Published in final edited form as:

Eur J Appl Physiol. 2010 January ; 108(2): 329–336. doi:10.1007/s00421-009-1203-6.

Longitudinal relationship between physical activity and cardiometabolic factors in overweight and obese adults

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Abstract

Few studies have reported longitudinal relationships between physical activity (PA) and cardiometabolic risk factors over time using repeated assessments in overweight or obese adults. We conducted a longitudinal study in 127 participants (81% with body mass index $> 30 \text{ kg/m}^2$) who completed a 12-month behavioral intervention for weight loss between 2003 and 2005 in Pittsburgh, PA, USA. Using absolute change scores from baseline to each time point (i.e., 6 and 12 months) for all studied variables ($\Delta = \text{time point} - \text{baseline}$), we performed mixed effects modeling to examine relationships between PA and cardiometabolic risk factors, after adjusting for body weight, energy intake and other covariates (i.e., age, gender, and ethnicity). PA was assessed as energy expenditure (kcal/week) using the Paffenbarger activity questionnaire. Over the 12-month period, energy expenditure increased ($\Delta 1,370 \text{ kcal/week}$ at 6 months vs. $\Delta 886 \text{ kcal/week}$ at 12 months); body weight decreased ($\Delta 8.9 \text{ kg}$ at 6 months vs. $\Delta 8.4 \text{ kg}$ at 12 months). The average increase in energy expenditure over 12 months was significantly and independently related to reductions in total cholesterol ($F = 6.25, p = 0.013$), low-density lipoprotein cholesterol (LDL-C) ($F = 5.08, p = 0.025$) and fasting blood glucose ($F = 5.10, p = 0.025$), but not to other

risk factors (i.e., fasting insulin, high-density lipoprotein cholesterol, triglycerides, and waist circumference). In conclusion, among overweight and obese adults undergoing a weight loss intervention, increased energy expenditure over 12 months may improve total cholesterol and LDL-C, important coronary risk factors, and fasting blood glucose, a metabolic risk factor.

Keywords

Physical activity; Obesity; Longitudinal studies; Lipids; Weight loss

Introduction

Obesity is seen today as a global epidemic, leading to an increased risk of morbidity and mortality as well as reduced life expectancy in the general population (Poirier et al. 2006). In the US, the prevalence of obesity has risen dramatically during the past 20 years, in parallel with the increased prevalence of type 2 diabetes mellitus (Ford et al. 2003, 2004). Furthermore, obesity is recognized as a predictor of both type 2 diabetes and cardiovascular disease (Poirier et al. 2006) and is associated with cardiometabolic risk factors including dyslipidemia, hyperglycemia, and insulin resistance (Grundy 2004).

Increasing physical activity (PA) may be an important strategy for reducing cardiometabolic risk factors in overweight or obese individuals, in combination with or independent of weight reduction. The Nurse's Health Study reported that PA was inversely associated with the risk for coronary heart disease, independent of body mass index (BMI) (Li et al. 2006). Recently, a prospective cohort study reported that an increase in PA over time, independent of adiposity, was associated with improvements in insulin sensitivity and fasting triglycerides in a population of white middle-aged adults (Ekelund et al. 2007). In addition, reducing total energy intake while maintaining a moderate fat diet and increasing physical activity can reduce metabolic abnormalities (Lichtenstein et al. 2006).

Few studies have reported independent relationships between changes in PA and changes in cardiometabolic risk factors in overweight or obese individuals, especially individuals who attempted to lose weight. Changes in cardiometabolic risk factors (i.e., plasma lipids and fasting insulin) may predominantly result from weight loss by the effects of increased PA and restricted energy intake (Dattilo and Kris-Etherton 1992; Wing et al. 1987). In this regard, there was little evidence of the effect of PA on those risk factors after controlling for weight loss and total energy intake. Furthermore, few studies have reported longitudinal relationships between changes in PA and cardiometabolic risk factors using repeated assessments, in overweight and obese adults.

