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Leg fidgeting during prolonged sitting improves postprandial glycemic control in people with obesity

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Abstract

Objective: Studies show fidgeting augments metabolic demand and increases blood flow to the moving limbs, whereas prolonged sitting suppresses these factors and exacerbates postprandial glucose excursions. Thus, we hypothesized that leg fidgeting during prolonged sitting would improve postprandial glycemic control.

Methods: Adults with obesity (n=20) participated in a randomized, crossover trial in which blood glucose and insulin concentrations were measured during a 3-h sitting period following the ingestion of a glucose load (75g). During sitting, subjects either remained stationary or intermittently fidgeted both legs (2.5 minutes off/2.5 minutes on). Accelerometer counts, oxygen consumption, and popliteal artery blood flow were also measured during the sitting period.

Results: As expected, fidgeting increased accelerometer counts ($P<0.01$), oxygen consumption ($P<0.01$), and blood flow through the popliteal artery ($P<0.05$). Notably, fidgeting lowered both glucose ($P<0.01$) and insulin ($P<0.05$) total area under the curve (AUC) and glucose incremental AUC ($P<0.05$). Additionally, there was a strong, negative correlation between fidgeting-induced increases in blood flow and reduced postprandial glucose AUC within the first hour ($r=-0.569$, $P<0.01$).

Conclusions: Leg fidgeting is a simple light-intensity physical activity that enhances limb blood flow and can be incorporated during prolonged sitting to improve postprandial glycemic control in people with obesity.

Keywords

blood flow; physical activity; OGTT; glucose; insulin

INTRODUCTION

Obesity is associated with insulin resistance and postprandial hyperglycemia [1, 2], the latter of which is an independent predictor of cardiovascular disease and mortality [3].

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Accordingly, it is essential to identify strategies that mitigate postprandial glycemic excursions. Postprandial skeletal muscle blood flow and insulin-dependent skeletal muscle glucose uptake are impaired with obesity, contributing to postprandial hyperglycemia [4]. Sitting, while common during the postprandial period, further exacerbates glucose excursions [5–9]. This is likely owing to the depressed metabolic demand [10] and the resulting suppression of lower limb blood flow, which is further aggravated by arterial angulations. Indeed, we have recently reported that sitting causes a ~70% reduction in leg blood flow [11] and that a large fraction of this reduction is attributed to vascular resistance instigated by limb bending [12, 13].

While non-seated physical activity and reducing sitting time can diminish postprandial glycemia [5–9] and exert other health benefits, many barriers exist that preclude the adoption of these positive behaviors [14, 15]. These barriers include, but are not limited to, a perceived lack of time, workplace culture and behavior norms, task-specific duties throughout the day, and a lack of personal knowledge and confidence in performing physical activities [14, 15]. In addition to these barriers, recently the COVID-19 pandemic has resulted in prolonged confinements and limited access to recreational facilities which has ultimately reduced time spent being physically active [16, 17]. Therefore, an onus exists to evaluate seated physical activity as a therapeutic strategy to lessen postprandial hyperglycemia.

Importantly, insulin-independent glucose uptake, which is achieved by skeletal muscle contractions, is not entirely compromised by obesity or insulin resistance [18, 19]. To this point, sporadic leg fidgeting, not only elevates metabolic demand [20] but is a simple seated physical activity that produces increases in leg blood flow despite limb bending, as well mitigates sitting-induced vascular dysfunction [21]. Accordingly, we reasoned that the concomitant increases in skeletal muscle metabolic demand and blood flow induced by leg fidgeting could promote glucose uptake during the seated postprandial state. Specifically, we hypothesized that leg fidgeting would improve postprandial glycemic control in people with overweight or obesity during prolonged sitting.

METHODS

Participants and Study Design

This study was approved by the University of Missouri Institutional Review Board, registered at ClinicalTrials.gov (NCT03419754), and all data collection was conducted on-site at the University of Missouri-Columbia. Participants were recruited via advertising on campus and around the Columbia city area. Recruitment and data collection occurred between January 2018 and January 2020. Written informed consent was obtained from all participants, who were then screened to determine eligibility. The following inclusion criteria were implemented: 1) females and males; 2) 20 to 60 years of age; 3) body mass index (BMI) >25 $kg \cdot m^{-2}$; 4) no known active cancer or cardiovascular, pulmonary, kidney, or liver disease; 5) no diagnosed diabetes mellitus; and 6) non-smoking.

Participants completed an unblinded, randomized, crossover study in which they remained stationary or fidgeted both legs intermittently during 3 hours of sitting following an oral

glucose challenge. Subjects completed the intervention conditions sequentially with the intervention order being allocated via simple randomization on a 1:1 basis by the consenting study representative. A crossover design was used so that each subject would serve as their own controls and that within-subject variation would be less than between-group variation. A minimum of seven days elapsed between the two experimental visits as a wash-out period to prevent any potential carry-over effects from the previous visit. Premenopausal women (n=9) had their experimental visits scheduled during the early follicular phase (days 1 – 7 of the menstrual cycle) to minimize the impact of the menstrual cycle on metabolic and vascular outcomes. Therefore ~28 days elapsed between experimental visits for these participants. Four women were taking oral contraceptives and were scheduled during their placebo week. Two women had an intrauterine device and self-reported regular menses. For all visits, participants arrived at the laboratory after an overnight fast (10–12 h). Subjects refrained from exercise for 24–48 h, caffeine for 12 h, and alcohol for 24 h before testing. Subjects could take prescription medications the night before and the morning of testing. No subject took any glucose regulating medication.

