Frequency of Breaks in Sedentary Time and Postprandial Metabolic Responses

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ABSTRACT

HAWARI, N. S. A., I. AL-SHAYJI, J. WILSON, and J. M. R. GILL. Frequency of Breaks in Sedentary Time and Postprandial Metabolic Responses. Med. Sci. Sports Exerc., Vol. 48, No. 12, pp. 2495–2502, 2016. Purpose: To compare the metabolic effects of breaking up sedentary time with prolonged periods of standing versus multiple shorter standing bouts with the same total duration to determine whether, in principle, altering the frequency of “standing breaks” in sedentary time, influences metabolic responses over the course of the day. Methods: Ten normoglycemic overweight/obese men (age, 33 ± 13 yr; body mass index, 28.3 ± 3.0 kg m⁻²; mean ± SD) each participated in three experimental trials in random order, in which they arrived fasted, then consumed a test breakfast (8 kcal kg⁻¹ body weight, with 37% energy from fat, 49% from carbohydrates, 14% from protein) and, 4 h later, an identical test lunch. Expired air and blood samples were taken fasted and for 8 h postprandially. In one trial (uninterrupted sitting), participants sat continuously throughout the observation period; in the prolonged standing (PRO-Stand) trial, participants stood still for 15 min every 30 min; and in the intermittent standing trial (INT-Stand), they stood for 1.5 min, 10 times every 30 min. Results: Compared with uninterrupted sitting, energy expenditure was 320 ± 62 kJ (10.7% ± 2.0% higher in PRO-Stand and 617 ± 76 kJ (20.4% ± 2.3% higher in INT-Stand) energy expenditure in INT-Stand was 296 ± 78 kJ (9.0% ± 2.3% higher than PRO-Stand (mean ± SEM; all P < 0.001). However, there were no significant differences between trials in postprandial glucose, insulin, or triglyceride responses. Conclusions: These data demonstrate an independent effect of frequency of sedentary breaks on energy expenditure which provides an explanation for the association between frequency of sedentary breaks and adiposity observed in epidemiological data. However, it may be necessary to break up sitting with activities of greater intensity than quiet standing to positively influence glucose, insulin, and triglyceride metabolism in relatively young, normoglycemic, overweight/obese men. Key Words: SITTING, STANDING, ENERGY EXPENDITURE, GLUCOSE, INSULIN, TRIGLYCERIDE

There is a large body of observational data showing strong associations between time spent engaged in sedentary behavior—defined as nonsleeping activities in a sitting or reclining posture with energy expenditure ≤1.5 METs (where 1 MET is resting energy expenditure) (25)—and a number of adverse health outcomes, including mortality, cardiovascular disease, type 2 diabetes, and obesity (5,12,29,30). These relationships are often independent of time spent engaged in moderate-to-vigorous physical activity (>3 METs) (5,12,29,30). In addition, recent observational data in almost 700 adults from the AusDiab study, using a postural sensor to objectively monitor time spent sitting, standing, and stepping, suggested that isometrically replacing sitting with standing was associated with favorable changes to glucose and lipid metabolism (13). There is also observational evidence to suggest that individuals who break up sedentary time more frequently have a more favorable cardiometabolic risk profile—particularly with respect to adiposity variables—than those who habitually engage in prolonged periods of uninterrupted sedentary time, independent of total time spent sedentary (3,11,12). However, the mechanisms by which more frequent breaks in sedentary time may impart these benefits, independent of total sedentary time, are unclear. A number of short-term intervention studies have shown that interrupting sedentary periods with multiple short (<3 min) bouts of light or moderate activity throughout the day can reduce postprandial glucose, insulin, and triglyceride (TG) responses, and blood pressure, on the same or following day (4,18,22,23). Other studies have shown that interrupting prolonged sitting with periods of static standing ranging from 5 min every 30 min (14) to 30 min every hour (28), can reduce postprandial glucose concentrations. However, in all of these studies, sedentary time was replaced by standing or walking, leading to a reduction in total time spent sedentary, so the effects of altering the frequency of breaks in sedentary time, independent of changing total time sedentary, on these metabolic responses are not known. It is also not known whether altering the frequency of breaks in sedentary time influences metabolic rate and
substrate utilization, which may provide an explanation for
the association between frequency of sedentary breaks and
adiposity observed in the epidemiological data (3,11,12). The
aim of this study was therefore to compare the metabolic ef-
fects of breaking up sedentary time with prolonged periods of
standing versus multiple shorter standing bouts with the same
total duration to determine whether—in principle—altering
the frequency of breaks in sedentary time, influences meta-
bolic responses over the course of the day.

