The effect of interrupting prolonged sitting time with short, hourly, moderate-intensity cycling bouts on cardiometabolic risk factors in healthy, young adults

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Altenburg TM, Rotteveel J, Dunstan DW, Salmon J, Chinapaw MJ. The effect of interrupting prolonged sitting time with short, hourly, moderate-intensity cycling bouts on cardiometabolic risk factors in healthy, young adults. J Appl Physiol 115: 1751–1756, 2013. First published October 17, 2013; doi:10.1152/japplphysiol.00662.2013.—Although detrimental associations of sitting time and health indicators have been observed in young adults, evidence of pathophysiological mechanisms is lacking. Therefore, this study tested the hypothesis that the acute cardiometabolic effects of prolonged sitting can be compensated by hourly interruptions to sitting in healthy, young adults. Additionally, leg muscle activation during sitting and moderate-intensity physical activity interruptions was assessed. Eleven apparently healthy adults (18–24 yr; five men/six women) participated in this randomized, crossover study, involving two experimental conditions: 1) 8 h prolonged sitting and 2) 8 h of sitting, interrupted with hourly, 8-min, moderate-intensity cycling exercise bouts. In both conditions, participants consumed two standardized, high-fat mixed meals after 1 and 5 h. Capillary blood samples were collected hourly during each 8-h experimental condition. Muscle activity was measured using electromyography. Muscle activity during cycling was seven to eight times higher compared with rest. Postprandial levels of C-peptide were significantly lower (unstandardized regression coefficient \( \beta = 0.19; \) confidence interval \([-0.35; -0.03]\); \( P = 0.017 \)) during interrupted sitting compared with prolonged sitting. Postprandial levels of other cardiometabolic biomarkers (e.g., glucose, triglycerides, cholesterol) were not significantly different between conditions. Hourly physical activity interruptions in sitting time, requiring a muscle activity of seven to eight times the resting value, led to an attenuation of postprandial C-peptide levels but not for other cardiometabolic biomarkers compared with prolonged sitting in healthy, young adults. Whether this acute effect transfers to chronic effects over time is unknown.

Sedentary behavior; prolonged sitting; interrupted sitting; cardiometabolic risk

Sedentary behavior [activities performed sitting during waking hours that typically require low-energy expenditure, i.e., 1–1.5 times higher than rest (30)] has been identified as an important and independent lifestyle risk factor for type 2 diabetes mellitus (T2DM) and cardiovascular disease (CVD) in adults (14, 24, 25). A number of reviews have been published about the association of sedentary behavior (e.g., television time, screen time, total sitting time) and cardiometabolic health in adults (12, 28, 34).

Recent population-based studies suggest that prolonged, unbroken sitting times, assessed by objective measures, such as accelerometry, exert detrimental effects on cardiometabolic biomarkers, irrespective of the total time spent sedentary and total time spent in moderate- to vigorous-intensity physical activity. For example, cross-sectional research has shown that adults with a less-frequent number of interruptions in sedentary time (prolonged sedentary time), on an average day, have a poorer cardiometabolic risk profile, such as elevated waist circumference, triglycerides, and 2-h plasma glucose, compared with those who had more frequent interruptions (breaks) in their daily sedentary time (15).

A recent experimental study about the acute effects of breaking up prolonged sitting in overweight/obese adults demonstrated that brief interruptions (i.e., 2-min walking interruptions every 20 min) to prolonged sitting significantly reduced postprandial glucose and insulin levels compared with prolonged sitting (5). Importantly, these results were observed, irrespective of the activity intensity (i.e., light or moderate intensity) of the interruptions (5). Similarly, regularly (i.e., every 30 min) breaking up prolonged sitting with short (i.e., 1-min, 40-s) bouts of physical activity significantly reduced postprandial glucose and insulin levels in healthy, normal-weight adults (26). These findings support the hypothesis that the loss of contractile stimulation in weight-bearing muscles may lead to a prolonged time in which cellular metabolism substrates are present in the vascular compartments, underpinning the biological consequences of prolonged sitting (2, 13). However, in contrast to the hypothesized mechanism, breaking up prolonged sitting with regular, short bouts of physical activity did not reduce postprandial triglycerides in healthy, normal-weight adults (26).