Screening procedures

During the screening visit, the subject's anthropometrics were recorded. A blood sample via a finger stick was collected to measure fasting glucose, total cholesterol, high-density lipoprotein cholesterol, and triglyceride levels. Seated brachial artery blood pressure was recorded in duplicate using an automated sphygmomanometer (SphygmoCor XCEL, AtCor Medical, Itasca, IL).

Experimental procedures

A schematic of the experimental visit is presented in Figure 1. Participants recorded their food intake for 24 h before arriving to the laboratory. Participants were given a copy of their food intake record and instructed to replicate the meals and timing of food intake, if possible, the day before their second visit. After arriving, subjects had their height and weight recorded, and an intravenous catheter placed in an antecubital vein. Subjects were then seated, and after a 15-minute acclimation period, an accelerometer (ActiGraph GTX3; ActiGraph, Pensacola, FL) was fixed on the front of the subject's right thigh, just above the knee cap. The leg designated for ultrasound recordings was positioned for optimal imaging of the popliteal artery, and skin location was marked for repeated ultrasound probe placement. The subject's foot placement and chair position were marked on the floor to ensure consistent positioning within- and between visits.

The subject was then fitted with a facemask to measure baseline oxygen consumption via indirect calorimetry for 20 minutes (TrueOne 240 Metabolic Measurement Cart; ParvoMedics, Sandy, UT). Afterward, baseline blood flow measurements were collected by recording 2 minutes of continuous popliteal artery diameter and blood velocity of the designated leg using duplex-Doppler ultrasound (Logiq P5; GE Medical Systems, Milwaukee, WI).

After these measurements, baseline blood samples (~6 mL) were collected for analysis of fasting glucose and insulin concentrations. A 75-g oral glucose load (OGTT; Thermo

Scientific, Inc.) was ingested within 5 minutes, and blood samples (3 mL) were collected at 15, 30, 45, 60, 90, 120, 150, and 180 minutes after ingestion. Blood samples were placed in EDTA vacutainers and immediately analyzed for glucose. Blood samples were later centrifuged (4°C, 3.5 rpm, for 15 minutes), and the plasma was aliquoted and stored at −80°C for later analysis of insulin levels. Postprandial oxygen consumption was recorded for 15 minutes leading up to every 30-minute blood draw after glucose ingestion. Postprandial blood flow measurements were collected at every 30-minute blood draw after glucose ingestion by recording 2 minutes of continuous popliteal artery diameter and blood velocity.

During the 3 h of sitting following the glucose challenge, subjects were randomized to either remain stationary or to fidget both legs intermittently. For the intermittent fidgeting intervention, subjects rested their legs for 2.5 minutes and then tapped their heels and bounced their knees at their natural cadence for 2.5 minutes, similar to as previously described [21]. This cycle was repeated for 3 h. Subjects followed auditory cues via a programmable timer on when to start and stop their fidgeting bouts. Due to the technical difficulty of obtaining Doppler ultrasound images during fidgeting, ultrasound recordings were initiated immediately after cessation of the fidgeting bout.

Measurements and calculations

Blood profile: Fasting total cholesterol, high-density lipoprotein cholesterol, and triglycerides were measured using Cholestech LDX lipid profile cassettes (Cat # 10-989) that were read by a Cholestech LDX analyzer (Alere Inc., San Diego, CA). Blood glucose concentrations were analyzed using the YSI 2300 STAT PLUS glucose analyzer (YSI Incorporated, Yellow Springs, OH). Plasma samples were run on commercially available ELISA kits for insulin (ALPCO Cat# 80-INSHU-E10.1, Salem, NH), according to the manufacturer guidelines. All samples from a participant, collected at each timepoint and across both conditions were run in duplicate on the same 96-well plate. Each 96-well plate accommodated the loading of all duplicated samples for two participants. The intra-assay coefficient of variance was 4.9% and the inter-assay coefficient of variance was 13.4%, as measured by the investigator.

Matsuda insulin sensitivity index: The Matsuda insulin sensitivity index (ISI) provided an estimate of whole-body insulin sensitivity in response to the OGTT and was calculated (10,000/square root of {fasting glucose × fasting insulin} × {mean glucose × mean insulin during OGTT}) as previously described [22]. The mean glucose and insulin concentrations during the OGTT were calculated from the 30, 60, 90, and 120-minute blood draws [22].

Accelerometer: The accelerometer recorded data at a sampling rate of 30Hz. Data for the 3 h of sitting after glucose ingestion were downloaded in 30 second epochs using the manufacturer's software (ActiGraph software v 6.13.3, Pensacola, FL). The sum of vector magnitude (VM) counts was used in the analysis. The VM is the magnitude of the resulting vector that forms when combining the sampled acceleration from all three axes of the accelerometer.

Oxygen consumption: The metabolic cart was gas-, volume-, and flow-calibrated per manufacturer instructions. All breathing parameters were averaged over 1-minute intervals. The last 10 minutes of each collection period was used for data analysis.