METHODS

Participants

Ten men, age 33 ± 13 yr, with body mass index, 28.3 ±
2.8 kg·m⁻²; waist circumference, 100.2 ± 9.5 cm [mean ± SD];
and low levels of habitual physical activity (less than 2 h·wk⁻¹
of moderate-to-vigorous physical activity as assessed by the
International Physical Activity Questionnaire), were recruited
for this study though personal contacts and local advertising.
All participants had a body mass index >25 kg·m⁻², were
nonsmokers, had no known history of CVD or diabetes (and
fasting glucose <6.0 mmol·L⁻¹ on screening), and were not
taking any medications known to affect lipid or glucose me-
tabolism. The study was conducted in accordance with the
principles of the Declaration of Helsinki and approved by the
College of Medical, Veterinary and Life Sciences Research
Ethics Committee at the University of Glasgow. All partici-
pants provided written informed consent.

Study Design

Participants each completed three 8-h experimental trials;
uninterrupted sitting (SIT), prolonged standing (PRO-Stand),
and intermittent standing (INT-Stand) in a randomized order,
with an interval of 1–2 wk between trials. Participants were
asked to weigh and record their food intake and refrain from
planned exercise (undertaking only the activities of normal
daily living) and alcohol on the 2 d preceding their first main
experimental trial and to replicate this for the 2 d preceding
subsequent trials. Physical activity and sedentary behavior
during these days was assessed using ActivPAL accelerome-
ters (PAL Technologies Ltd., Glasgow, United Kingdom). The
experimental protocol is shown in Figure 1 and described below.

Experimental Protocol

SIT trial. Participants arrived at the metabolic suite after
a 12-h overnight fast. They sat comfortably for 10 min, be-
fore two sequential 5-min expired air samples were collected
via a mouthpiece into a Douglas bag to calculate metabolic
rate and substrate utilization using indirect calorimetry (9).
The second of these samples was used as the baseline value.
A cannula was then inserted into an antecubital vein for re-
peated blood sampling, which was kept patent by flushing

![Figure 1](http://www.acsm-msse.org)
with saline throughout the day. A baseline fasting blood sample was drawn in K$_2$ ethylenediamine tetraacetic acid tube and placed immediately on ice. Participants were then given a standardized breakfast comprising a buttered bagel and a meal replacement drink (Complan Foods Ltd, UK) made up with whole milk, which provided 8 kcal energy per kg body mass (37% energy from fat, 49% carbohydrates, 14% protein) and was consumed within 10 min. Further blood samples were taken at 30, 60, 120, 180, and 240 min after breakfast. Four hours after breakfast, participants consumed a test lunch, which was identical to breakfast, and further blood samples were taken 30, 60, 120, 180, and 240 min after lunch (i.e., 270, 300, 360, 420, and 480 min after breakfast). Expired air samples for the determination of metabolic rate and substrate utilization were collected at 15-min intervals throughout the 8-h observation period. Participants sat comfortably (reading, watching TV, doing paperwork, and so on) throughout the observation period and were permitted to drink water throughout the day. Comfort breaks to the toilet (which was ~20 m from the metabolic investigation suite) were permitted.

**PRO-Stand trial.** This was identical to the SIT trial, except that in each 30-min period throughout the day, participants were asked to sit for 15 min and stand stationary for 15 min, so that in total, they stood for 4 h and sat for 4 h, with 16 sit-to-stand and 16 stand-to-sit transitions over the 8-h observation period. All blood samples were taken during 15-min sitting periods.

**INT-Stand trial.** This was identical to the SIT trial, except that in each 30-min period, participants sat for 5 min, then undertook 10 cycles of standing for 90 s followed by sitting for 30 s (20 min in total), then sat for 5 min. Thus, they stood for 15 min and sat for 15 min every 30 min, but the standing occurred in 10 × 90-s blocks, rather than a single 15-min block. Thus, over the 8-h observation period, they stood for 4 h and sat for 4 h, with 160 sit-to-stand and 160 stand-to-sit transitions. All blood samples were taken during the 10 min of continuous sitting in each 30-min period.