The purpose of our study was to conduct a secondary analysis of a longitudinal study to determine whether there were independent relationships of changes in PA with concurrent changes in lipids [i.e., total cholesterol, low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and triglycerides], fasting levels of glucose and insulin, and waist circumference, independent of changes in weight and energy intake. We conducted this study in a sample of overweight and obese adults using data measured at baseline, 6 and 12 months, in a 12-month weight loss intervention.

Methods

Study participants

The participants were obtained from the PREFER trial, a randomized clinical trial, using a four-group design, that tested treatment preference and two dietary options (i.e., treatment

preference-yes vs. treatment preference-no; standard fat- and calorie-restricted vs. fat- and calorie-restricted, lacto-ovo-vegetarian diet) in a weight loss intervention over 18 months (Burke et al. 2007). The 18-month study period had an active intervention phase of 12 months (32 sessions) followed by a no-contact maintenance phase of 6 months. The participants of the trial were healthy adults aged 18–53 years with a BMI between 27 and 43 kg/m² and were free of clinical diseases (i.e., cardiovascular disease, diabetes, or kidney disease). The study protocol is described in detail elsewhere (Burke et al. 2006). All participants provided written informed consent. The study was approved by the Institutional Review Board at the University of Pittsburgh.

These secondary analyses focus on the 12-month intervention phase of the study. Of the 176 participants in the original sample, 127 (72%) who completed the 12-month assessment and had non-zero scores on the Paffenbarger activity questionnaire at all of the time points (baseline, 6 and 12 months) were included in the final analysis. The baseline characteristics of 127 participants (i.e., age, education, weight, and cardiometabolic risk factors) did not differ from those excluded ($n = 49$).

Intervention

The intervention in the PREFER trial was a standard cognitive-behavioral intervention for weight management that included goal setting and self-monitoring among other behavioral change strategies. The PA intervention was designed to gradually increase participants' leisure time PA over the first 6 weeks, primarily through brisk walking, until they reached a minimum goal of 150 min per week, which approximates an energy expenditure of 1,000 calories per week. Participants were also encouraged to add other modes of activity (e.g., using stationary bicycles or step machines, swimming, aerobics, and strength training). We instructed participants to self-monitor and record in their paper diaries the type and amount of PA (in minutes) they performed; the interventionists reviewed their diary entries and provided feedback weekly for the first 6 months, biweekly for months 7–9, and monthly for months 10–12 (Burke et al. 2007). Regardless of the diet option (standard fat- and calorie-restricted vs. fat- and calorie-restricted, lacto-ovo-vegetarian diet), we gave all participants individualized calorie and fat gram goals based on their baseline body weight (1,200 kcal if <200 lbs; 1,500 kcal if ≥200 lbs for females; plus 300 kcal for males) and then monitored their dietary intake using the paper diaries. If the individual's weight was reduced to less than 200 lbs the calorie goal was adjusted downward to either 1,200 or 1,500 kcal per day. The intervention has been described in detail elsewhere (Burke et al. 2007).

Measurements

All measures were taken at baseline, 6 and 12 months. Energy expenditure was self-reported using the Paffenbarger activity questionnaire, as an average of total energy expenditure during daily living for the past 7 days. This questionnaire consists of three items that pertain to stairs climbed, blocks walked, and other leisure time activities (Paffenbarger et al. 1995). In previous studies, the instrument demonstrated good validity and reliability ($r = 0.34–0.72$) (Ainsworth et al. 1993, 2000). A metabolic equivalent (MET) value was assigned to each leisure time activity. On the basis of the energy cost of each activity (Ainsworth et al. 2000), we estimated a combined energy expenditure (kcal/week) through walking, stair climbing, and all sports and leisure time activities.

Total energy (kcal/day) and fat intake (% of energy) were obtained from 3-day food records. The data were analyzed using the Nutrition Data System for Research (NDS-R, <http://www.ncc.umn.edu>) software program by staff at the Obesity/Nutrition Research Center at the University of Pittsburgh.