Popliteal artery blood flow: Two minutes of simultaneous popliteal artery diameter and blood velocity were measured using 2D/Doppler ultrasound. Popliteal artery diameter and velocity were recorded using an 11 MHz linear array transducer just distal to the popliteal fossa. Signals were obtained in duplex mode at a pulsed frequency of 5 MHz and corrected with an insonation angle of 60°. The sample volume encompassed the lumen of the vessel without extending beyond the walls, and the cursor was set mid-vessel. Ultrasound recordings were analyzed offline using commercially available edge-detection software (Cardiovascular Suite Version 3, Quipu srl, Pisa, Italy). Blood flow was calculated from the continuous diameter and mean blood velocity recordings using the following equation and with flow reported as mL/min: Blood flow = 3.14 * diameter (cm)/2^2 * mean blood velocity (cm/s) * 60.

Sample Size

Previous studies have reported a decrease in postprandial glucose area under the curve (AUC) in response to interrupting prolonged sitting with light physical activity by 24.1% in people with overweight or obesity [6], and a decrease of 39% in patients with type 2 diabetes [5]. Using an effect size of $d = 0.84$ and a two-tailed α level <0.05 with 90% power as previously reported [5, 6], it was determined that 17 paired observations would be needed to detect a significant treatment effect. Sample size was calculated using G*Power (v3.1.9.2, Dusseldorf, Germany).

Statistical analysis

A two-way repeated-measures ANOVA with fidgeting and time as factors were used to analyze blood glucose and plasma insulin concentration curves. Total AUC responses were calculated with the trapezoidal method using SPSS (Version 25, IBM Inc.) for blood glucose and plasma insulin over the 3 h. Incremental AUC (iAUC) responses were calculated by subtracting the fasting area from the calculated total AUC for each participant. Total AUCs and iAUCs for glucose and insulin responses were compared with a paired samples t-test, respectively.

Blood flow curves over the 2-minute recording time were plotted for each collection timepoint (0, 30, 60, 90, 120, 150, and 180-minutes). As expected (13), following cessation of each bout of fidgeting there is a decay in blood flow. To assess if blood flow curves were different between the no fidget and fidget conditions at each of the collection timepoints, independent two-way repeated measures ANOVAs were performed for each collection timepoint with main effects of fidgeting and time (1 data point per second over the two-minute recording period).

When all blood flow curves were superimposed, curves within the fidgeting condition appeared different as the time over the 3 h progressed. Therefore, to assess if time over the 3 h of sitting had any influence, AUCs for each blood flow curve were calculated and

compared against one another. Blood flow AUCs were generated for each collection timepoint (0, 30, 60, 90, 120, and 180-minutes) by using the trapezoidal method over each two-minute recording timeframe (i.e. 1 data point per second over the two-minute recording period). A two-way repeated measures ANOVA with main effects of fidgeting and collection timepoint (0, 30, 60, 90, 120, and 180-minutes) was performed.

A mixed-effects model with fidgeting and time as fixed effects were used to analyze oxygen consumption to accommodate missing observations (n=3). Pairwise corrections were performed with Bonferroni adjustment. Paired samples t-tests were used to compare the following variables between the no fidget and fidgeting conditions: baseline anthropometrics, Matsuda ISI, and accelerometer VM. Possible relationships of interest were examined using Pearson correlation. Cohen's *d* (paired *t* tests) was used to calculate effect size. Statistical analyses were performed using SPSS (Version 25, IBM Inc.). Significance was accepted if $P<0.05$. Data are presented as means ± SEM.

RESULTS

Participants: The number of participants at each stage of the study are presented in Figure 2. Twenty subjects completed both trials and were included in the analysis, unless otherwise stated (15 females, 5 males; Age: 42±3 years; Weight: 102.7±5.1 kg; BMI: 37.5±2.1 kg/m^2; Data are presented as means ± SEM). Additional subject demographics are presented in Table 1. Subject weight was slightly higher in the fidgeting visit (No Fidget: 102.4±4.9 vs. Fidget: 103±5.1 kg; $P<0.05$), but BMI was similar between conditions (No Fidget: 37.5±2 vs. Fidget: 37.4±2 kg/m^2; P=NS). Twelve subjects met the criteria for having metabolic syndrome [23]. The fidgeting protocol and study design did not produce any harms or adverse events. The study ended upon the estimated sample size being obtained and the effect size and power being verified for the primary outcomes of postprandial glycemic responses.

Accelerometer counts and Oxygen consumption: As designed, fidgeting accumulated more accelerometer counts (No Fidget: 1549±579 vs. Fidget: 29160±8021; $P<0.01$, Fig. 3a). Due to potential leakage during gas collection on two separate occasions, two participants were excluded from the oxygen consumption analysis (n=18). Baseline oxygen consumption was similar between conditions (No Fidget: 245±14 vs. Fidget: 244±14 mL/min; P=NS). Fidgeting increased oxygen consumption above baseline and was higher than the no fidget condition at each subsequent time point ($P<0.01$, Fig. 3b).

Popliteal artery blood flow: Blood flow curves were similar between conditions at baseline (P=NS, Fig. 3c). At each collection timepoint (30, 60, 90, 120, 150, and 180-minutes) following glucose ingestion, fidgeting bouts increased popliteal artery blood flow that tapered off during the 2-minute recording periods after fidgeting cessation (P<0.001, Fig. 3c).

When all blood flow curves were superimposed, curves within the fidgeting condition appeared different as the time over the 3 h progressed. Therefore, to assess if time over the 3 h of sitting had any influence, AUCs for each blood flow curve were calculated and

compared against one another. Blood flow AUCs during the no fidget condition were similar across all time point collections (P=NS, Fig. 3d). In response to fidgeting, blood flow AUCs were higher than the baseline value from 60-minutes onwards (P<0.05, Fig. 3d). Within the fidget condition, there were no differences in blood flow AUCs from 60-minutes onwards (P<0.05, Fig. 3d). Blood flow AUCs were higher in the fidget condition than the no fidget condition from 60 minutes onwards (P<0.05, Fig. 3d). Log transformed data for blood flow AUCs did not alter the results that were found.