**Calculation of Energy Expenditure and Substrate Utilization**

Energy expenditure and energy substrate utilization were calculated using indirect calorimetry (9). For these calculations, urinary nitrogen excretion was assumed to be 0.11 mg·kg$^{-1}$·min$^{-1}$ throughout each trial, based on data from previous studies in the literature (8,21).

**Blood Processing and Analysis**

Venous blood samples were collected into K$_2$ ethylenediamine tetraacetic acid tubes, placed immediately on ice, and centrifuged to separate plasma within 15 min. Plasma glucose concentrations were measured immediately using a benchtop analyzer (YSI 2300 STAT Plus™ Glucose and Lactate Analyser; YSI (UK) Ltd.). The remaining of plasma was stored at −80°C for later analysis. Insulin concentration was determined using a commercially available enzyme linked immunosorbent assay (Mercodia AB, Uppsala, Sweden). TG concentrations were determined by commercially enzymatic colorimetric kit (Randox Laboratories, Crumlin, UK) using an autoanalyzer (ILab™ 600; Clinical Chemistry System, Instrumentation Laboratory, USA).

**Power Calculation**

As the most consistent association between frequency of sedentary breaks and health outcomes related to adiposity variables (3,11,12), we primarily based our sample size on the number of participants needed to detect a difference in overall energy expenditure over the observation period. Our previous data had shown that the within-person SD for difference in resting oxygen uptake was 6.1% (6). We assumed that the within-person SD for differences in energy expenditure between trials here would be similar. Accordingly, we calculated that 10 participants would enable detection of approximately 6% difference in energy expenditure between trials with 80% power at $P < 0.05$. In addition, based on our earlier observations that the within-person SD for postpran
dial glucose, TG, and insulin responses were 3.4%, 10.1%, and 22.9%, respectively (10), our sample would enable detection of respective differences between trials of approximately 3%, 10%, and 23%, in glucose, TG, and insulin responses.

**Statistical Analysis**

Statistical analyses were performed using Statistica (Version 10, StatSoft, Inc.) and Minitab (Version 14, Minitab Inc.). Data were tested for normality using the Anderson–Darling normality test, and where necessary, data were logarithmically transformed before statistical analysis. The area under curve (AUC), calculated using the trapezium rule, was used as a summary measure of the postprandial responses. Comparisons between trials were made using repeated-measures ANOVA, with post hoc Fisher LSD tests used to identify where any differences lay. Cohen $d$ effect sizes were calculated to describe the magnitude of differences between trials (>0.8 large, 0.5–0.8 medium, <0.5 small, <0.2 trivial) (2).

Data are presented as mean ± SEM unless otherwise stated, and $P < 0.05$ was considered significant.

**RESULTS**

**Baseline values.** There were no differences in body mass, rates of energy expenditure, fat oxidation or carbohydrate oxidation between trials. Table 1 shows the baseline values. Further tests of differences were performed on log-transformed data. There were no significant differences in any variable between trials.

### Table 1. Baseline values in the fasted state in the three experimental conditions.

<table>
<thead>
<tr>
<th>Condition</th>
<th>SIT</th>
<th>PRO-Stand</th>
<th>INT-Stand</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body mass (kg)</td>
<td>89.9 ± 3.4</td>
<td>89.8 ± 3.4</td>
<td>89.7 ± 3.3</td>
</tr>
<tr>
<td>Energy expenditure (kJ·min$^{-1}$)</td>
<td>5.61 ± 0.18</td>
<td>5.39 ± 0.16</td>
<td>5.41 ± 0.19</td>
</tr>
<tr>
<td>Fat oxidation (g·min$^{-1}$)</td>
<td>0.08 ± 0.01</td>
<td>0.07 ± 0.01</td>
<td>0.08 ± 0.01</td>
</tr>
<tr>
<td>Carbohydrate oxidation (g·min$^{-1}$)</td>
<td>0.10 ± 0.02</td>
<td>0.11 ± 0.01</td>
<td>0.08 ± 0.01</td>
</tr>
<tr>
<td>Plasma glucose (mmol·L$^{-1}$)</td>
<td>5.2 ± 0.2</td>
<td>5.2 ± 0.1</td>
<td>5.4 ± 0.2</td>
</tr>
<tr>
<td>Plasma insulin (mU·L$^{-1}$)</td>
<td>7.4 ± 0.9</td>
<td>8.1 ± 1.4</td>
<td>8.8 ± 1.7</td>
</tr>
<tr>
<td>Plasma TG (mmol·L$^{-1}$)</td>
<td>1.18 ± 0.24</td>
<td>1.24 ± 0.14</td>
<td>1.16 ± 0.21</td>
</tr>
</tbody>
</table>