To date, it is unclear whether less-frequent physical activity interruptions (i.e., less than every 20/30 min) can exert similar beneficial effects on cardiometabolic biomarkers, thereby approximating the recommendation of physical activity in short sessions (1a). It is hypothesized that the loss of contractile stimulation in weight-bearing muscles may underpin the detrimental consequences of prolonged sitting (2, 13). Therefore, we aimed to examine whether hourly physical-activity interruptions in sitting time would attenuate possible detrimental, acute effects on postprandial cardiometabolic biomarkers compared with prolonged, uninterrupted time spent sitting. Since
postprandial lipid responses to moderate- to vigorous-intensity (continuous and intermittent) exercise bouts are demonstrated to occur 13–18 h after completion of the exercise bout (9, 16, 20, 21, 26), changes in postprandial lipids, induced by interrupting prolonged sitting, might not be expected within 1 day. Therefore, postprandial indicators of glucose metabolism were defined as primary outcome measures, whereas postprandial indicators of lipid metabolism were defined as secondary outcomes. We assessed the amount of leg-muscle activation corresponding to sitting and moderate-intensity physical-activity interruptions compared with resting muscle activity.

METHODS

Participants

Five men and six women, aged 18–24 yr, participated in this study. Participants were recruited through distribution of flyers, announcements on university websites, and Dutch recruitment websites. Participants were included if they were of normal weight and apparently healthy, were Dutch or English speaking, and signed an informed consent. Exclusion criteria were major illness/injury and physical problems that may limit the ability to perform the experiment. Participants were screened by a health-check questionnaire, including questions about participants’ medical history (e.g., heart/kidney/joint/muscle/asthmatic complaints, coagulation problems, chest pain). Moreover, they were requested to refrain from any moderate- to vigorous-intensity physical activity for at least 72 h before the experiment and to avoid drinking alcohol and smoking for at least 24 h before the experiment.

Study Design

This crossover study included two acute experimental conditions of 8 h duration in a laboratory setting, was approved by the Medical Ethics Committee of the VU University Medical Center in Amsterdam, and was in accordance with the Declaration of Helsinki. The experimental conditions were: 1) prolonged sitting (SIT) and 2) sitting with hourly interruptions of 8-min moderate-intensity cycling bouts (SIT-CYCLE). To eliminate potential carryover effects, there was a minimum washout of 7 days between each condition. The order of the experimental conditions was assigned randomly.

Procedures

Experimental day protocol. After an 8-h fast, participants visited the research room. During the first visit, the informed-consent document and a health history were completed, and baseline measurements [blood measurements, anthropometrics, and muscle activity (details below)] were collected [time (t) = 0]. The participants then sat quietly for 1 h to achieve a “steady state.” Subsequently, participants consumed a standardized, high-fat mixed meal (for details, see Standardized meals below), which they were requested to drink within 10 min. After consuming the standardized meal, in the SIT condition, participants remained seated in a comfortable, reclining lounge chair for the next 7 h. They were allowed to use the computer with a DVD player and internet access and reading materials. During the sitting, participants were instructed to minimize excessive movement but were allowed to visit the toilet. In the SIT-CYCLE condition, 1 h after the initial 1-h steady-state period and the standardized meal, participants completed an 8-min moderate-intensity cycling bout every hour, approximating the recommendation of physical activity in short sessions (1a). This procedure was repeated five times, resulting in a total cycling duration of 48 min. All participants consumed a second standardized, high-fat mixed meal after 5 h of sitting (t = 5). For the SIT-CYCLE condition, the cycling bout was performed directly after the meal was consumed. Blood samples were collected hourly during each 8-h experimental condition (i.e., nine blood samples). In the SIT-CYCLE condition, blood samples were taken just before the onset of the cycling bouts to eliminate the acute effects of the moderate-intensity cycling bout on measures.
Muscle activity. Since it is hypothesized that the loss of contractile stimulation in weight-bearing muscles underlies the consequences of prolonged sitting (2, 13), we measured muscle activity of the rectus femoris (RF), vastus lateralis, and gastrocnemius (GAS) muscles using electromyography (EMG). Previous studies have shown that EMG amplitude is a reliable measure of muscle activation (32, 35), both during short- and long-term intervals (18). After shaving and cleaning the skin with 70% ethanol, two electrodes (lead-off area 1.0 cm²; Blue Sensor; Ambu, Ølstykke, Denmark) were placed on the belly of each muscle in a bipolar configuration (interelectrode distance of 25 mm). A reference electrode was placed on the patella.