Blood samples were obtained following a 12-h fast after at least a 5-min resting period. The methods of measurements of biochemical factors (i.e., glucose, insulin, total cholesterol, HDL-C, LDL-C, and triglycerides) were described in detail elsewhere (Burke et al. 2007). Weight (kg) was measured after an overnight fast in light clothing without shoes, using the Tanita bioelectrical impedance scale (Tanita Corporation of America, Inc., IL, USA). Height was measured on a wall-mounted stadiometer. BMI was computed as weight (kg) divided by height (m) squared. Waist circumference (cm) was measured using a Gullick II measuring tape at the line of the uppermost border of the iliac crest.

Statistical analysis

Statistical analyses were performed using SAS (version 9.1.3; SAS Institute Inc., Cary, NC). The significance level for two-sided hypothesis was set at 0.05. Descriptive statistics were computed as means and standard deviations (or medians and interquartiles) for continuous variables and frequency and percentages for categorical variables. For all the studied variables, the absolute changes from baseline to each time point (i.e., 6 months and 12 months) were expressed as scores (Δ = time point – baseline). To investigate independent relationships between absolute changes in PA and cardiometabolic risk factors after controlling for weight loss and caloric restriction, we applied the repeated-measures analysis using linear mixed effects modeling via PROC MIXED in SAS. Expected total observations were 254 (i.e., observations of PA energy expenditure changes at two time points) derived from 127 participants. However, we used 250 observations in our final analysis because four participants did not come to the 6-month assessment. The unadjusted models included the main effects for energy expenditure changes and time and their interaction. The adjusted models additionally included age, gender, ethnicity, weight change, caloric change, and the interactions of weight change with time and energy intake change with time. When modeling LDL-C, the adjusted model was further expanded to include saturated fat. We excluded the dietary option in the adjusted models because there were almost no differences in parameter estimates and *p* values for main outcomes between models with and without further adjustment for dietary option. In addition, the same analyses were repeated for women (*n* = 110) by excluding men from data set.

Sensitivity analyses were performed to assess data points that had large residuals (i.e., poorly fitted by the model) for each model. These data points were excluded from the analysis to minimize the impact of unrealistic PA scores on outcome variables. The results for models without influential data points were reported. The observation numbers (i.e., number of data points) used in unadjusted and adjusted models are detailed in Tables 3 and 4.

Results

The study participants (*N* = 127) had a mean age of 44.6 years (Table 1). They were predominantly White (69%) and female (87%) and had a mean BMI of 33.7 kg/m²; 19% of the participants were overweight and 81% were obese according to the criteria of World Health Organization (2006). The group mean for total cholesterol was borderline high; the mean LDL-C level was above optimal among all participants according to the National Cholesterol Education Program (NCEP) ATP III Guidelines (National Heart Lung and Blood Institute 2004). The mean total energy intake was 2,496 kcal/day in men and 1,969 kcal/day in women; the mean total energy expenditure by leisure time activities was 2,829 kcal/week in men and 2,210 kcal/week in women.

Table 2 shows absolute change scores of all studied variables at each time point (6 and 12 months) from baseline. Over the 12-month period, energy expenditure increased (Δ 1,370 kcal/week at 6 months vs. Δ 886 kcal/week at 12 months); body weight decreased (Δ 8.9 kg at

6 months vs. $\Delta 8.4$ kg at 12 months). Total cholesterol levels ($\Delta 8.1$ mg/dL at 6 months vs. $\Delta 6.3$ mg/dL at 12 months) and LDL-C levels ($\Delta 3.6$ mg/dL at 6 months vs. $\Delta 4.7$ mg/dL at 12 months) decreased over 12 months. HDL levels decreased at 6 months ($\Delta 2.2$ mg/dL) and increased at 12 months (0.7 mg/dL). Fasting glucose and insulin levels decreased at 6 and 12 months, respectively. Waist circumference also decreased (7.8 cm at 6 months vs. 7.7 cm at 12 months) over 12 months.

