Impaired postprandial glucose and no improvement in other cardiometabolic responses or cognitive function by breaking up sitting with bodyweight resistance exercises: a randomised crossover trial

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Abstract

Purpose: The effects of breaking up sitting with resistance exercise on cardiometabolic health and cognitive function in young healthy adults is unknown. This study evaluated the acute effects of breaking up sitting with bodyweight resistance exercise on postprandial glucose, lipids, blood pressure and cognitive function. **Methods:** A randomised crossover design was used. Twelve normal-weight participants aged 25±6 years took part in two, 5 h conditions: (1) uninterrupted sitting (SIT), and (2) sitting with 3 min of bodyweight resistance exercise breaks every 30 min (REX). Dietary intake was standardised across conditions. Linear mixed models were used to compare outcomes between conditions. **Results:** Postprandial glucose was significantly higher in the REX condition than in SIT (incremental area under the curve 346.3 [95% confidence interval: 233.9, 458.7] and 256.9 [144.4, 369.3] mmol/L·5 h, respectively, p=0.045). Blood pressure, lipids and cognitive function outcomes were not different between conditions (p≥0.05). **Conclusion:** This study suggests that breaking up sitting with bodyweight resistance exercise does not benefit cardiometabolic health or cognitive function acutely in young healthy adults. The longer-term effects of breaking up sitting with resistance exercise warrants investigation to appropriately inform public health guidelines.

Keywords: sedentary time; sitting; resistance exercise; cardiometabolic risk; cognitive function

Introduction

Sedentary behaviour is defined as any waking behaviour with a low energy expenditure while in a sitting or reclined posture (Tremblay et al. 2017). A high amount of sedentary time is associated with an increased risk of cardiovascular disease, Type 2 diabetes and premature mortality (Bailey et al. 2019a; Wilmot et al. 2012). These associations are largely independent of physical activity, although engaging in ≥60-75 min/day of moderate-intensity physical activity may offer protection against these health outcomes (Ekelund et al. 2016). Prolonged bouts of sitting can increase postprandial glucose and triglyceride levels, and lead to deteriorations in insulin action (Dempsey et al. 2018; Stephens et al. 2011). Research has demonstrated that breaking up sitting with short, regular bouts of light or moderate-intensity activity (e.g. 2-5 min every 20-30 min) can acutely attenuate postprandial glucose levels (Saunders et al. 2018). However, there is conflicting evidence that suggests breaking up sitting may have negligible cardiometabolic effects (Altenburg et al. 2013), regardless of the frequency of the activity breaks (Thorsen et al. 2019). The effects of breaking up sitting on postprandial triglycerides are also inconsistent (Saunders et al. 2018). In adults with Type 2 diabetes, breaking up sitting with 3 min of simple resistance exercises every 30 min significantly reduced postprandial glucose, triglycerides and resting blood pressure over a single day (Dempsey et al. 2016a; Dempsey et al. 2016b). However, hourly resistance exercise breaks did not improve glucose, triglycerides or blood pressure levels in individuals with increased cardiometabolic risk (Kowalsky et al. 2019). This may have been due to the exercise breaks not being frequent enough. The effects of breaking up sitting with more frequent simple resistance exercises should thus be evaluated in healthy adults to inform strategies for the prevention of cardiometabolic abnormalities.

Breaking up prolonged sitting could protect against cognitive decline via reductions in glycaemic variability (Wheeler et al. 2017). Poor glycaemic control is implicated in the impairment of brain structure and cognitive function, which could lead to neurodegenerative conditions like dementia (Geijselaers et al. 2015). The acute decline in cerebral blood flow

from prolonged sitting is prevented when sitting is interrupted with 2 min of light-intensity walking every 30 min (Carter et al. 2018). Breaking up sitting could thus prevent acute declines in cognitive function as a result of maintained cerebral blood flow and reduced postprandial hyperglycaemia. Improvements in cognitive function were observed when sitting was interrupted with 3 min of moderate-intensity walking every 30 min (Chrismas et al. 2019a), whereas breaking up sitting with hourly, 5 min moderate-intensity walking bouts or 3 min light-intensity walking bouts every 30 min did not affect cognition (Bergouignan et al. 2016; Wennberg et al. 2016). The effects of breaking up sitting with resistance exercise on cognition have not yet been evaluated.

The aims of this study, therefore, were to evaluate the acute effects of breaking up prolonged sitting with bodyweight resistance exercises on postprandial glucose (primary aim), lipids, blood pressure and cognitive function (secondary aims) in healthy adults. It was hypothesised that breaking up sitting with bodyweight resistance exercises would lead to acute improvements in cardiometabolic health and cognitive function compared with prolonged sitting.