Values are mean ± SEM, $n = 10$.

*Statistics performed on log-transformed data. There were no significant differences in any variable between trials.
oxidation, or plasma glucose, insulin or TG concentrations between the three experimental conditions in the fasted state, before the interventions were commenced (Table 1), indicating that control of lifestyle in the days preceding the trials was sufficient to ensure that the baseline metabolic state in all trials was the same.

**Energy expenditure and substrate utilization during the interventions.** Energy expenditure and substrate utilization over the 8-h observation period are shown in Figure 2, with summary data for these responses shown in Table 2. Compared with the SIT trial, total energy expenditure over the 8 h was 320 ± 62 kJ (10.7% ± 2.0%) higher in the PRO-Stand trial, and 617 ± 76 kJ (20.4% ± 2.3%) higher in the INT-Stand trial: energy expenditure in the INT-Stand trial was 296 ± 78 kJ (9.0% ± 2.3%) higher than the PRO-Stand trial (all P < 0.001). The Cohen d effect sizes for all of these differences were large. Total fat oxidation over the observation period was 7.1 ± 1.9 g (20.2% ± 6.7%) greater in the INT-Stand trial than the SIT trial (P < 0.01), with a large effect size, but the 2.5 ± 2.2 g (7.6% ± 5.4%) difference in fat oxidation between the PRO-Stand and SIT trials was not statistically significant, and the effect size was small. Total fat oxidation was 4.6 ± 2.6 g (13.7% ± 7.6%) greater in the INT-Stand trial than the PRO-Stand trial (P = 0.06), with a large effect size. Compared with the SIT trial, total carbohydrate oxidation was 14.4 ± 5.2 g (30.8% ± 12.6%) higher in the PRO-Stand trial (P < 0.05), and 22.0 ± 6.0 g (44.0% ± 12.8%) higher in the INT-Stand trial (P < 0.01). The difference in carbohydrate oxidation between the INT-Stand and PRO-Stand trials (7.6 ± 7.8 g; 15% ± 12.4%) was not statistically significant and had a small effect size. In post hoc observations, it became apparent that the pattern of substrate utilization between trials differed between the postbreakfast (0–240 min) and postlunch (240–480 min) postprandial observation periods. We therefore decided to analyze these periods separately. In the postbreakfast period, 19.6 ± 1.5 g, 20.1 ± 1.5 g, and 25.0 ± 1.8 g of fat were oxidized in the SIT, PRO-Stand, and INT-Stand trials, respectively. Fat oxidation over this period was significantly higher in the INT-Stand trial than the other two trials (P < 0.001 for both), but did not differ significantly between the SIT and PRO-Stand trials (P = 0.68). In contrast, fat oxidation over the postlunch period did not differ significantly between any of the trials (SIT, 18.9 ± 1.4 g; PRO-Stand, 20.8 ± 1.8 g; INT-Stand, 20.5 ± 1.4 g). In the postbreakfast period, carbohydrate oxidation was significantly higher than SIT (31.2 ± 3.2 g) in the PRO-Stand stand trial (41.0 ± 3.3 g) (P = 0.007) and tended to be higher than SIT in the INT-Stand trial (38.0 ± 2.7 g) (P = 0.055), but did not differ significantly between the PRO-Stand and INT-Stand trials (P = 0.36). Carbohydrate oxidation was significantly higher in the INT-Stand trial (48.1 ± 3.6 g) than both the SIT trial (32.8 ± 3.1 g) (P = 0.002) and the PRO-Stand trial (37.4 ± 3.4 g) (P = 0.02) but did not differ significantly between the SIT and PRO-Stand trials (P = 0.30). Thus, the increment in energy expenditure in the INT-Stand trial over the PRO-Stand trial was largely mediated by an increase in fat oxidation in the postbreakfast period and an increase in carbohydrate oxidation in the postlunch period.