The multivariate-adjusted models are presented for the total sample ($N = 127$) (Table 3) and for only female participants ($n = 110$) (Table 4). The significant findings for female participants were nearly comparable to those for the total sample. In the multivariate-adjusted models for the total sample (Table 3), the average increase in energy expenditure over 12 months was significantly related to the decreases in total cholesterol ($b = -0.002$; $F = 6.25$; $p = 0.013$), LDL-C ($b = -0.002$; $F = 5.08$; $p = 0.025$), and fasting glucose ($b = -0.0003$; $F = 5.10$; $p = 0.025$). There were no significant interactions with time (at 6 vs. 12 months) in the associations between changes in energy expenditure and any cardiometabolic risk factors. The weight loss over 12 months was significantly related to decreases in total cholesterol ($b = 0.999$; $F = 12.61$; $p < 0.001$), LDL-C ($b = 0.936$; $F = 10.77$; $p = 0.001$), triglycerides ($b = 1.934$; $F = 16.64$; $p < 0.001$), fasting insulin ($b = 0.232$; $F = 13.80$; $p < 0.001$), and waist circumference ($b = 0.898$; $F = 153.67$; $p < 0.001$). The association between weight loss and triglycerides at 6 months differed significantly from that at 12 months [$b(\text{weight} \times \text{time}) = 1.386$; $F = 4.07$; $p = 0.046$].

Discussion

This study examined longitudinal relationships between changes in leisure time energy expenditure and cardiometabolic risk factors among overweight and obese adults, using absolute change scores from baseline to 6 and 12 months. We found that the average increase in energy expenditure over 12 months was significantly related to decreases in total cholesterol, LDL-C and fasting glucose, controlling for weight loss, dietary restriction, and other confounding factors. However, we did not observe independent relationships between changes in energy expenditure and the other risk factors (i.e., HDL-C, triglycerides, fasting insulin, and waist circumference).

The increase in energy expenditure (kcal/week) was significantly related to decreases in total cholesterol and LDL-C concentration, even after controlling for weight change and dietary restriction. The lowering of LDL-C is currently the primary target therapy of the NCEP ATP III (National Heart Lung and Blood Institute 2004). The Women's Health Study reported that a higher amount of energy expenditure was independently associated with lower levels of LDL-C in 27,158 healthy women, after adjusting for BMI and other confounding factors (Mora et al. 2006). Additionally, a meta-analysis of randomized controlled trials elucidated significant effects of at least 8 weeks of aerobic exercise (i.e., 8–102 weeks) on reductions in total cholesterol and LDL-C for women (Kelley et al. 2004). However, evidence on an exercise-induced reduction in LDL-C is still scarce among overweight and obese individuals. It has been suggested that weight and body mass loss may be more important correlates of the reduction in LDL-C (Kelley et al. 2005; Nieman et al. 2002; Stefanick 1999). Kraus et al. (2002) conducted a randomized controlled trial among 111 overweight and obese men and women who were instructed to maintain their weight to investigate the effects of different amounts and intensity of exercise on LDL subfractions. Kraus and colleagues reported that exercise training had no significant effect on the total or LDL-C concentrations; however, they did find greater improvements in LDL subfractions (i.e., total LDL particles, small LDL cholesterol, and intermediate density lipoprotein cholesterol) occurred with a high amount of high intensity exercise (equivalent to jogging 20 miles per week at 65–80% of peak oxygen consumption) after 8 months. Durstine et al.

(2002) suggested that favorable changes in lipids with exercise require energy expenditures of 1,200–2,200 kcal/week or the higher end of this range. Thus, our data are not totally consistent with the previous findings, but the lowering effect on LDL-C in our study may be explained by a large volume of change in energy expenditure (i.e., 1,370 kcal/week at 6 months and 886 kcal/week at 12 months from baseline) that outweigh a weight loss-induced effect.

