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Original research

Breaking up sedentary time, physical activity and lipoprotein metabolism☆

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ABSTRACT

Objectives: Some authors report delayed responses of lipid metabolism after prolonged and interrupted sitting. To date no experimental studies have shown acute physical activity (PA) related changes of lipoprotein metabolism during sedentary behavior (SB).

The purpose of this study was to examine immediate effects of a single PA-bout vs. regular PA-breaks on lipoprotein metabolism during postprandial SB.

Design: Eighteen female subjects (25.6years ± 2.6; 21.5 kg/m2 ± 2.0) participated in a balanced crossover experiment with three trials: (1) 30 min PA prior to 4 h sitting (PRE), (2) 5×6 min PA within 4 h sitting (BREAK) (3) 4 h sitting (CTRL). PA was ergometer cycling at 70%VO_2max. Participants consumed one standardized breakfast in each trial.

Methods: Total cholesterol (tChol), high density lipoprotein (HDL), low density lipoprotein (LDL) and triacylglycerol (TAG) readings were taken in fasted state at the beginning (before), and immediately after each trial. Multiple 2×3-ANOVAs (trial-arm*time) and post hoc t-tests were used.

Results: ANOVAs revealed trial*time-effects for tChol (p = 0.043) and HDL (p = 0.007) and a tendency for LDL (p = 0.078). Before-to-after differences (Δ) of tChol during BREAK-trial (-0.89 ± 5.49 mg/dl) significantly differed to Δ during PRE-trial (+3.56 ± 5.32 mg/dl) (p = 0.005) but not to ΔCTRL (+2.16 ± 8.72). ΔHDL during BREAK-trial (-2.11 ± 3.31 mg/dl) significantly differed to ΔPRE (+0.83 ± 4.26 mg/dl) (p = 0.007) and ΔCTRL (+0.06 ± 4.15 mg/dl) (p = 0.028).

Analysis of TAG revealed a time effect (p = 0.007) and significant differences between TAG before and after trial within BREAK (79.22 ± 38.69 mg/dl to 94.78 ± 44.86 mg/dl) and CTRL (80.94 ± 32.94 mg/dl to 97.72 ± 41.19 mg/dl) but not in PRE (78.83 ± 33.13 mg/dl to 89.06 ± 33.83 mg/dl).

Conclusions: Breaking sedentary behavior with vigorous intensity PA seem to decelerate postprandial lipoprotein metabolism during sitting. In contrast, prior PA, might be a stimulus for lipid metabolization.

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1. Introduction

Sedentary behavior (SB) is increasingly regarded as a major risk factor for metabolic dysfunction.[1] Observational data indicate an association between SB and metabolic precursors of major chronic diseases such as cardiovascular disease, type 2 diabetes[2] and various cancerous diseases.[3] Consequently, SB may lead to development of metabolic syndrome and further chronic diseases.[4]

It has been suggested that prolonged sitting and too little physical activity (PA) may evoke distinct physiological effects.[5] Underlying biological mechanisms are, as of now, not fully understood. In animal models the lack of muscle contraction in prolonged immobilization leads to suppression of skeletal muscle lipoprotein-lipase (LPL) activity and changes in muscle energy metabolism.[6,7] Some authors suggest that uninterrupted periods of prolonged sitting may lead to comparable biological consequences in humans as well.[8,9]

Findings of bed rest studies support this assumption. Such extreme inactivity is associated with increased plasma triacyl-glycerol (TAG) levels, decreased levels of high-density lipoprotein (HDL) cholesterol and decreased insulin sensitivity.[1]

One of the earlier accelerometer studies shows a beneficial association of breaks in sedentary time with metabolic biomarkers.[10] In contrast, a subsequent study failed to confirm favorable associations with lipoprotein concentrations in a larger, representative study sample.[11] Another work based on the National Health and Nutrition Examination Survey (NHANES) report that patients with

metabolic syndrome spend a greater percentage of time as sedentary with longer bouts and fewer breaks.[12] In people with newly diagnosed type 2 diabetes breaks in SB showed a linear association with higher HDL cholesterol in an observational study.[13] Underlying biological mechanism could be modulated by energy expenditure[14] and skeletal muscle metabolism[9] as well as long term modifications in muscle quality or lower limb fat storage.[15]

Current experimental studies on acute effects of interrupting prolonged sitting have focused primarily on short term modulation of glucose[16–20] and fat metabolism.[18,17,16] These studies suggest that PA bouts interrupting SB may have acute positive effects on insulin sensitivity and serum glucose but not on serum TAG or other markers of fat metabolism. Only a limited number of studies have investigated the acute effects of breaks during SB on lipoprotein metabolism. Altenburg et al. investigated the effects of sitting eight hours continuously or interrupted by hourly 8 min moderate intensity cycling bouts on total-, HDL, and low density lipoprotein (LDL) cholesterol and TAG.[16] Bailey and Locke compared the effects of 2-min bouts of standing or light walking every 30 min in a 3 armed design.[17] In both studies PA breaks did not modulate markers of lipoprotein and fat metabolism. Contrastingly, a recent experiment of Miyashita et al. found differences of TAG while markers of glucose metabolism were comparable during continuous and interrupted sitting.[21]

Some studies, however, report lowering effects of PA breaks during SB on fasting[22] or postprandial TAG[23,24] on the following day.