**Blood glucose, insulin, and TG responses during the interventions.** Blood glucose, insulin, and TG responses over the 8-h observation period are shown in Figure 3, with summary data for these responses shown in Table 2. There were no significant differences between the three trials in glucose, insulin, and TG responses. The effect sizes for the differences between trials in the insulin and TG responses were trivial to small. Although not statistically significant, a medium effect size was observed when comparing the
glucose response in the PRO-Stand trial with the SIT trial ($P = 0.16$) and the INT-Stand trial ($P = 0.48$).

**DISCUSSION**

The main finding of the this study is that increasing the frequency of breaks in sedentary time, while keeping total sedentary time constant, increased energy expenditure and fat oxidation over an 8-h postprandial observation period. This is the first time that an independent effect of the number of sedentary breaks on day-long metabolic responses has been demonstrated, and these findings provide an explanation for the association between frequency of sedentary breaks and adiposity observed in the epidemiological data (3,11,12).

A number of studies have reported that energy expenditure during quiet standing is 2%-33% higher than observed during sitting (16,19,24,27). The present findings are consistent with this. In the PRO-Stand condition—where participants alternated 15 min of sitting with 15 min of standing throughout the observation period—energy expenditure was 10.7% higher than the SIT condition, an absolute increase in expenditure of 320 kJ over 8 h. In the INT-Stand condition—where participants undertook ten 1.5-min bouts of standing in every half-hour—there was a further increase in energy expenditure of 9.0% (296 kJ), despite participants sitting and standing for the same total duration in both trials. To put these figures into context, if participants replicated the protocol in the trial for 4 wk, energy expenditure in the PRO-Stand and INT-Stand conditions would be 9.0 MJ and 17.3 MJ higher than the SIT condition. Assuming no change in energy intake, this would equate to approximately 1.2 kg weight loss, relative to SIT, in the PRO-Stand condition and approximately 2.2 kg weight loss in the INT-Stand condition. Interestingly, a large proportion of the increase in energy expenditure from increasing the frequency of sedentary breaks was in fat oxidation. Participant oxidized 7.1 g more fat and 7.7 g more carbohydrate in the INT-Stand compared with the PRO-stand trials, which equates to 277 kJ increased fat and 131 kJ increased carbohydrate oxidation in terms of energy. This disproportionate increase in fat oxidation with increasing sit-to-stand transitions may have implications for the long-term regulation of body weight because high levels of fat oxidation have been shown to be protective against long-term weight gain, independent of metabolic rate (20,26,31).

The increased energy expenditure in the INT-Stand compared with the PRO-Stand condition was likely mediated by the increased concentric and eccentric muscular activity associated with the larger number of sit-to-stand transitions. A recent study by Júdice and colleagues (16) attempted to quantify the energy expended in sit-to-stand transitions per se by comparing the energy expended over 10 min when participants stood and sat down immediately once per minute for 10 min with 10 min of sitting, reporting the energy cost of a single sit-to-stand transition was approximately 0.02 kJ kg$^{-1}$ body mass. In the present study, participants stood for 4 h and sat for 4 h, with 16 sit-to-stand (and 16 stand-to-sit) transitions in the PRO-Stand condition and stood and sat for the same duration but with 160 sit-to-stand (and 160 stand-to-sit) transitions in the INT-Stand condition. Thus, the 296-kJ difference in energy expenditure represents the energy expended in 144 sit-to-stand/stand-to-sit transitions, i.e., ~2 kJ per transition or ~0.02 kJ kg$^{-1}$, in line with Júdice et al.’s calculations. Thus, the present findings suggest that the “snapshot” calculation of the energy expended during short-duration sit-to-stand transitions in the fasted state can be extended over the course of a day under “real-life” postprandial conditions.