At the beginning of each experimental condition (during baseline measurements), participants were asked to lie down for 10 min to obtain resting EMG values. EMG recordings of 30 min were made three times during sitting (both conditions; after 1, 3, and 5 h of sitting), and 8-min EMG recordings were made during all cycling bouts (SIT-CYCLE condition). EMG signals were amplified (×1,000) with a biosignal amplifier (0.01–10 kHz; input impedance 110 MΩ; g.tec), analog-to-digital converted with a Simultaneous Sampling AtoD board (PCI-6143; National Instruments, Austin, TX), digitized (10 kHz), band-pass filtered (10–400 Hz), and stored with the torque signal on a computer disk. Rectified EMG signals during the sitting periods and the cycling bouts were averaged over a time period of 20 and 6 min, respectively (e.g., time interval in which rectified EMG was stable), and expressed as percentage of the resting rectified EMG. However, due to noise caused by movement of the EMG wires, we did not attain reliable EMG data for all participants and during all measures. Therefore, participant numbers varied between conditions and muscles, and signals were sometimes averaged over a shorter time period (range: 5–20 min during sitting periods and 2–6 min during cycling bouts).

Statistics

Descriptive participant characteristics [median (minimum/maximum)] were calculated at baseline. Data were not distributed normally, and Wilcoxon signed-rank tests were used to test for differences between conditions at baseline and also to test for differences between EMG during sitting and rest and during rest and cycling. The average of the blood samples at t = 0 and t = 1 (i.e., at the beginning and the end of the 1st h) was considered as steady state and used as baseline blood sample.

Generalized estimating equations (GEE) were used to assess the difference between prolonged and interrupted sedentary time for each cardiometabolic biomarker. All assumptions of the GEE models were met, indicating that GEE analysis was appropriate for the data of the current study. This longitudinal analysis technique was used to correct for dependency within the repeated measures (i.e., eight blood samples and two conditions) for each participant. Since we used a crossover design in this study, we did not adjust for demographic variables, such as age, gender, and weight status. All statistic procedures were performed using SPSS software (version 18.0). Statistical significance was set at P < 0.05.

RESULTS

Table 1 shows the baseline participant characteristics. Baseline steady-state blood values for triglycerides, glucose, total cholesterol, HDL cholesterol, LDL cholesterol, and C-peptide were not different between the SIT and SIT-CYCLE conditions. The intensity of the cycling bouts in the SIT-CYCLE condition was, on average, 52.0 ± 3.2% HRR, indicating that the physical activity interruptions were performed at the intended, moderate intensity. Borg RPE scores were, on average, 11.2 (SD 1.6), ranging from seven to 15.

Figure 1 demonstrates levels of all cardiometabolic biomarkers throughout 1 day of prolonged sitting and 1 day of interrupted sitting. GEE analysis for the 7-h period (e.g., including the response to both standardized meals) revealed that C-peptide levels were significantly higher during prolonged vs. interrupted sitting (unstandardized regression coefficient = −0.19; confidence interval = [−0.35; −0.03]; P = 0.017; Table 2). Levels of triglycerides, total cholesterol, HDL cholesterol, LDL cholesterol, and glucose were not different between the SIT and the SIT-CYCLE conditions (Table 2). GEE analysis for the 4-h response (e.g., including the response to the first standardized meal only) revealed similar results.

Average rectified EMG of the RF and GAS muscles during sitting was not significantly different from resting EMG (Table 3). During the moderate-intensity cycling interruptions, averaged rectified EMG was seven times the resting EMG for the RF muscle (P < 0.05) and eight times the resting EMG for the GAS muscle (P < 0.05).

DISCUSSION

Relative to prolonged sitting, brief, hourly moderate-intensity, 8-min physical activity interruptions in sitting time were associated with significantly lower postprandial plasma levels of C-peptide but not of other cardiometabolic biomarkers in healthy young adults. EMG, during the moderate-intensity physical activity interruptions, was seven to eight times the resting values.