Methods

Study Overview

This two-way randomised crossover trial received ethical approval from the University of Bedfordshire School of Sport Science and Physical Activity Research Ethics Committee (approval number 2017SSPA001). Participants provided written informed consent prior to taking part in the study. All testing procedures were undertaken at the University of Bedfordshire Sport and Exercise Science Laboratories. Following a preliminary testing session, participants completed two experimental conditions that were each 5 h in duration: (1) uninterrupted sitting, and (2) sitting with 3 min of bodyweight resistance exercise breaks every 30 min. The order of the experimental conditions was randomly determined by the research team using an online tool (www.randomizer.org) applying a simple randomisation method.

Participants

Males and females aged 18-50 years who self-reported sitting for >7 h/day and were non-obese (i.e. waist circumference <88 and <102 cm for males and females, respectively) were eligible to take part. Exclusion criteria included the presence of a known blood-borne disease, participation in >300 min/week of moderate-to-vigorous physical activity (MVPA), self-reported diabetes, using glucose or lipid medication, pregnancy, current or recent smoker, and any contraindications to physical activity. Sitting time and MVPA were determined using the short form International Physical Activity Questionnaire (Craig et al. 2003).

Preliminary measures and familiarisation session

Prior to experimental conditions, participants were required to attend a session to complete preliminary measures and familiarisation with the study protocols. Stature was measured to the nearest 0.01 m using a stadiometer (Holtain Ltd., Crymych, UK) and weight to the nearest 0.1 kg using electronic weighing scales (Tanita BWB-800S digital scales; Tanita Corp., Tokyo, Japan). Participants then practiced the bodyweight resistance exercises after viewing an

explanation and demonstration on a video until the research team were satisfied that each participant could safely perform the proper technique for each exercise. Following this, they were familiarised with the Borg Rating of Perceived Exertion (RPE) scale (Borg 1982). After an explanation of the cognitive tests, participants completed the full cognitive test protocol once (as described below) to minimise any practice effect (Hope et al. 1998).

Experimental protocol

The experimental protocol is shown in Figure 1. Conditions were separated by a washout of ≥7 days to avoid any carryover effects of physical activity on insulin sensitivity (Mikines et al. 1988). The 5 h experimental conditions were:

(1) Uninterrupted sitting (SIT): participants were asked to minimise excessive movement while remaining seated at a desk.

(2) Resistance exercise breaks (REX): sitting was interrupted with 3 min of bodyweight resistance exercises every 30 min. At the start of the condition, participants were re-familiarised with each exercise by viewing a video containing explanations and demonstrations. During the exercise breaks, participants completed two sets of the following exercises with each set lasting 20 s: half-squats, upright wall push-ups, knee raises and calf raises. This equated to 160 s of exercise with the remaining 20 s of the break allowing for transition time between sets. There was no instruction provided regarding the rate or number of repetitions to be completed. To enhance external validity and practicality of the exercise breaks, participants were advised to complete the repetitions at a rate comfortable to them while following the proper technique. At the end of each 3 min break, RPE was recorded.

Participants were required to avoid exercise for 48 h and refrain from consuming alcohol or caffeine for 24 h before each condition. They were also asked to record their dietary intake for 24 h prior to their first condition and replicate the exact timing and volume of dietary

intake for the 24 h prior to their second condition. Travel to the laboratories in motorised transport was requested to minimise physical activity levels prior to the conditions.

Upon arrival to the laboratory, participants were seated for ≥10 min, after which resting blood pressure was measured. Following this, a fasting blood sample was obtained. Participants then completed the cognitive function tests in a silent environment. After this, a standardised breakfast meal was consumed and the 5 h experimental period then commenced. At 2.5 h, participants consumed a lunchtime snack. At the end of each condition, participants immediately completed another set of cognitive function tests. Participants were supervised by a researcher throughout each condition to ensure compliance with the protocols and they read or used a laptop computer during the sitting periods. A researcher pushed participants in a wheelchair to transport them to the food consumption area and toilets when required.

Standardisation of dietary intake

Dietary intake was standardised across conditions. The breakfast meal consisted of cornflakes, whole milk, and butter croissant with a macronutrient composition of 57% carbohydrate, 31% fat and 12% protein. The lunchtime snack consisted of ready salted crisps, low fat yoghurt and milk chocolate with a macronutrient composition of 51% carbohydrate, 39% fat, and 9% protein. The breakfast and lunchtime snack meals provided participants with 30% and 20%, respectively, of their estimated daily energy requirements. This was estimated based on prediction equations (Mifflin et al. 1990) with an activity factor of 1.4 applied to represent a sedentary day. Participants were required to consume each meal within a maximum of 15 min. Consumption times were recorded in the first condition and participants were asked to replicate these consumption times in the second condition. Water was consumed ad libitum throughout the 5 h postprandial period during the first condition. This volume was replicated in the second condition via the provision of two equal volumes of water to be consumed between 0 h to 2.5 h and 2.5 to 5 h.