The increase in energy expenditure was independently related to the decrease in fasting glucose levels, but weight loss was not related to glucose levels over 12 months. Norris et al. (2005) conducted a systematic review of dietary, physical activity, or behavioral weight loss intervention trials for adults with prediabetes. The weight loss interventions of a 1- or 2-year duration had a significant effect on weight loss, but provided limited results on improvements in fasting glucose. In contrast, Shaw et al. reported in their systematic review that exercise, as a sole weight loss intervention, resulted in significant reductions in fasting glucose in overweight and obese individuals (Shaw et al. 2006); also, exercise intensity was a determinant of reduced fasting glucose levels. In this context, the reduction in fasting glucose levels may also be primarily related to exercise or physical activity in a weight loss program and this relationship may become stronger when considering exercise quality.

We did not observe a significant relationship between changes in energy expenditure and fasting insulin levels, which may be due to the median insulin levels being within the normal range at baseline. Ross et al. (2004) reported in a randomized controlled trial among obese women that exercise with or without weight loss had no effect on fasting insulin levels compared to a control group. Nevertheless, there is evidence suggesting the beneficial effects of exercise training on a persistent increase in insulin sensitivity in skeletal muscle from obese individuals (Hawley and Lessard 2008).

The increase in energy expenditure was not significantly related to changes in HDL-C and triglycerides. Studies have reported that a small increase in HDL-C was related to exercise and blunted in obese individuals compared to lean or moderately obese individuals (Kelley et al. 2005; Nicklas et al. 1997; Shaw et al. 2006). Similarly, we observed small decreases in HDL-C following 6 months with a small increase in HDL-C at 12 months. As a result, a marginal change of HDL-C over 12 months may be a possible explanation for the non-significant relationships with change in either energy expenditure or weight loss. As for the change in triglycerides, most, but not all studies, showed that aerobic exercise decreases triglycerides in overweight and obese adults, independent of changes in body composition (Kelley et al. 2005; Nieman et al. 2002; Shaw et al. 2006). It has been noted that an improvement in triglycerides with exercise may be determined by baseline concentrations (Durstine et al. 2002). Similarly, the lack of a relationship between energy expenditure and triglycerides in our study may be partially explained by the normal range of triglyceride level at baseline.

We did not observe a significant relationship between changes in energy expenditure and waist circumference, but the beneficial effect of physical activity on waist circumference has been reported (Kay and Fiatarone Singh 2006). However, the longitudinal impact of physical activity on abdominal adiposity is not clear, particularly using a multivariate analysis that includes weight loss. Some studies have suggested that increased energy expenditure in overweight or obese adults reduces abdominal fat only modestly or not at all (Kay and Fiatarone Singh 2006; National Heart Lung and Blood Institute, Obesity Education Initiative Expert Panel on the Identification Evaluation and Treatment of Overweight and Obesity 1998).

Our study has limitations. The use of self-reported assessment of energy expenditure based on a recall of daily activities for the past week may have resulted in a non-representative estimate of the individual's physical activity for the 6-month interval. However, this or similar questionnaires are used in most studies examining weight loss and energy expenditure. Future studies need to include an objective measure of physical activity to strengthen the assessment of PA. We did not collect information on the use of oral contraceptives and hormone replacement therapy, which may affect lipid outcomes, and thus we were not able to adjust for these in our analyses. Another potential limitation is that we did not control the time period (e.g., 24 or 48 h rest) after the last exercise session prior to blood sampling, although the acute effects of a moderate exercise session on lipid outcomes may be minimal (Gordon et al. 1998; Imamura et al. 2000). In addition, our study was not powered to examine gender-differences in independent relationships between PA and cardiometabolic risk factors because of a small sample size of men ($n = 17$) even though the gender variable was adjusted. Our study also has strengths, notably its longitudinal nature and good retention of study participants.

In conclusion, among overweight and obese adults undergoing a behavioral weight loss intervention, increased energy expenditure over 12 months may improve total cholesterol and LDL-C, important coronary risk factors and fasting blood glucose, a metabolic risk factor.