Postprandial glucose levels: Baseline glucose values were similar between conditions (No Fidget: 4.98±0.15 vs. Fidget: 4.89±0.16 mmol/L; *P*=NS), and the pattern of glucose response to the glucose challenge was similar. However, glucose concentrations were lower in the fidgeting condition than the no fidget condition at time points 30, 45, 60, 90, and 180 minutes (*P*<0.05, Fig. 4a). Overall, fidgeting lowered total glucose AUC by a large effect size (No Fidget: 1331.10±71.74 vs. Fidget: 1242.38±75.02 mmol/L × min over 3 h; *P*<0.01, *d*=0.86, Fig. 4b), with seventeen subjects positively responding to the fidgeting protocol (Fig. 4e). Furthermore, fidgeting lowered glucose iAUC by a moderate effect size (No Fidget: 443.59±52.55 vs. Fidget: 379.56±48.94 mmol/L × min over 3 h; *P*<0.05, *d*=0.61), with twelve subjects positively responding to the fidgeting protocol.

Postprandial insulin levels: Baseline insulin concentrations were similar between conditions (No Fidget: 117.27±24.37 vs. Fidget: 96.93±17.10 pmol/L; *P*=NS). The pattern of insulin response was also similar between conditions, with insulin concentration only being significantly lower at time 180 minutes due to fidgeting (*P*<0.05; Fig. 4c). Overall, fidgeting lowered insulin total AUC by a moderate effect size (No Fidget: 106838.10±13920.42 vs. Fidget 92686.27±13120.77 pmol/L × min over 3 h; *P*<0.05, *d*=0.53, Fig. 4d), with fifteen subjects responding positively to the fidgeting protocol (Fig. 4f). However, fidgeting did not lower insulin iAUC (No Fidget: 1437.68±218.27 vs. Fidget 1255.86±201.14 pmol/L × min over 3 h; *P*=NS). The Matsuda ISI was higher in the fidgeting condition than the sedentary condition by a moderate effect size (No Fidget: 3.3±0.4 vs. Fidget: 4.3±0.7; *P*<0.05, *d*=0.54).

Correlations: Of the correlations performed, there was a strong, negative correlation between fidgeting-induced lowering of glucose AUC and increased blood flow AUC, within the first hour of sitting (r=−0.569, *P*<0.01; Fig. 5).

DISCUSSION

Postprandial glucose excursions are accentuated during inactive sitting in healthy lean individuals [8] and people with overweight or obesity, with [5] and without type 2 diabetes [6, 7, 9]. This exacerbation with sitting is likely attributed to the dampened metabolic demand and consequent suppression of leg blood flow, further depressed because of arterial bending. Interrupting sitting with non-seated physical activity can lower postprandial glucose [5–9]. Interestingly, interrupting prolonged sitting with seated arm ergometry has also been shown to lower postprandial glucose levels [24]. Yet it is unknown if physical activity localized to the lower body during sitting can lower postprandial glucose. Herein, we provide novel evidence that fidgeting during prolonged sitting increased metabolic

demand and leg blood flow despite the restrictive effects of limb bending, improving postprandial glycemic control in people with obesity.

Previous studies show that interrupting prolonged sitting with physical activity breaks can lower postprandial metabolic responses. Dunstan et al. [6] showed that interrupting 5 h of sitting, with 2-minute bouts of either light- or moderate-intensity walking, every 20 minutes, lowered incremental postprandial glucose and insulin AUC responses, in people with overweight or obesity. Dempsey et al. [5] found similar responses in patients with type 2 diabetes when 8 h of sitting was broken up with 3-minute bouts of either light-intensity walking or body weighted resistance exercises, every 30 minutes. Henson et al. [7] showed that disrupting 7.5 h of sitting, with 5-minute bouts of either standing or light-intensity walking, every 30 minutes, lowered incremental postprandial glucose, insulin, and non-esterified fatty acid AUC responses within insulin-resistant, postmenopausal women. Similarly, Bailey and Locke [9] found that 2-minute bouts of light walking every 20 minutes lowered postprandial glucose AUC in people with overweight. Improvements in postprandial responses may be in part due to inducing an energy deficit, as Bailey et al. [25] found no significant impact on appetite or gut hormone concentrations. Our findings agree with observations from these previous studies but expand upon them by demonstrating that seated leg fidgeting can also be effective at reducing postprandial glucose and insulin AUC responses.

Recent meta-analyses have attempted to assess the effectiveness of interrupting prolonged sitting to lower postprandial glucose and insulin responses [26–28]. The effectiveness of physical activity breaks to lower postprandial glucose iAUC in people with obesity, impaired fasting glucose, or type 2 diabetes greatly varied across 24 studies presented within a meta-analysis by Loh et al. [26]. Overall, physical activity breaks moderately attenuated postprandial glucose and insulin iAUC within these dysglycemic populations [26]. In another study that had people with obesity interrupt prolonged sitting with seated physical activity breaks, McCarthy et al. [24] found seated arm ergometry performed at an intensity equivalent to light walking (3km/h) had a large effect on reducing postprandial glucose iAUC and a small effect on reducing postprandial insulin iAUC.