Cognitive function tests

Cognitive function tests were completed using the Psychomotor Evaluation computer programme (Hope et al. 1998). A set of three tests were used to assess cognition in the following order: numeric vigilance, discrete simple reaction time and probed memory. All participants performed the tests using the same computer with a 1 min rest between each test. For the numeric vigilance test, 3-digit numbers flashed on a screen 80 times per min for a duration of 4 min. Participants needed to press the spacebar when a 3-digit number was duplicated. The number of correct, missed and false responses were recorded. The reaction time test consisted of the participant holding down the spacebar with an index finger and then releasing it as quickly as possible to press one of the target keys (numeric keys 4-9) after a small 'sun' icon appears at a random location on the screen. The reaction time test consisted of 20 stimuli and thinking time and movement time were recorded with an exactitude of 1 ms. The probed memory test consisted of the participant being presented with a sequence of eight consonants. Each new consonant was added every second. Once the full list of eight was presented for 1 s, the full sequence of letters was blanked out. A random probe letter is then presented and the participant is required to indicate the letter that immediately preceded the probe letter in the sequence. A list of 20 presentations was used and the percentage of correct responses was recorded.

Blood collection and biochemistry

Capillary blood samples were collected using a finger-prick method to obtain valid measures of glucose and lipid concentrations (Kruijshoop et al. 2004; Rubin et al. 2003). The first sample was taken in a fasted state at baseline with subsequent samples at 60, 120, 180, 240, and 300 min during each condition. Samples were taken immediately prior to the activity bouts during the REX condition. The hand was placed in warm water for 2-3 min prior to the sample being taken to encourage capillary perfusion. The fingertip was pricked using a lancet and the first drop of blood was discarded. The whole finger was gently squeezed to encourage bleeding with samples being collected into two, 300 µL EDTA prepared microvette capillary

198 tubes. Approximately 500 µL of blood was collected at each time point. Blood glucose and
199 triglyceride concentrations were immediately analysed using the YSI 2300 STAT Plus glucose
200 and lactate analyzer (YSI Inc., Yellow Springs, Ohio, USA) and the Reflotron Plus system
201 (Roche Diagnostics, Hoffmann-La Roche Ltd., Burgess Hill, UK), respectively. Following this,
202 the remaining blood was centrifuged for 5 min at 2000 x g. High-density lipoprotein cholesterol
203 (HDL-C) was then measured from plasma using the Reflotron Plus system. For triglyceride
204 and HDL-C measurements, whole blood and plasma, respectively, was pipetted onto a
205 Reflotron test strip that was inserted into the Reflotron Plus system for analysis. The YSI
206 analyzer and Reflotron Plus system are considered valid and reliable methods for the
207 measurement of glucose and lipids, respectively. The YSI has a between-batch coefficient of
208 variation (CV) of 1.7% to 5.1% and a bias of −1.7% compared with wet chemistry methods
209 (Johnson and Baker 2001). The Reflotron Plus has an 8.3% day-to-day CV and a 0.97
210 correlation coefficient for triglycerides concentrations compared with wet chemistry analysis
211 (Rohac and Gabl 1987). For HDL-C, within-day CV is 3.4-4.0% and a bias of -1.3% compared
212 with a reference method (Ng et al. 1991).
213
214 ***Blood pressure***
215 All blood pressure measurements were taken on the right arm while the participant was seated
216 using an Omron M6 AC automatic blood pressure monitor (Omron Healthcare Co. Ltd.,
217 Matsusaka, Japan). Baseline blood pressure was measured twice with a 2 min rest between
218 measures. Single measures were then taken at 0.5 h and every 30 min thereafter. Readings
219 were taken immediately prior to the activity bouts during the REX condition.
220
221 ***Sample size***
222 Sample size calculations were conducted using GPower (Faul et al. 2007). Incremental area
223 under the glucose curve was the primary outcome. To detect an effect size of d=1.12 at 90%
224 power with a correlation among repeated measures of 0.5 and an alpha level of 0.05, 11

participants were required for the study. These calculations were based on data from previous studies in healthy participants (Bailey and Locke 2015; Peddie et al. 2013).