Acknowledgments

This work was supported by National Institutes of Health grants NIH, NIDDK, #RO1-DK58631; LE Burke's effort was partially covered by grant 1K24 NR010742. The conduct of the study was also supported by the Data Management Core of the Center for Research in Chronic Disorders NIH-NINR #P30-NR03924, the Obesity and Nutrition Research Center NIH-NIDDK #DK-046204, and the General Clinical Research Center, NIH-NCRR-GCRC #5MO1-RR00056 at the University of Pittsburgh. We thank the study participants who graciously shared their time and information with us.

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Table 1Baseline characteristics of the participants in 2003–2005 ($N = 127$)

	Mean (SD)		
	Female ($n = 110$)	Male ($n = 17$)	All ($n = 127$)
Age (years)	44.5 (8.5)	44.9 (7.4)	44.6 (8.3)
White, n (%)	75 (68)	13 (77)	88 (69)
Body weight (kg)	92.5 (13.5)	105.3 (13.8)	94.2 (14.2)
BMI (kg/m^2)	33.8 (4.1)	33.5 (3.5)	33.7 (4.0)
Overweight (25 to <30), n (%)	21 (19)	3 (18)	24 (19)
Obese (≥ 30), n (%)	89 (81)	14 (82)	103 (81)
Total cholesterol (mg/dL)	205.9 (37.9)	198.4 (51.8)	204.9 (39.9)
LDL-C (mg/dL)	126.5 (35.0)	121.3 (44.6)	125.8 (36.3)
HDL-C (mg/dL)	54.4 (11.0)	41.9 (9.1)	52.8 (11.6)
Triglycerides (mg/dL) ^a	116.0 (82.0–146.0)	160.0 (87.0–240.0)	118.0 (82.0–160.0)
Fasting glucose (mmol/L)	95.9 (7.9)	99.8 (7.8)	96.5 (8.0)
Fasting insulin ($\mu\text{mol}/\text{L}$) ^a	16.5 (12.5–21.9)	16.9 (13.1–27.3)	16.5 (12.5–22.0)
Waist circumference (cm)	103.5 (13.3)	111.9 (11.2)	104.6 (13.4)
Energy intake (kcal/day)	1,969 (567)	2,496 (807)	2,039 (627)
Saturated fat (% of energy)	11.9 (3.0)	11.0 (2.1)	11.7 (2.9)
PA energy expenditure (kcal/week)	2,210 (2,632)	2,829 (2,120)	2,293 (2,571)

BMI body mass index, *LDL-C* low-density lipoprotein cholesterol, *HDL-C* highdensity lipoprotein cholesterol, *PA* physical activity

^aValues are expressed as median and interquartile range

Table 2Absolute changes from baseline in all studied variables at 6 and 12 months ($N = 127$)

	Mean (SD)	
	Δ (6 months-baseline)	Δ (12 months-baseline)
PA energy expenditure (kcal/week)	1,370.0 (3,202)	885.8 (3,238)
Body weight (kg)	-8.9 (5.9)	-8.4 (7.0)
Energy intake (kcal/day)	-605 (510.9)	-552 (577.8)
Saturated fat (% of energy)	-3.9 (4.0)	-2.8 (4.1)
Cardiometabolic factors		
Total cholesterol (mg/dL)	-8.1 (26.8)	-6.3 (29.4)
LDL-C (mg/dL)	-3.6 (23.2)	-4.7 (24.3)
HDL-C (mg/dL)	-2.2 (7.3)	0.7 (9.0)
Triglycerides (mg/dL)	-11.4 (57.4)	-10.6 (55.8)
Fasting glucose (mmol/L)	-1.8 (8.3)	-1.5 (8.7)
Fasting insulin (μ mol/L)	-4.4 (5.7)	-3.3 (7.5)
Waist circumference (cm)	-7.8 (7.7)	-7.7 (9.1)

SD standard deviation, *PA* physical activity, *HDL-C* high-density lipoprotein cholesterol, *LDL-C* low-density lipoprotein cholesterol