The lower levels of postprandial C-peptide, reflecting reduced endogenous insulin secretion (27, 37), during interrupted sitting compared with prolonged sitting, are comparable with previous findings in overweight and obese adults (5) and in healthy, normal-weight adults (26). Dunstan et al. (5) found that interrupted (i.e., 2-min bouts of walking every 20 min) sitting time reduces postprandial insulin levels by 23% in overweight/obese adults. Similarly, Peddie et al. (26) demonstrated that breaking up prolonged sitting (i.e., 1-min, 40-s bouts of walking every 30 min) reduced postprandial insulin levels by 26% in healthy, normal-weight adults. The present study shows that interrupting prolonged sitting every hour may also be important for acute health outcomes in young and

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Table 1. Descriptive participant characteristics [median (25th–75th percentile); n = 11]

| Age, years | 21.4 (19.5–23.1) |
| Gender, % men | 45 |
| Height, cm | 175.8 (169.5–184.8) |
| Weight, kg | 71.8 (65.4–75.0) |
| BMI, kg/m² | 23.2 (20.1–26.1) |
| Waist circumference, cm | 79.7 (71.7–82.9) |

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PSIT-CYCLE compared with SIT condition

*Significant higher levels of C-peptide for SIT and SIT-CYCLE. Standardized, high-time biomarkers between prolonged and interrupted sedentary time.

Table 2. Difference (B and 95% CI) in cardiometabolic biomarkers between prolonged and interrupted sedentary time

<table>
<thead>
<tr>
<th>Biomarker</th>
<th>B [95% CI]</th>
</tr>
</thead>
<tbody>
<tr>
<td>Triglycerides, mmol/l</td>
<td>0.19 (0.18, 0.57)</td>
</tr>
<tr>
<td>Total cholesterol, mmol/l</td>
<td>-0.05 (0.28, 0.18)</td>
</tr>
<tr>
<td>HDL cholesterol, mmol/l</td>
<td>-0.04 (-0.11, 0.03)</td>
</tr>
<tr>
<td>LDL cholesterol, mmol/l</td>
<td>0.11 (0.14, 0.16)</td>
</tr>
<tr>
<td>Glucose, mmol/l</td>
<td>-0.03 (-0.17, 0.11)</td>
</tr>
<tr>
<td>C-peptide, mmol/l</td>
<td>-0.19 (-0.35, -0.03)*</td>
</tr>
</tbody>
</table>

B, unstandardized regression coefficient; CI, confidence interval. Note that a negative B indicates a lower blood level for the SIT-CYCLE condition compared with the SIT condition. *Significant difference between conditions (P < 0.02).

In line with the findings of Peddie et al. (26), we did not observe any differences in postprandial triglycerides during sitting with hourly physical-activity interruptions compared with prolonged sitting. In addition, we did not observe any differences in postprandial total cholesterol, HDL cholesterol, and LDL cholesterol during interrupted sitting compared with prolonged sitting. These findings are in contrast with one of the hypothesized mechanisms underlying the biological consequences of prolonged sitting, suggesting that loss of local contractile stimulation in weight-bearing muscles leads to the suppression of skeletal muscle lipoprotein lipase (LPL) activity (2, 13). The acute loss of LPL activity at the vascular endothelium impairs several aspects of lipid metabolism (23) and may contribute to cardiometabolic risk over sustained periods of time (i.e., years or indeed, a lifetime). In our study population, the standardized, high-fat mixed meals induced only slight increases in triglycerides, total cholesterol, HDL cholesterol, and LDL cholesterol, possibly reflecting the relatively “metabolically healthy” profile of our participants. This may be

Table 3. Rectified EMG as percentage of resting EMG during prolonged sitting (SIT) and during sitting with hourly physical activity interruptions (SIT-CYCLE; n = 8)

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Condition</th>
<th>EMG during Sitting</th>
<th>EMG during Cycling</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rectus femoris</td>
<td>SIT</td>
<td>1.74 ± 2.02</td>
<td>NA</td>
</tr>
<tr>
<td></td>
<td>SIT-CYCLE</td>
<td>0.90 ± 0.27</td>
<td>7.14 ± 2.96*†</td>
</tr>
<tr>
<td>Gastrocnemius</td>
<td>SIT</td>
<td>1.54 ± 1.68</td>
<td>NA</td>
</tr>
<tr>
<td></td>
<td>SIT-CYCLE</td>
<td>1.49 ± 0.77</td>
<td>8.14 ± 4.50*‡</td>
</tr>
</tbody>
</table>