Data analysis

Total area under the curve (tAUC) for glucose, triglycerides and HDL-C was calculated using the trapezoidal method for each 5 h condition. Net incremental area under the curve (iAUC) was then calculated by subtracting the area under the baseline concentration from tAUC. Mean arterial pressure (MAP) was calculated using the equation:

$$MAP \cong P_{Dias} + \frac{1}{3}(P_{Systolic\ blood\ pressre} - P_{Diastolic\ blood\ pressure}).$$

Blood pressure values were averaged over the course of each 5 h condition period. Statistical analyses were conducted with SPSS v26 (IBM, Armonk, New York, USA). Q-Q plots were used to assess normality of the variables, which were each deemed plausible. Linear mixed models were used to analyse differences in the outcomes between conditions. Condition and baseline values (as covariates for the cardiometabolic outcome analyses) were entered as fixed factors and participants entered as a random factor. For cognitive function outcomes, time was additionally entered as a fixed factor. Data is presented as mean (95% confidence interval) unless stated otherwise. The level of significance was set as $p<0.05$.

Results

Participants were recruited between December 2017 and March 2018. The flow of participants throughout the study is shown in Figure 2. Thirteen participants were randomised after screening with 12 participants completing both conditions. Descriptive characteristics of the sample can be seen in Table 1. Baseline values were 4.09 (3.87, 4.33) and 4.11 (3.88, 4.34) mmol/L for glucose, 1.00 (0.85, 1.14) and 1.01 (0.87, 1.16) mmol/L for triglycerides, 1.24 (1.05, 1.43) and 1.26 (1.07, 1.45) mmol/L for HDL-C, and 86 (81, 92) and 85 (79, 91) mmHg for MAP in the SIT and REX conditions, respectively. The RPE of the resistance exercise breaks was 7.9±1.4, which corresponds to a perceived exertion of “very light”. During the experimental conditions, participants consumed 94±12 g of carbohydrate, 23±3 g of fat, 19±3 g of protein and 2778±356 kJ at the breakfast meal and 57±7 g of carbohydrate, 19±2 g of fat, 11±1 g of protein and 1853±238 kJ at the lunch meal.

Glucose, triglyceride and HDL-C responses over time for each condition are shown in Figure 3. Glucose iAUC (346.3 [233.9, 458.7] and 256.9 [144.4, 369.3] mmol/L·5 h for the REX and SIT conditions, respectively, p=0.045, medium effect d=0.42) and tAUC (1577.7 [1465.3, 1690.1] and 1488.2 [1375.8, 1600.7] mmol/L·5 h, respectively, p=0.045; medium effect d=0.42) were significantly higher in REX than SIT. Postprandial triglyceride and HDL-C concentrations did not differ significantly between conditions. Triglyceride iAUC for the REX and SIT conditions was 56.5 (14.2, 98.7) and 69.0 (26.8, 111.2) mmol/L·5 h, respectively (p=0.537, small effect d=0.16). For triglyceride tAUC, concentrations were 357.9 (315.7, 400.2) and 370.5 (328.3, 412.7) mmol/L·5 h, respectively (p=0.537, small effect d=0.16) for the REX and SIT conditions. During the REX and SIT conditions, HDL-C iAUC was -7.9 (-32.1, 16.3) and -30.3 (-54.5, -6.1) mmol/L·5 h, respectively (p=0.117, medium effect d=0.49), whereas tAUC was 366.4 (342.2, 390.6) and 344.0 (319.8, 368.2) mmol/L·5 h, respectively (p=0.117, medium effect d=0.49). MAP did not differ significantly between the REX (86 [83, 88] mmHg) and SIT (85 [82, 87] mmHg, p=0.537, small effect d=0.21) conditions.

Cognitive function outcomes did not differ significantly between conditions with trivial to small effect sizes for each between-condition comparison. The number of correct (23.7 [21.1, 26.2] and 24.1 [21.5, 26.6] for the SIT and REX conditions, respectively, p=0.179; d=0.08), missed (10.5 [7.7, 13.2] and 9.9 [7.2, 12.6], p=0.496; d=0.12) and false responses (4.3 [2.2, 6.3] and 4.9 [2.8, 7.0], p=0.475; d=0.15) did not differ significantly between conditions. During SIT and REX, probed memory scores were 49.7 (38.0, 61.3) and 45.0 (33.4, 56.6), respectively (p=0.261; d=0.21). In the simple reaction time test, reaction thinking time was 477.1 (400.5, 553.7) and 505.0 (428.4, 581.6) ms, respectively, in the SIT and REX conditions (p=0.431; d=0.19). Reaction movement time was 196.6 (150.7, 242.6) ms in SIT and 185.0 (139.0, 231.0) ms in REX (p=0.378; d=0.13). The main effect of time (all p>0.144) and the condition x time interaction (all p>0.178) was not significant for any of the cognitive function outcomes (see Table 2).