Therefore the question arises what acute metabolic changes during SB may mediate this delayed responses. Furthermore it is of great interest what influence the intensity, overall volume or the resulting energy expenditure of PA might have on fatty acid (FA) metabolization in muscle or adipose tissue during SB.

To increase the understanding of chronic effects of SB and to differentiate the influence of breaks during SB and overall PA on lipoprotein and fat metabolism it is important to analyze acute metabolic modulation during prolonged sitting which may mediate these long term effects.

Currently no study has compared the effects of prior continuous PA and short PA breaks on lipoprotein metabolism during prolonged sitting.

The purpose of this study was to evaluate acute influences of prolonged sitting and possible moderating effects of continuous PA or breaks in SB. We compared the immediate effects of a single PA (PA) bout vs. workload-matched regular PA breaks on lipoprotein metabolism in postprandial SB in healthy young women.

2. Methods

The balanced cross-over study was approved by the local ethic commission (121/13) and was in accordance with the Declaration of Helsinki and guidelines for good clinical practice. Written informed consent was obtained from all study participants prior to study enrollment.

Sample size, calculated prior to study enrollment and based on the study of Miyashita et al.,[24] revealed a total sample size of n = 15. Assuming a dropout rate of 20%, eighteen subjects were included in the study. Participants were recruited through advertisement at the university campus. Based on the inconsistent data on mixed,[16,17,22] female only[21] and male only samples[23,24] and to control for confounding effects we opted for a homogeneous sample of young healthy premenopausal women (n = 18, 25.6 years ± 2.6). General exclusion criteria included pregnancy, acute or chronic physical and psychological diseases and drug abuse. Subjects were only recruited if they had resting arterial pressure <140/90 mmHg and body mass index (BMI; in kg/m^2) <30. Subjects were asked to refrain from alcohol, caffeine and strenuous PA 24 h prior to the preliminary medical examination and the main trials respectively. In order to standardize dietary intake participants were also requested to maintain their regular diet for the whole duration of the study. Participants had to complete a short questionnaire recording PA of the last 48 h and the last meal prior to first trial enrollment using open questions and confirming that ingestion of the last meal was ≥8 h prior to trial start and that they refrained from alcohol and caffeine. Further trials only commenced if participants verified their compliance with the PA and dietary protocol.

Detailed anamnesis and physical examination were used to confirm health status. Maximal exercise capacity was assessed with a ramp shaped cardiopulmonary exercise test (CPET) until volitional exhaustion on an electrically braked cycle ergometer during which oxygen uptake (VO_2) was measured using a breath-by-breath gas analyzer (Oxycon Mobile, Viasys Healthcare GmbH, Wuerzburg, Germany). The highest 30 s floating mean of VO_2 within the testing time was defined as maximal oxygen uptake (VO_2max). Workload in watts (W) at 70% VO_2max was used as individual PA intensity during trials. CPET data of oxygen consumption and carbon dioxide elimination were used to estimate respiratory quotient, energy expenditure and macronutrient utilization during 30 min of cycling with the intensity mentioned above.

Each subject completed three trials: (1) A single bout of 30 min PA prior to continuous sitting; (2) sitting interrupted by 5 activity breaks of 6 min; (3) continuous sitting without PA. The three trials were separated by a minimum of 7 days and the sequence of trials was balanced within the study group. The participants arrived in the morning after an overnight fast. Each trial began with baseline sampling immediately after a rest period of 15 min. During all trials participants consumed a test meal consisting of white bread, cheese and jam. The caloric value of 1 portion (85 g) was 228.15 kcal. Macronutrients composition (51% carbohydrate (29 g); 35% fat (9 g); 14% protein (8 g)) reflected typical diet of western women based on the german national food survey.[25] Participants were free to select the number of portions they consumed. This amount was kept constant across all trials. Participants consumed the test meal within the first 15 min of sitting time. As shown in Fig. 1, in trial PRE they cycled for 30 min at 70% VO_2max on a standardized ergometer after baseline sampling and before meal ingestion. This PA bout was followed by test meal ingestion and 4 h sitting time. In BREAK subjects interrupted the 4 h sitting period with 5 bouts of 6 min cycling at 70% VO_2max. The third trial (CTRL) included 4 h uninterrupted sitting without PA.

Venous blood samples (antecubital) were taken in fasted state at the beginning and immediately at the end of all three trials. Enzymatic, colorimetric assays (Roche/Hitachi cobas c systems) were used for triacylglycerol and total, HDL- and LDL cholesterol analysis.

Descriptive statistics included the mean ± standard deviation (SD). Correlation analysis was used to control for confounding influences of caloric content of the experimental meal. Data were analyzed by multiple 2 × 3 ANOVAs (trial*time). In case of significance, t-tests for post-hoc analysis were used (SPSS Inc., Chicago, IL, USA). In case of significant "trial*time" effects, the delta (Δ) of before and after values for each trial arm was calculated for post hoc analysis between trials. In case of significant time effects t-tests were used to analyze before and after differences within a trial arm. p < 0.05 was considered statistically significant.

3. Results

Table 1 shows baseline participant characteristics. Baseline values for TAG, total-, HDL- and LDL cholesterol were not different between trials. Group average calorie intake was 519.7 ± 211.5 kcal (between subject CV: 0.40) (8.9 ± 3.7 kcal/kg; CV:0.42). Subjects