We found no significant effects of either prolonged or intermittent standing breaks on postprandial incremental glucose, insulin, or TG responses. The effect sizes for the difference in incremental insulin and TG responses between trials were trivial to small. Thus, the lack of a statistically significant effect of prolonged or intermittent standing on these responses appears to reflect the absence of a physiologically important influence of the standing interventions on these outcomes, rather than a lack of statistical power to detect a clinically relevant effect. The postprandial glucose response was approximately 3% lower in the PRO-Stand trial, but approximately 1% higher in the INT-Stand than the SIT trial. Neither of these differences were statistically significant, but there was a medium effect size for the difference between the PRO-Stand and SIT conditions, suggesting that this difference could conceivably be physiologically relevant, but that the study did not have sufficient statistical power to detect it. However, although we cannot definitively exclude

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**TABLE 2. Summary postprandial responses over the 8-h postprandial observation period in the three experimental conditions.**

<table>
<thead>
<tr>
<th></th>
<th>SIT</th>
<th>PRO-Stand</th>
<th>INT-Stand</th>
<th>SIT vs PRO-Stand</th>
<th>SIT vs INT-Stand</th>
<th>PRO-Stand vs INT-Stand</th>
<th>Effect size</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total energy expenditure (kJ)</td>
<td>2980 ± 78</td>
<td>3301 ± 112</td>
<td>3597 ± 139</td>
<td>1.64***</td>
<td>2.56***</td>
<td>1.19***</td>
<td></td>
</tr>
<tr>
<td>Total fat oxidation (g)</td>
<td>38.4 ± 2.7</td>
<td>40.9 ± 2.9</td>
<td>45.5 ± 3.0</td>
<td>0.96</td>
<td>1.19***</td>
<td>0.54***</td>
<td></td>
</tr>
<tr>
<td>Total carbohydrate oxidation (g)</td>
<td>64.1 ± 5.9</td>
<td>78.4 ± 5.6</td>
<td>86.1 ± 5.5</td>
<td>0.87***</td>
<td>1.17***</td>
<td>0.31</td>
<td></td>
</tr>
<tr>
<td>Plasma glucose AUC (mmol L$^{-1}$ h$^{-1}$)</td>
<td>47.3 ± 1.4</td>
<td>46.0 ± 1.5</td>
<td>47.9 ± 1.9</td>
<td>0.78</td>
<td>−0.19</td>
<td>−0.61</td>
<td></td>
</tr>
<tr>
<td>Plasma insulin AUC (mU L$^{-1}$ h$^{-1}$)</td>
<td>365 ± 47</td>
<td>335 ± 60</td>
<td>323 ± 46</td>
<td>0.23</td>
<td>0.24</td>
<td>0.06</td>
<td></td>
</tr>
<tr>
<td>Plasma TG AUC (mmol L$^{-1}$ h$^{-1}$)</td>
<td>14.3 ± 2.4</td>
<td>15.0 ± 1.6</td>
<td>13.5 ± 1.9</td>
<td>−0.17</td>
<td>0.23</td>
<td>0.38</td>
<td></td>
</tr>
</tbody>
</table>

Values are mean ± SEM, n = 10.

*Statistics performed on log-transformed data.

**P < 0.001.

*P < 0.01.

***P = 0.06.

****P < 0.05.
a potential glucose-lowering effect of PRO-Stand—albeit a relatively modest one—it is intriguing that a similar pattern was not observed for INT-Stand, where the glucose response was not lower than the SIT condition. This could conceivably be a consequence of the concentric and eccentric muscular activity associated with the repeated sit-to-stand and stand-to-sit transitions in INT-Stand condition, which are essentially equivalent to performing 160 bodyweight squats over the observation period. Thus, the INT-Stand condition could be considered analogous to a session of resistance exercise spread over a number of hours. Although resistance exercise training programs have been shown to improve insulin sensitivity and reduce glucose concentrations over the long term, particularly in people with type 2 diabetes (15), there is evidence of a transient increase in plasma glucose concentrations in response to resistance exercise (7,17). Thus, it is conceivable that an acute muscle contraction-mediated glucose-raising effect could have offset any potential glucose-lowering effect of standing per se in the INT-Stand condition. Further work is therefore needed to confirm whether this hypothesis is correct and, importantly, to determine whether over the longer-term adaptations in skeletal muscle in response to such repeated contractions could elicit favorable effects of high frequency breaks in sedentary behavior on glucose metabolism.