Discussion

This randomised crossover trial in young generally healthy adults found no acute benefit of breaking up sitting with bodyweight resistance exercise on cardiometabolic risk markers. In contrary to the study hypothesis, postprandial glucose was higher during the resistance breaks condition than uninterrupted sitting. The literature is conflicting in terms of the acute benefits of breaking up sitting on glucose. A number of studies have reported reduced postprandial glucose levels in response to breaking up sitting with standing, walking and cycling (Bailey and Locke 2015; Bailey et al. 2019b; Dempsey et al. 2016a; Henson et al. 2016), whereas other studies have reported no change (Altenburg et al. 2013; Bailey and Locke 2015; Maylor et al. 2019). In studies using resistance based exercise to break up sitting, like in the current study, glucose was unaffected in overweight participants (Hawari et al. 2019) and in individuals with increased cardiometabolic risk (Kowalsky et al. 2019). However, in adults with Type 2 diabetes, postprandial glucose levels were significantly reduced with 3 min of bodyweight resistance exercise performed every 30 min (Dempsey et al. 2016a). Individuals with more advanced metabolic dysfunction may thus benefit more from breaks in sitting due to a greater capacity for improvement. In studies that replaced sitting with standing, 15 min of continuous standing every 30 min and 10 standing bouts (each 1.5 min in duration) over a 30 min period did not significantly affect postprandial glucose compared with prolonged sitting (Hawari et al. 2016). Similar to the present study, 2 h of continuous standing desk work led to a significant increase in postprandial glucose (Gao et al. 2017). It was theorised that the increase in glucose could have been due to shifts in energy substrate utilisation from carbohydrate to fat oxidation. It is not possible to make direct comparisons to the present study as substrate utilisation was not measured and the type, duration and frequency of activity was different. The efficacy of resistance exercise to break up sitting requires further investigation across different population groups due to the limited and conflicting research in this area.

312 The reason that the resistance exercise breaks led to higher postprandial glucose in the
313 present study is not clear. It could be postulated that there was an increased glucose output
314 to ensure that sufficient energy was available for exercise (Holmstrup et al. 2014). Increased
315 hepatic gluconeogenesis, hepatic glycogenolysis and postprandial glucose concentrations
316 can occur in response to longer durations (60-120 min) of continuous light or moderate-
317 intensity exercise (Knudsen et al. 2014; Trimmer et al. 2002). Higher glucose levels have
318 also been reported in response to a high-intensity resistance exercise session in strength
319 trained athletes (Tesch et al. 1986). This was attributed to greater intra-muscular glycogen
320 breakdown to meet the exercise demands as opposed to blood glucose uptake. These
321 findings may have limited application in the current study in which the resistance exercise
322 bouts were of a lower intensity and spread across the day in shorter bouts. There could also
323 be a reduced carbohydrate and/or increased fat oxidation in response to resistance exercise
324 (Binzen et al. 2001). The mechanisms underpinning glucose responses to breaking up
325 sitting with resistance exercise should thus be explored.
326
327 Resistance exercise breaks did not significantly affect lipid or blood pressure in the current
328 study. This was also the case when participants with increased cardiometabolic risk
329 engaged in hourly resistance exercise breaks (Kowalsky et al. 2019) and when healthy,
330 overweight/obese, and dysglycaemic individuals interrupted their sitting with short bouts of
331 standing, walking, or chair squats every 20-30 min (Bailey and Locke 2015; Hawari et al.
332 2019; Henson et al. 2016; Maylor et al. 2019). The effects of physical activity breaks on
333 postprandial triglycerides may not be realised until 8-22 h later; this is the time it takes
334 lipoprotein lipase activity to peak after a continuous bout of moderate-intensity exercise
335 (Greiwe et al. 2000). However, reductions in triglycerides and increases in HDL-C have been
336 seen over 8 h in response to breaking up sitting with simple resistance exercise and light-
337 intensity walking in individuals with Type 2 diabetes (Dempsey et al. 2016a) and high-
338 intensity treadmill exercise in healthy sedentary participants (Maylor et al. 2018b). Blood
339 pressure was also beneficially affected by resistance exercise breaks in individuals with

Type 2 diabetes (Dempsey et al. 2016b). Lipid and blood pressure levels may thus be improved over a single day if the activity breaks are of a high intensity or in participants with adverse cardiometabolic health. Nonetheless, a medium effect was seen for HDL-C in the present study with higher concentrations in the resistance exercise breaks condition. This could suggest potential efficacy of resistance exercise breaks for lipid metabolism, but this requires investigation within a study powered to detect changes in HDL-C.