While physical activity breaks appear effective, the effectiveness of standing breaks is inconsistent. In a meta-analysis by Saunders et al. [28], standing breaks did not significantly lower postprandial glucose and insulin iAUCs. However, Henson et al. [7] showed that standing and light walking breaks similarly induced large reductions in postprandial glucose and insulin iAUCs within insulin-resistant, postmenopausal women. In agreement with some analyses, we found leg fidgeting moderately lowered postprandial glucose iAUC in people with obesity but did not lower postprandial insulin iAUC. Future studies are needed to directly compare leg fidgeting’s effectiveness against other physical activity break strategies in reducing postprandial glucose levels in people with obesity.

Strategies that increase energy expenditure during prolonged sitting improve postprandial glucose responses [29]. Leg fidgeting using under-the desk devices can increase energy expenditure above rest by ~20–30%, whereas exercising to a workout video and light-walking can double energy expenditure above rest [20]. We found leg fidgeting increased

oxygen consumption by ~20% during sitting, despite the movement involving only a small amount of muscle mass. Thus, like resistance exercise and walking [5], leg fidgeting while seated increased energy expenditure, and this increase was sufficient to lower postprandial glucose and insulin.

According to the findings presented herein, the prescription of light-intensity physical activity to lessen postprandial glycemia during sitting should be considered. While reducing sitting time is crucial for health outcomes and should be promoted as much as possible, barriers can hinder the feasibility of people engaging in such behaviors during working hours [14, 15]. Moreover, most people even choose to sit during their free-leisure time [30]. Leg fidgeting or, more specifically, plantar dorsiflexion of the feet, can be performed anywhere, anytime, without external equipment, and may be of added benefit to those who find walking challenging. Nevertheless, further work is needed to examine if leg fidgeting and other seated physical activities are effective at offsetting the long-term metabolic consequences of sitting.

As with many human studies, there are strengths and limitations. Firstly, we were unable to recruit equal numbers of men and women for this study but assessing sex-differences in response to fidgeting was not a purpose for this study. Additionally, despite the lack of men in our study, our large sample size of middle-aged overweight-obese individuals adds to the small number of studies performed in a population with cardiometabolic disease risk. Secondly, we did not explicitly control for food intake the evening before the subject's overnight fast by providing a standardized meal. The subjects were asked to record food intake for 24 h before their experimental visit via a food diary which was then given to the subject a few days before the second study day for them to replicate for their dietary intake. This allowed them to maintain their dietary eating habits and subsequently, fasting glucose and insulin levels were similar between conditions.

CONCLUSION

Our findings support the implementation of leg fidgeting during prolonged sitting as a simple strategy to improve postprandial glycemic control in people at high risk for cardiometabolic disease. The amelioration of postprandial glucose levels with leg fidgeting is likely attributed to repeated skeletal muscle contractions and resulting in an increase in skeletal muscle blood flow, both contributing to glucose disposal.

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All the individual participant data collected during the trial, after deidentification will be shared, beginning 9 months, and ending 36 months following article publication. The study protocol will also be shared. Data will be shared with investigators whose proposed use of the data has been approved by an independent review committee identified for this purpose. The data will be available for individual participant data meta-analysis. Proposals may be submitted up to 36 months following article publication. After 36 months the data will be available in our University's data warehouse but without investigator support other than deposited metadata. Information regarding submitting proposals and accessing data may be found with the corresponding author.

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STUDY IMPORTANCE QUESTIONS

What is already known about this subject?

- Sitting reduces metabolic demand and markedly suppresses blood flow to the lower limbs, mainly because of arterial angulations associated with the posture, which together could contribute to the exacerbated postprandial glycemic excursions observed during inactive sitting. Yet, interrupting prolonged sitting with physical activity strategies such as walking or simple resistance exercises improves postprandial glycemic responses in people with obesity, likely due to increasing metabolic demand.
- Similarly, leg fidgeting augments metabolic demand and increases blood flow to the moving limbs, and hence leg fidgeting during sitting might improve postprandial glycemic responses in people with obesity

What are the new findings in your manuscript?

- Leg fidgeting during prolonged sitting can improve postprandial glycemic responses in people with obesity.

How might your results change the direction of research or the focus of clinical practice?

- These findings emphasize that physical movement even while sitting is an important strategy for reducing postprandial glucose excursions, especially for individuals at a higher risk for cardiovascular disease and type 2 diabetes.

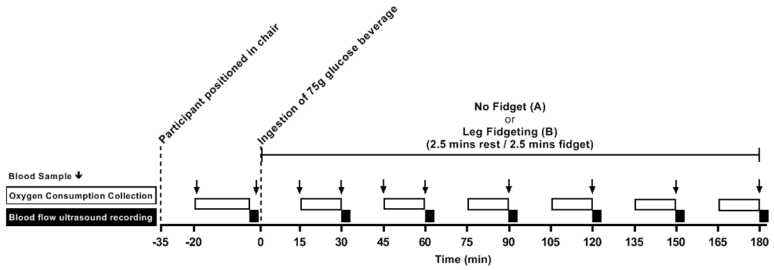

Fig. 1 -
Experimental visit study design.