A number of previous reports have demonstrated that breaking up continuous sitting time with ≤3-min intervals of light or moderate intensity physical activity every 20–30 min can reduce postprandial glucose, insulin, and TG concentrations (4,18,22,23). Studies evaluating the effects of breaking up sitting with static standing on these postprandial blood responses have had more mixed results. Henson and colleagues (14) recently reported that in postmenopausal women (mean age, 66 yr) with impaired glucose regulation, breaking up sitting time with 5 min of quiet standing every 30 min over a 7.5-h postprandial observation period reduced the glucose and insulin incremental AUC by 34% and 20%, respectively, with no significant effect on the postprandial TG response. In an intervention by Thorp and colleagues (28), in which overweight/obese middle-age participants (mean age, 48 yr) performed normal work tasks over an 8-h workday either seated or alternating 30 min of sitting and 30 min of standing using a sit-to-stand workstation, the incremental glucose response was 11% lower in the sit-to-stand condition, but there was no significant effect of the intervention on insulin or TG responses. In contrast, Bailey and Locke (1) recently reported that in young (mean age, 24 yr) nonobese adults, breaking up prolonged sitting with 2 min of standing every 20 min had no effect on postprandial glucose or TG responses over a 5-h period, but breaking up sitting with 2-min breaks of light ambulation (3.2 km h⁻¹ walking) every 20 min reduced glucose (but not TG) responses by approximately 16%. In the present study, we found no significant effects of either prolonged or intermittent standing breaks on postprandial incremental glucose, insulin, or TG responses in our group of relatively young (mean age, 33 yr), overweight/obese, normoglycemic men, although we could not definitely exclude a modest potential glucose-lowering effect in the PRO-Stand condition. Thus, no intervention study has observed a statistically significant acute effect of standing on postprandial insulin or TG concentrations in normoglycemic adults—in contrast to the findings of studies where sitting was broken up by light to moderate physical activity (4,18,22,23)—suggesting that a greater stimulus than
standing is needed to positively alter these responses in young to middle-age adults without preexisting dysglycemia. Observational data from AusDiab study of middle-age and older adults (mean age, 57.9 yr) reported that reallocation of 2 h of sitting with 2 h of standing per day was associated with approximately 2% lower fasting glucose and approximately 11% lower fasting TG concentration (13). Although the causality and direction of these associations cannot be confirmed from such a cross-sectional analysis, these data do raise the possibility that metabolic benefits of standing may be more clearly observed in interventions undertaken in an older population. Further study is therefore needed to determine 1) whether interventions to replace sitting with standing improve postprandial glucose, insulin, and TG metabolism in older individuals; and 2) whether interventions to increasing the frequency of interruptions to sitting might enhance the previously reported benefits of standing breaks on postprandial glucose, insulin, and TG metabolism in those with glucose dysregulation (14).

In conclusion, this study was designed to determine whether, in principle, the number of transitions between sitting and standing could influence postprandial metabolic responses, independent of total time spent sitting and standing. Our data clearly indicate that the frequency of interruptions to sedentary time has a marked independent influence on metabolic rate, which is likely due to the increased energy expended due to muscular contractions in the sit-to-stand and stand-to-sit transitions. Each additional sit-to-stand transition cycle expended approximately 2 kJ energy, which can help explain the epidemiological observation between sedentary breaks and adiposity (3,11,12). Although our INT-Stand protocol, with 20 sit-to-stand transition cycles per hour is clearly impractical to implement in “real-world” settings, these findings can help inform the design of practical interventions to reduce sedentary behavior. For example, performing four sit-to-stand transition cycles per hour (i.e., standing then sitting once every 15 min) over the course of the waking day would lead to approximately 100–120 kJ of additional daily energy expenditure over and above the increment in metabolic rate elicited by standing per se. We found no evidence that standing, either in prolonged bouts or intermittent bouts could influence postprandial insulin or TG responses in these normoglycemic participants (although we cannot definitively exclude a potential modest glucose lowering effect of prolonged standing from the present data), suggesting that it may be necessary to break up sitting with activities of greater intensity than quiet standing to positively influence postprandial metabolism in relatively young, normoglycemic overweight/obese men.

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