The present study provides novel evidence that breaking up sitting with resistance exercise does not affect cognitive function. Similar to the present findings, breaking up sitting with 3 min of light-intensity walking every 30 min (Wennberg et al. 2016) or 5 min of moderate-intensity walking every 60 min did not affect cognitive function (Bergouignan et al. 2016). In contrast, cognitive function was improved in response to 3 min of moderate-intensity walking every 30 min (Chrismas et al. 2019a). Cognition-related physiological responses that may be elicited through higher intensity physical activity (e.g. increased brain-derived neurotrophic factor, heart rate and endorphins) may not occur as a result of light-intensity exercise (Chang et al. 2012). This may explain why beneficial responses were observed by Chrismas et al. (2019a) but not in studies using light-intensity activity breaks. Further research is thus needed to evaluate the effects of higher intensity resistance exercise breaks.

Multicomponent workplace interventions incorporating height-adjustable workstations have effectively reduced workplace and daily sitting by 45-83 min/day and prolonged sitting by 45 min/shift after 12 months (Edwardson et al. 2018; Healy et al. 2016). These changes led to improvements in fasting glucose and overall cardiometabolic health (Healy et al. 2017). However, the number of breaks in sitting was not reported in these studies and sitting was replaced primarily with standing as opposed to physical activity or resistance exercises. Another workplace intervention reduced prolonged sitting by 39 min and increased the number of breaks from sitting (7.8 breaks) and stepping time (12 min) per 8 h work shift after eight weeks (Maylor et al. 2018a). This led to an improvement in waist circumference but not

368 in other cardiometabolic risk markers. These studies demonstrate the potential of workplace
369 interventions for reducing sitting and increasing breaks from sitting over the longer term. The
370 cardiometabolic and cognitive effects of breaking up sitting with resistance exercise,
371 however, should be evaluated in such studies to extend upon the findings of the acute study
372 herein.
373 The randomised crossover design, controlled laboratory environment and standardisation of
374 dietary intake before and during the experimental conditions are strengths of this study. The
375 use of an objective measure of cognitive function is a further strength. However, the study
376 was not powered to detect differences in cognitive function outcomes, which should be
377 addressed in future research. Insulin levels were also not measured. Several studies in
378 which glucose levels were unaffected in response to breaking up sitting have reported a
379 reduction in postprandial insulin (Chrismas et al. 2019b; Hawari et al. 2019; Maylor et al.
380 2019). It cannot be disregarded that improvements in insulin levels could have occurred in
381 the present study, despite postprandial glucose not being beneficially affected by resistance
382 exercise breaks. However, other studies have also reported that both glucose and insulin
383 were unaffected by breaks in sitting (Hawari et al. 2016; Thorsen et al. 2019). Differences in
384 work rates during resistance exercise may have the potential to influence hepatic glucose
385 production via changes in adrenaline (Kraemer and Ratamess 2005). The work rate or
386 number of repetitions of the exercise breaks was not evaluated in the present study and
387 should be considered in future research. Moreover, sedentary individuals may engage in a
388 greater volume of activity per hour than in the resistance exercise breaks condition in the
389 present study, which may limit the external validity of the findings. However, the number of
390 activity breaks across the 5 h condition is relatively similar to the number of breaks reported
391 in office workers (Keown et al. 2018; Maylor et al. 2018a). Furthermore, sedentary
392 individuals typically engage in multiple bouts of prolonged sitting across the day (Keown et
393 al. 2018; Maylor et al. 2018a). The findings of breaking up prolonged sitting in this study are
394 thus relevant for informing public health guidelines.
395

In conclusion, the findings of this study suggest that breaking up prolonged sitting with bodyweight resistance exercise does not significantly improve postprandial cardiometabolic health or cognitive function acutely in young healthy adults. Instead, resistance exercise breaks may increase glucose levels compared with prolonged sitting. The effects of breaking up sitting with resistance exercise on cardiometabolic health and cognitive function in the longer term should be investigated to elucidate the relevance of this type of sedentary behaviour reduction strategy for long-term health and work performance.

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Data availability statement: The data that support the findings of this study are available from the corresponding author, DPB, upon reasonable request.

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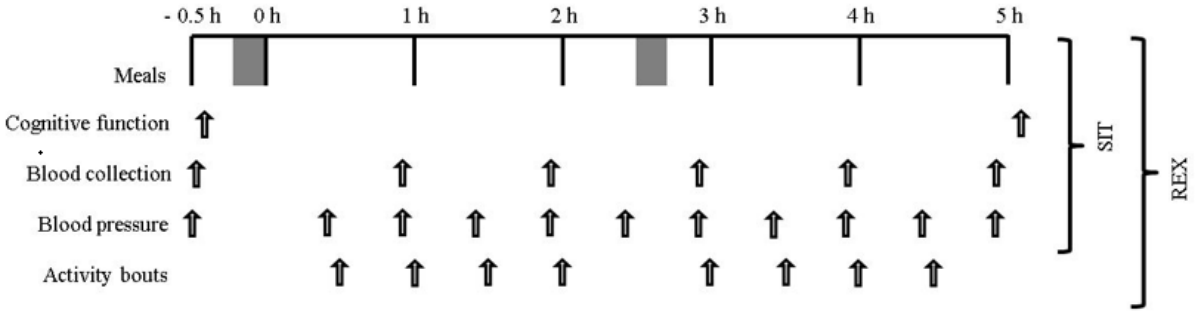