Table 3

Relationships between changes in physical activity and changes in cardiometabolic factors for total participants ($N = 127$ and total observations = 250)

Change scores	<i>F</i> value							
	Obs. ^a	PA	Time	PA × time	Energy intake	Energy intake × time	Weight	Weight × time
Total cholesterol (mg/dL)								
Unadjusted	250	9.03 **	1.36	4.32 *				
Adjusted	244	6.25 *	0.02	3.57	1.32	0.19	12.61 ***	0.85
LDL-C (mg/dL) ^b								
Unadjusted	250	8.07 **	0.03	2.79				
Adjusted	244	5.08 *	0.03	2.01	0.02	0.40	10.77 **	0.01
HDL-C (mg/dL)								
Unadjusted	250	0.00	19.66 ***	0.32				
Adjusted	244	0.24	1.64	0.46	7.13 **	1.05	2.58	0.38
Triglycerides (mg/dL)								
Unadjusted	250	1.10	0.27	1.89				
Adjusted	244	0.19	2.29	1.78	0.03	0.00	16.64 ***	4.07 *
Fasting glucose (mmol/L)								
Unadjusted	250	7.50 **	0.02	0.00				
Adjusted	244	5.10 *	1.78	0.07	2.84	0.03	2.26	2.53
Fasting insulin (μmol/L)								
Unadjusted	250	0.03	4.17 *	0.00				
Adjusted	244	0.03	0.14	0.01	0.00	0.30	13.80 ***	1.72
Waist circumference (cm)								
Unadjusted	249	3.45	0.60	2.69				
Adjusted	243	0.51	0.28	0.87	0.06	1.88	153.67 ***	0.10

PA physical activity, HDL-C high-density lipoprotein cholesterol, LDL-C low-density lipoprotein cholesterol

 $p < 0.001$,

**
 $p < 0.01$,

*
 $p < 0.05$

^a Observation numbers that were used in each model

^b For the model for LDL-C, saturated fat (% of energy) was further adjusted

Table 4

Relationships between changes in physical activity and changes in cardiometabolic factors for female participants ($N = 110$ and total observations = 216)

Change scores	F value							
	Obs. ^a	PA	Time	PA × time	Energy intake	Energy intake × time	Weight	Weight × time
Total cholesterol (mg/dL)								
Unadjusted	216	8.07 **	0.16	5.75 *				
Adjusted	211	7.51 **	0.06	3.96 *	4.00 *	0.23	11.94 ***	0.10
LDL-C (mg/dL) ^b								
Unadjusted	216	7.55 **	0.96	2.15				
Adjusted	211	5.77 *	0.01	1.00	0.50	0.05	15.00 ***	0.39
HDL-C (mg/dL)								
Unadjusted	216	0.22	24.05 ***	0.45				
Adjusted	211	0.92	2.03	0.80	5.59 *	0.07	3.40	1.47
Triglycerides (mg/dL)								
Unadjusted	216	0.42	0.01	7.88 **				
Adjusted	211	0.35	2.49	7.37 **	1.85	0.41	7.23 **	3.59
Fasting glucose (mmol/L)								
Unadjusted	216	6.31 *	0.02	0.15				
Adjusted	211	4.07 *	1.44	0.52	2.57	0.01	3.19	2.14
Fasting insulin (μmol/L)								
Unadjusted	216	1.65	1.34	0.06				
Adjusted	211	1.60	0.06	0.06	1.35	0.82	4.95 *	0.98
Waist circumference (cm)								
Unadjusted	215	3.28	0.50	5.10 *				
Adjusted	210	0.88	0.01	0.74	0.44	1.80	107.58 ***	1.37

PA physical activity, HDL-C high-density lipoprotein cholesterol, LDL-C low-density lipoprotein cholesterol

 $p < 0.001$,

**
 $p < 0.01$,

*
 $p < 0.05$

^a Observation numbers that were used in each model

^b For the model for LDL-C, saturated fat (% of energy) was further adjusted