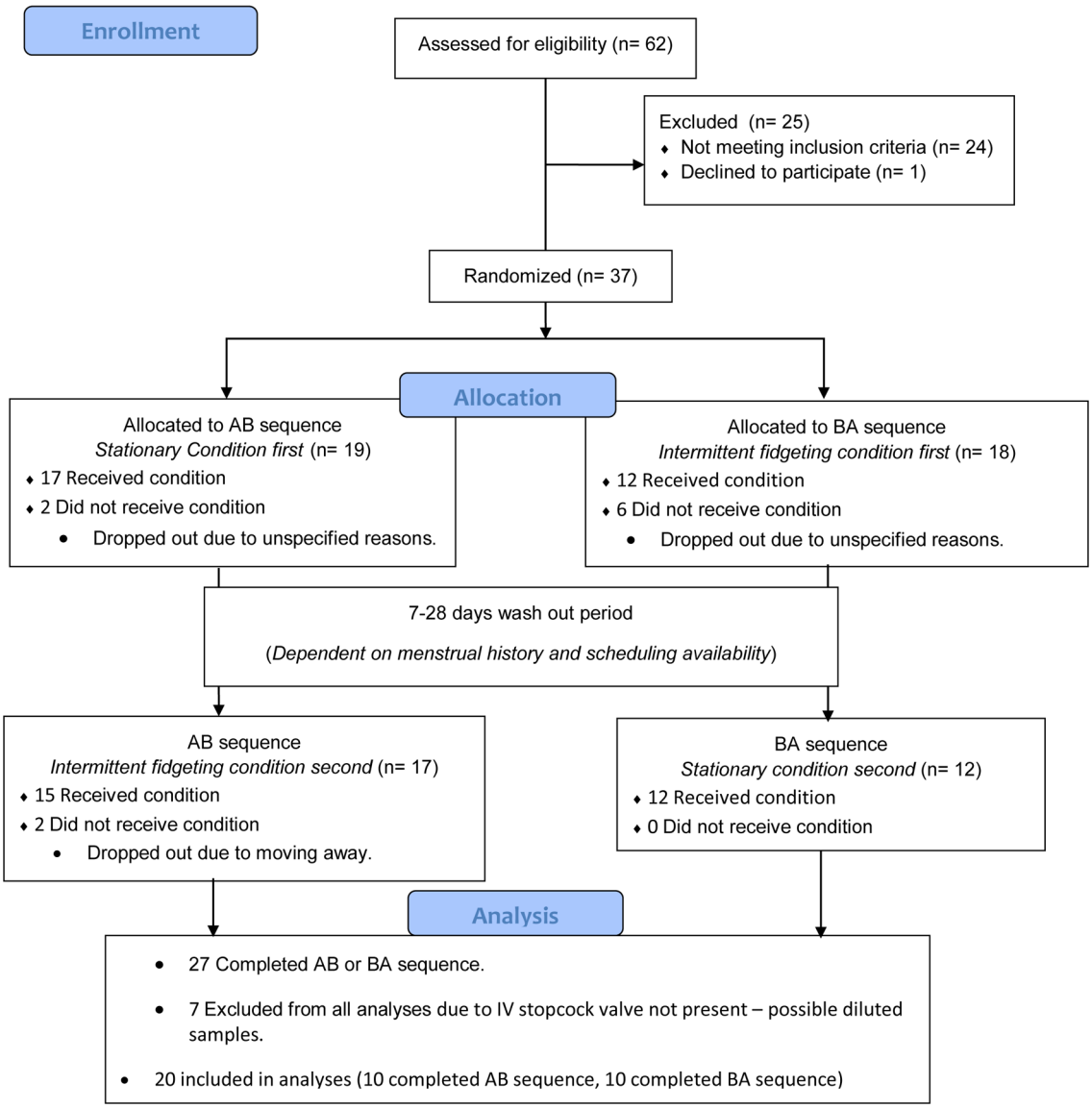

Fig 2. –.

Flow diagram of study design and the number of participants at each stage.