EMG, electromyography. *Significantly different from 1.0 (i.e., mean resting EMG value); †n = 7; ‡n = 6.
one explanation for the lack of significant effects of prolonged vs. interrupted sitting on these parameters. A second possible explanation might be that longer uninterrupted sitting time than what was used (>$8 \text{ h}$) may be needed to detect significant detrimental changes in this measure. Previous studies about the effects of exercise bouts on postprandial triglycerides demonstrated that postprandial lipid responses generally occur $>13 \text{ h}$ after completing moderate- to vigorous-intensity (continuous or intermittent) exercise (9, 16, 20, 21, 26). Additionally, skeletal muscle lipoprotein lipase activity is thought to peak $>8 \text{ h}$ after exercise (31).

Stephens et al. (33) tested the hypothesis that the negative health effects of sitting may be partly due to a positive energy balance. They demonstrated that 1 day of sitting considerably reduced insulin action relative to continuous standing/light ambulation; however, this effect was minimized but not prevented when energy intake was reduced to match expenditure. Additionally, when controlling for sitting time, physical exercise, and daily energy expenditure, Duvivier et al. (6) demonstrated that the adverse effects of sitting on insulin sensitivity and plasma lipids could not be compensated by 1 h of daily continuous physical activity. Due to these distinctive differences in study design and specific research questions, the results of our study cannot be compared with the studies mentioned above. Additionally, another major distinction is that the participants in our study were assessed while in a postprandial state (i.e., throughout 1 day of prolonged and 1 day of interrupted sitting), whereas the participants in the studies of Stephens et al. (33) and Duvivier et al. (6) were assessed in a fasted state (i.e., the morning after the experimental conditions).

Activity of the RF muscle during sitting varied considerably, although not significantly, between the 2 experimental days (e.g., 1.7 and 0.9 times the resting values). An explanation could be that the electrode placement for the RF muscle was slightly different between the experimental days, which may have affected the EMG measurement (11, 18). Furthermore, all participants performed the physical activity bouts within the targeted range of 40–60% HRR, i.e., moderate intensity, with HRR varying between 46% and 57% HRR.

A strength of our study and novel aspect included the measurement of muscle activity of the GAS and RF weight-bearing muscles in young adults. Since the biological mechanism underlying the health consequences of prolonged sitting suggests the necessity of muscle activity, it is important to establish how much muscle activity is sufficient to attenuate these consequences. As the present study only collected these data from a subsample of participants, it was not possible to examine whether muscle activity potentially mediated the effects of interrupted sitting on health outcomes. This could be examined in future research. Another strength was the focus on the effects of prolonged vs. interrupted sitting on lipid metabolism, which have not been reported to date. Our sample of healthy, young (18–24 yr) adults is an additional strength of our study, since it is less likely to be influenced by confounding effects, such as aging and disease progression (e.g., obesity, T2DM). The crossover design also strengthens our study. By experimentally imposing 8 h of sitting and 8 h of interrupted sitting in a separate session (assigned randomly), we were able to study the effects of prolonged vs. interrupted sitting in a systematic way.

A limitation of our study was the capillary blood-sampling method, which is considered to be inferior to venous blood sampling, although this method is considered to be a valid alternative to venous blood sampling, as linear regression analysis revealed that slopes differed no more than 10% from 1.0 (representing near-perfect agreement), except for glucose (10). This should be kept in mind when interpreting our results. Another limitation is the small sample size, which could explain the lack of statistical significance for postprandial glucose and blood lipids. Finally, we were unable to obtain information on the level of hydration during the physical-activity interruptions, which might have influenced the measurement precision of the assessed cardiometabolic biomarkers.

We conclude that short, hourly, moderate-intensity physical-activity interruptions to prolonged sitting, requiring muscle activity of the weight-bearing muscles of seven to eight times the resting value, may prevent increases in postprandial levels of C-peptide. It is important that detrimental cardiometabolic effects may already occur within 1 day of prolonged sitting, even in young and healthy adults. Our findings further support recent suggestions for interrupting sitting time, in addition to meeting the physical activity guidelines to prevent cardiometabolic risk (8). Future studies should examine different frequencies and durations of physical activity interruptions to prolonged sitting and effects on cardiometabolic health to determine the most advantageous pattern of prolonged vs. interrupted sitting in preventing cardiometabolic risk.

**REFERENCES**