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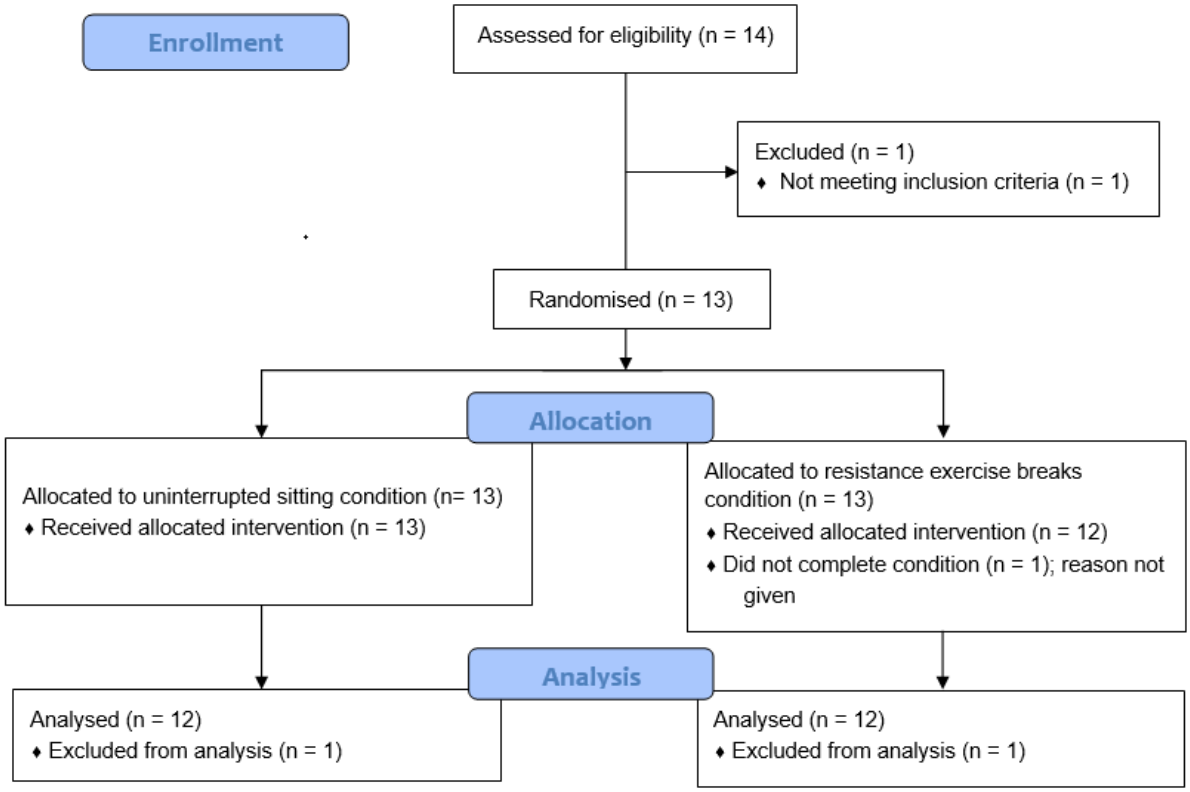
578 Figure 1. Schematic of experimental conditions. SIT, uninterrupted sitting condition; REX,

579 resistance exercise breaks condition.

580



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582 Figure 2. Participant flow throughout the trial.