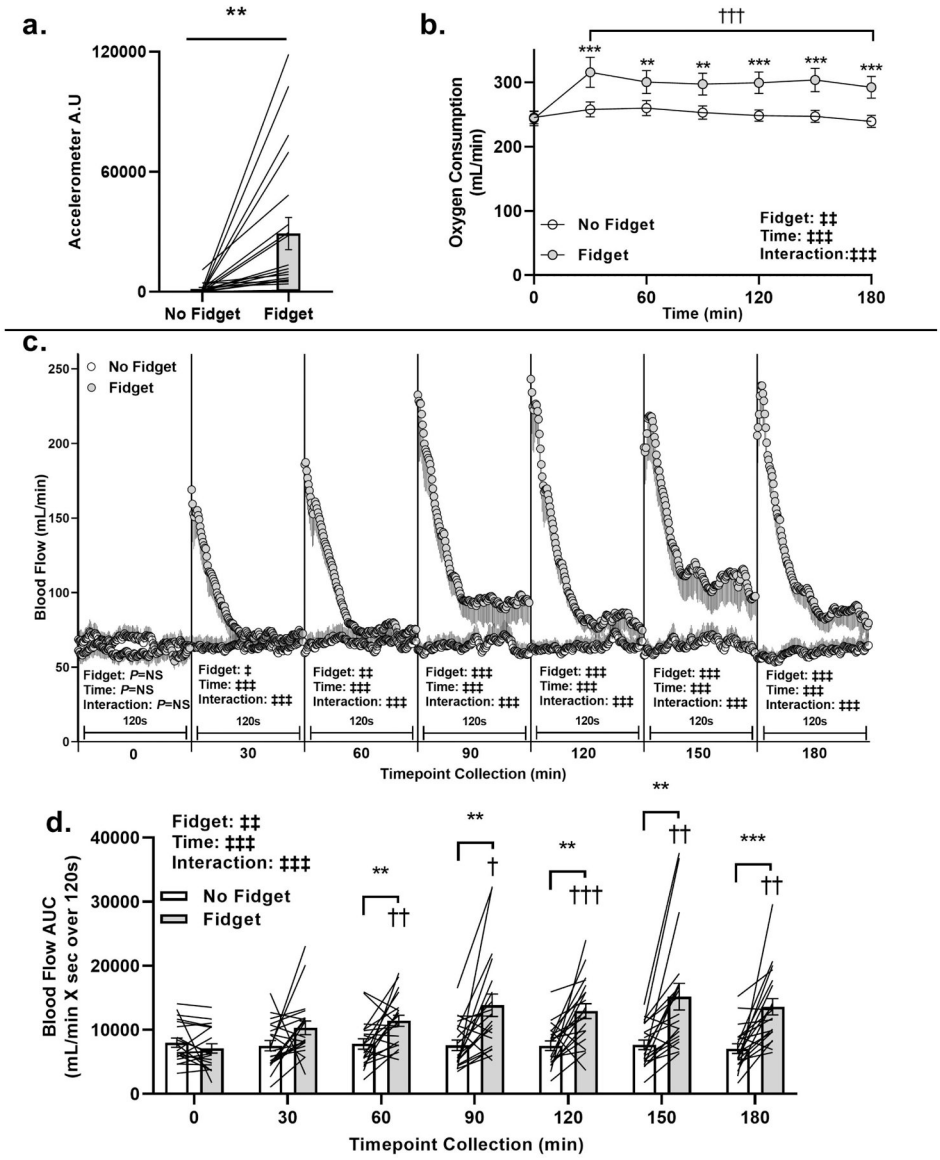

Fig. 3 -

Effect of seated bilateral leg fidgeting on (a) accelerometer counts, (b) oxygen consumption, and (c-d) popliteal artery blood flow during prolonged sitting (3 h) after a 75-g oral glucose challenge. Data are means ± SEM. Individual responses are overlayed as spaghetti plots, where appropriate. * P <0.05, ** P <0.01, *** P <0.001, between No fidget vs. Fidget. † P <0.05, †† P <0.01, ††† P <0.001, Fidget 0 mins vs. other timepoint within Fidget condition. ‡ P <0.05, ‡‡ P <0.01, ‡‡‡ P <0.001, two-way repeated measures ANOVA.

