insulin sensitivity into the next day in obese adults. Diabetes Care 36(9):2516–2522. https://doi.org/10.2337/dc12-2606

Parolin ML, Spriet LL, Hultman E, Hollidge-Horvat MG, Jones NL, Heigenhauser GJ (2000) Regulation of glycogen phosphorylase and PDH during exercise in human skeletal muscle during hypoxia. Am J Physiol Endocrinol Metab 278(3):E522–E534. https://doi.org/10.1152/ajpendo.2000.278.3.E522

Porter JW, Pettit-Mee RJ, Emerson TS, McCrae CS, Lastra G, Vieira-Potter VJ et al (2021) Modest sleep restriction does not influence steps, physical activity intensity or glucose tolerance in obese adults. J Sleep Res 30(6):e13381. https://doi.org/10.1111/jsr.13381

Rao MN, Neylan TC, Grunfeld C, Mulligan K, Schambelan M, Schwarz JM (2015) Subchronic sleep restriction causes tissue-specific insulin resistance. J Clin Endocrinol Metab 100(4):1664–1671. https://doi.org/10.1210/jc.2014-3911

Reynolds AC, Dorrian J, Liu PY, Van Dongen HP, Wittert GA, Harmer LJ et al (2012) Impact of five nights of sleep restriction on glucose metabolism, leptin and testosterone in young adult men. PLoS ONE 7(7):e41218. https://doi.org/10.1371/journal.pone.0041218. (**Epub 2012/07/31**)

Rosa D, Terzoni S, Dellafiore F, Destrebecq A (2019) Systematic review of shift work and nurses' health. Occup Med (Lond) 69(4):237–243. https://doi.org/10.1093/occmed/kqz063

Saner NJ, Bishop DJ, Bartlett JD (2018) Is exercise a viable therapeutic intervention to mitigate mitochondrial dysfunction and insulin resistance induced by sleep loss? Sleep Med Rev 37:60–68. https://doi.org/10.1016/j.smrv.2017.01.001

Saner NJ, Lee MJ, Kuang J, Pitchford NW, Roach GD, Garnham A et al (2021) Exercise mitigates sleep-loss-induced changes in glucose tolerance, mitochondrial function, sarcoplasmic protein synthesis, and diurnal rhythms. Mol Metab 43:101110. https://doi.org/10.1016/j.molmet.2020.101110. (**Epub 20201031**)

Schmid SM, Hallschmid M, Jauch-Chara K, Wilms B, Lehnert H, Born J et al (2011) Disturbed glucoregulatory response to food intake after moderate sleep restriction. Sleep 34(3):371–377. https://doi.org/10.1093/sleep/34.3.371

Shan Z, Ma H, Xie M, Yan P, Guo Y, Bao W et al (2015) Sleep duration and risk of type 2 diabetes: a meta-analysis of prospective studies. Diabetes Care 38(3):529–537. https://doi.org/10.2337/dc14-2073

Solomon TPJ, Tarry E, Hudson CO, Fitt AI, Laye MJ (2020) Immediate post-breakfast physical activity improves interstitial postprandial glycemia: a comparison of different activity-meal timings. Pflugers Arch 472(2):271–280. https://doi.org/10.1007/s00424-019-02300-4

Spiegel K, Tasali E, Leproult R, Van Cauter E (2009) Effects of poor and short sleep on glucose metabolism and obesity risk. Nat Rev Endocrinol 5(5):253–261. https://doi.org/10.1038/nrendo.2009.23

Sproston NR, Ashworth JJ (2018) Role of C-reactive protein at sites of inflammation and infection. Front Immunol 9:754. https://doi.org/10.3389/fimmu.2018.00754

Stevens J, Truesdale KP, McClain JE, Cai J (2006) The definition of weight maintenance. Int J Obes (Lond) 30(3):391–399. https://doi.org/10.1038/sj.ijo.0803175

Strain T, Kelly P, Gibb R, Allison M, Mutrie N, Murphy M (2024) Is Scotland walking in the right direction? A cross-sectional analysis of trends in walking by socioeconomic status. J Phys Act Health 21(7):707–716. https://doi.org/10.1123/jpah.2023-0635

Sweeney EL, Jeromson S, Hamilton DL, Brooks NE, Walshe IH (2017) Skeletal muscle insulin signaling and whole-body glucose metabolism following acute sleep restriction in healthy males. Physiol Rep. https://doi.org/10.14814/phy2.13498

Sweeney EL, Peart DJ, Kyza I, Harkes T, Ellis JG, Walshe IH (2020) Impaired insulin profiles following a single night of sleep restriction: the impact of acute sprint interval exercise. Int J Sport Nutr Exerc Metab 30(2):139–144. https://doi.org/10.1123/ijsnem.2019-0235. (**Epub 2019/12/08**)

Taylor HL, Buskirk E, Henschel A (1955) Maximal oxygen intake as an objective measure of cardio-respiratory performance. J Appl Physiol 8(1):73–80. https://doi.org/10.1152/jappl.1955.8.1.73

Taylor HL, Wu CL, Chen YC, Wang PG, Gonzalez JT, Betts JA (2018) Post-exercise carbohydrate-energy replacement attenuates insulin sensitivity and glucose tolerance the following morning in healthy adults. Nutrients. https://doi.org/10.3390/nu10020123

VanHelder T, Symons JD, Radomski MW (1993) Effects of sleep deprivation and exercise on glucose tolerance. Aviat Space Environ Med 64(6):487–492

Wang S, Rossheim ME, Nandy RR (2023) Trends in prevalence of short sleep duration and trouble sleeping among US adults, 2005–2018. Sleep. https://doi.org/10.1093/sleep/zsac231

Watson NF, Badr MS, Belenky G, Bliwise DL, Buxton OM, Buysse D et al (2015) Recommended amount of sleep for a healthy adult: a joint consensus statement of the american academy of sleep medicine and sleep research society. Sleep 38(6):843–844. https://doi.org/10.5665/sleep.4716. (**Epub 20150601**)

Wilms B, Chamorro R, Hallschmid M, Trost D, Forck N, Schultes B et al (2019a) Timing modulates the effect of sleep loss on glucose homeostasis. J Clin Endocrinol Metab 104(7):2801–2808. https://doi.org/10.1210/jc.2018-02636

Wilms B, Leineweber EM, Molle M, Chamorro R, Pommerenke C, Salinas-Riester G et al (2019b) Sleep loss disrupts morning-to-evening differences in human white adipose tissue transcriptome.

J Clin Endocrinol Metab 104(5):1687–1696. https://doi.org/10.1210/jc.2018-01663

Wolever TM, Jenkins DJ (1986) The use of the glycemic index in predicting the blood glucose response to mixed meals. Am J Clin Nutr 43(1):167–172

Wong PM, Manuck SB, DiNardo MM, Korytkowski M, Muldoon MF (2015) Shorter sleep duration is associated with decreased insulin sensitivity in healthy white men. Sleep 38(2):223–231. https://doi.org/10.5665/sleep.4402