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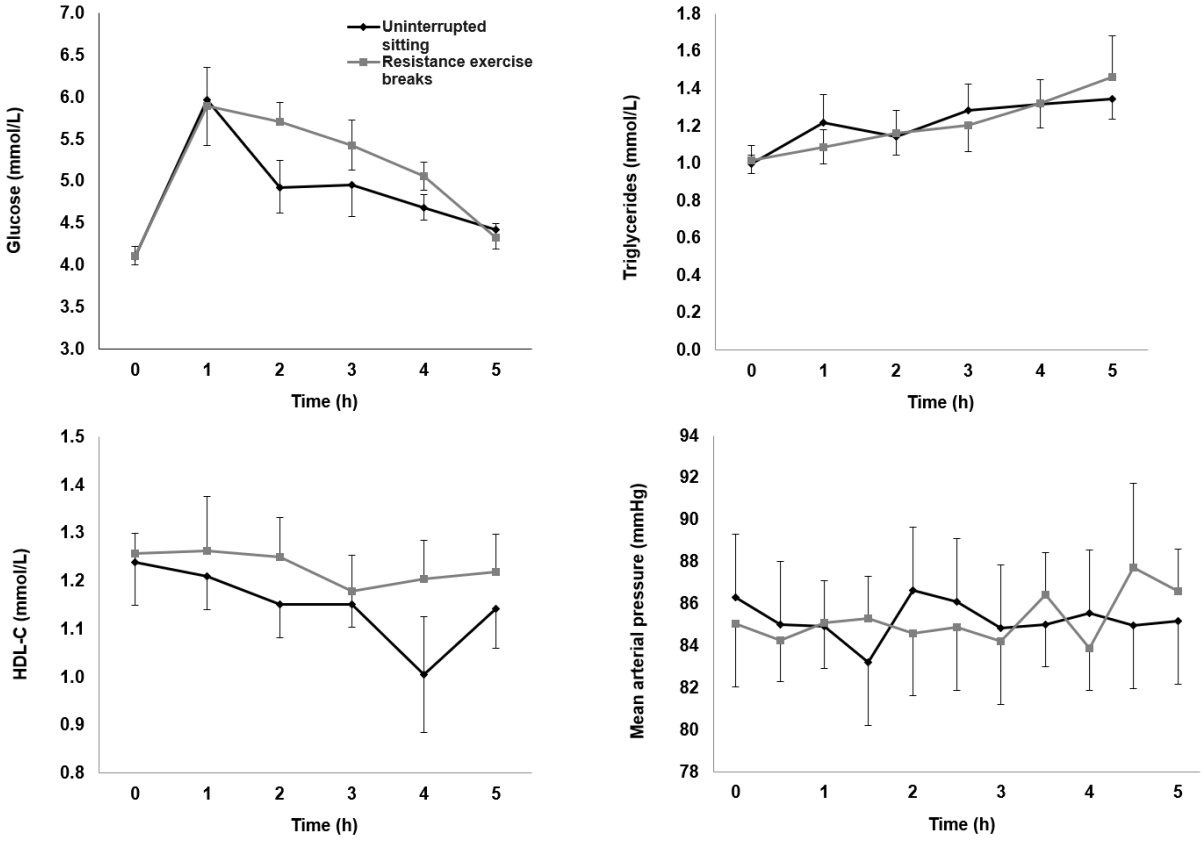

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585 Figure 3. Glucose, triglyceride, high-density lipoprotein cholesterol and mean arterial
586 pressure responses over time during the uninterrupted sitting and resistance exercise
587 breaks conditions. HDL-C, high-density lipoprotein cholesterol. Some error bars have been
588 omitted for clarity.

589 **Table 1**. Participant characteristics (n=12)

Variables	
Female (n)	7
Age (y)	25±6
Body weight	72.0±16.6 kg
Body mass index (kg/m^{-2})	24.7±4.9
Waist circumference (cm)	78.8±13.5
MVPA (min/week)	165±113
Physical activity (MET-min/week)	1455±1166
Sitting time (min/day)	521±125

590 Data presented as mean±SD.

591 MVPA, moderate-to-vigorous physical activity; MET, metabolic equivalent of task.

Table 2. Cognitive function test scores during the uninterrupted sitting and resistance exercise breaks conditions (n=12). Data presented as mean (95% confidence interval)

	Uninterrupted sitting		Resistance exercise breaks	
	Baseline	5-h	Baseline	5-h
Vigilance correct responses (n)[a]	22.3 (19.1, 25.5)	23.5 (20.3, 26.7)	25.0 (21.8, 28.2)	24.7 (21.5, 27.9)
Vigilance missed responses (n)[b]	9.6 (6.7, 12.5)	11.3 (8.4, 14.3)	10.2 (7.2, 13.1)	9.6 (6.7, 12.5)
Vigilance false responses (n)[b]	5.2 (2.7, 7.6)	3.3 (0.9, 5.8)	5.0 (2.6, 7.4)	4.8 (2.4, 7.3)
Probed memory recall (% of correct responses)[a]	49.2 (36.5, 61.9)	50.2 (37.2, 63.1)	42.9 (30.2, 55.6)	47.1 (34.4, 59.8)
Reaction thinking time (ms)[a]	475.5 (385.6, 565.4)	478.7 (388.8, 568.5)	538.2 (448.3, 628.0)	471.8 (381.9, 561.6)
Reaction movement time (ms)[a]	207.5 (158.7, 256.3)	185.8 (137.0, 234.5)	193.6 (144.8, 242.3)	176.4 (127.7, 225.2)

[a]higher scores indicate better cognitive function.

[b]lower scores indicate worse cognitive function.