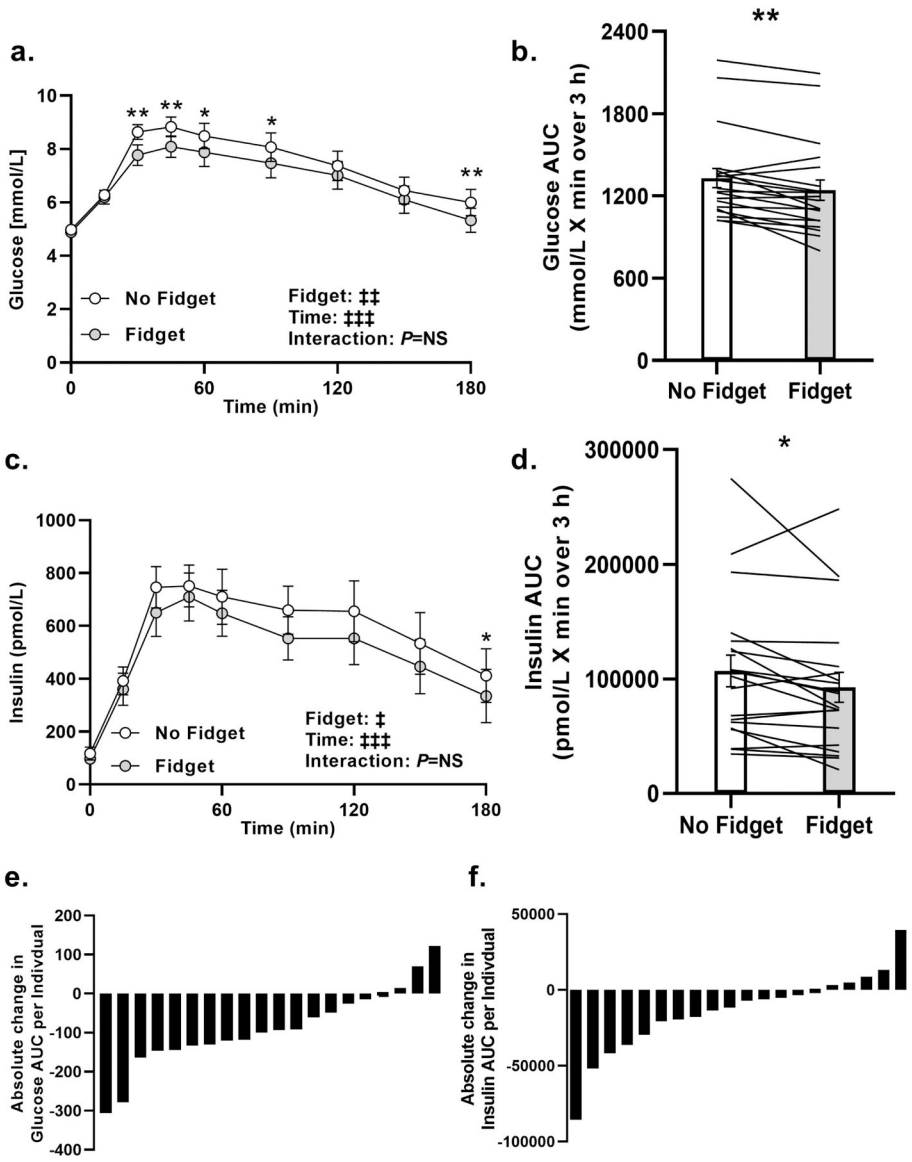

Fig. 4 -

Effect of seated bilateral leg fidgeting on postprandial glucose and insulin levels during prolonged sitting (3 h) after a 75-g oral glucose challenge. No fidget and fidget postprandial (a) glucose and (c) insulin curves with corresponding total area under the curve responses (b and d, respectively). Absolute change in (e) glucose and (f) insulin total area under the curve per individual, reading from left on the X-axis is the greatest response to fidgeting, irrespective of subject order. Data are means ± SEM. Individual responses are overlayed as spaghetti plots, where appropriate. * $P<0.05$, ** $P<0.01$, *** $P<0.001$, between No fidget vs. Fidget. ‡ $P<0.05$, ‡‡ $P<0.01$, ‡‡‡ $P<0.001$, two-way repeated measures ANOVA.

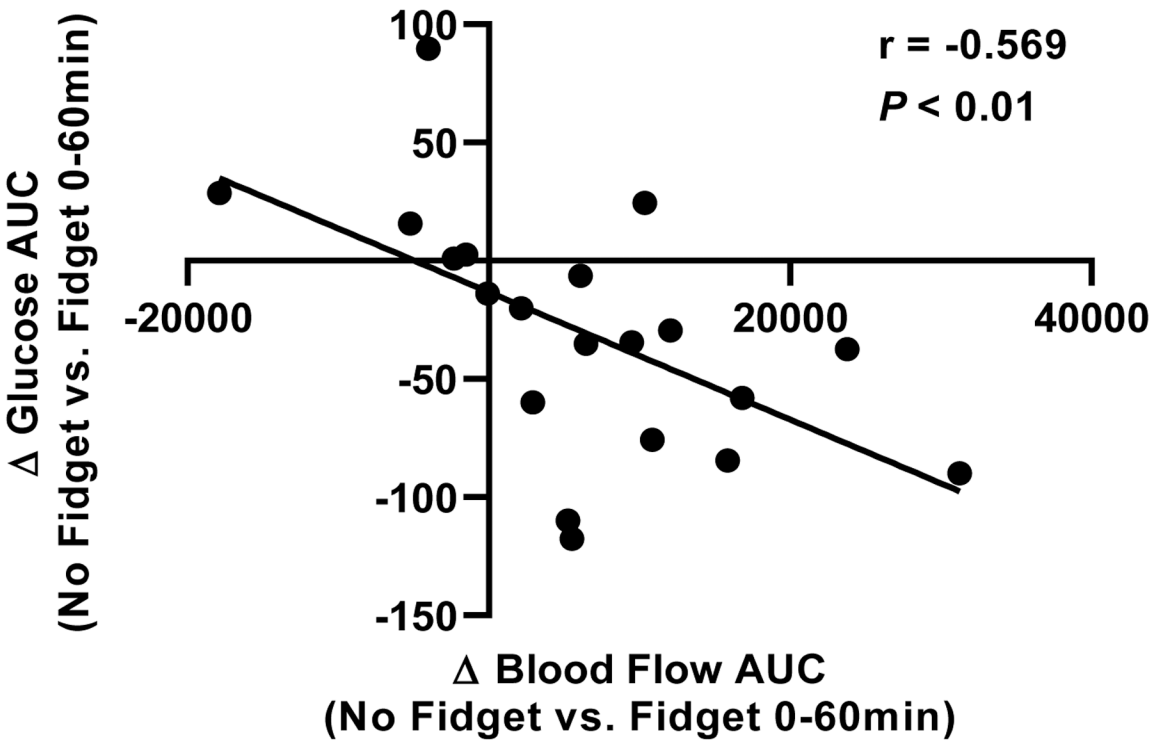

Fig. 5 -

Correlation between fidgeting-induced reductions in glucose total AUC and increased blood flow AUC within the first hour of sitting.

Table 1.

Subject characteristics, anthropometrics, blood profile parameters, and medications.

Age (years)	42±3
Sex (F/M)	(15/5)
Race (number of subjects)	
Asian	1
Black	3
Non-Hispanic White	16
Height (cm)	165.9±2.0
Weight (kg)	102.7±5.1
Body Mass Index (kg/m^2)	37.5±2.1
Waist circumference (cm)	114.7±3.6
Hip circumference (cm)	125.2±3.0
Waist-to-Hip Ratio	0.92±0.02
Systolic blood pressure (mmHg)	130±3
Diastolic blood pressure (mmHg)	82±2
Fasted blood glucose (mmol/L)	4.97±0.13
Lipids (mmol/L)	
Triglycerides	1.24±0.13
Total cholesterol	4.85±0.12
High Density Lipoprotein (HDL) cholesterol	1.31±0.10
Low Density Lipoprotein (LDL) cholesterol	3.08±0.22
Subjects with Metabolic Syndrome	12
Medications (number of subjects)	
Angiotensin-converting enzyme (ACE) Inhibitor	3
Diuretic	1
Estrogen modulator	1
Intrauterine device (IUD)	2
Oral contraceptive	4
Synthetic Thyroid Hormone	1

Data as mean±SEM.