Exercise training and/or diet on reduction of intra-abdominal adipose tissue and risk factors for cardiovascular disease

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Abstract

BACKGROUND/OBJECTIVES: To test the effects of weight loss with and without exercise training (aerobic or resistance) on intra-abdominal adipose tissue (IAAT) and risk factors for cardiovascular disease (CVD). Additionally, CVD risk factors was evaluated before and after weight loss using previously established IAAT cut-points.

SUBJECTS/METHODS: One-hundred and twenty-two overweight premenopausal women were randomly assigned to one of three groups: 1) diet only (Diet); 2) diet and aerobic training (Diet + AT) or; 3) diet and resistance training (Diet + RT); until a BMI of <25 kg/m^2 was reached. Computerized tomography was used to measure IAAT and blood lipids were measured by assay. Evaluations were made before and after weight loss.

RESULTS: Though no group-by-time effects were found after weight loss, we observed significant time-effects for: IAAT (-38.0%, $P<0.001$), total cholesterol (TC) (-2.2%, $P=0.008$), low-density lipoprotein cholesterol (LDL-C) (-4.8%, $P<0.001$), high-density lipoprotein cholesterol (HDL-C) ($+20.2\%$, $P<0.001$), triglycerides (-18.7%, $P<0.001$), TC/HDL-C (-16.3%, $P<0.001$) and LDL-C/HDL-C (-18.0%, $P<0.001$). Following weight loss 40.2% of all participants reduced IAAT to <40 cm^2 (IAAT associated with low CVD risk). Furthermore, only 2.5% of participants had an IAAT >110 cm^2 (IAAT associated with high CVD risk) after weight loss. We also observed that decreases of IAAT were associated with decreased CVD risk factors after weight loss independent of race, changes in %fat mass and changes in maximal oxygen uptake.

CONCLUSIONS: Caloric restriction leading to significant weight loss with or without exercise training appears to be equally effective for reducing IAAT and CVD risk factors.

Keywords

blood lipid; cardiovascular risk factors; diet; fat distribution; visceral fat

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INTRODUCTION

Although obesity and overweight is associated with increased health problems,[1] intra-abdominal adipose tissue (IAAT), sometimes defined as visceral fat, may play the most important role in increasing risk of cardiovascular disease (CVD).[2–4] Subjects with central fat distribution, especially visceral fat, are more likely to have an adverse blood lipid profile. [1,5,6] The combination of small particles of low-density lipoprotein cholesterol (LDL-C), low high-density lipoprotein cholesterol (HDL-C) and high triglycerides (TG) is recognized as the "atherogenic lipid triad", i.e., a major CVD risk factor.[1] Effective strategies for reducing central fat are diet,[7–11] aerobic exercise training (AT),[9,10,12–18] resistance exercise training (RT)[9–11,14,15,17,19,20] and combined (aerobic/resistance) exercise training.[14,15] Similarly, decreases in central fat following weight loss were observed for diet + AT, diet + RT, and diet.[7,9–11,15,17]

Previously investigators have developed cut-points for IAAT that are associated with CVD risk factors.[21] Using cross-sectional data and receiver-operating characteristics analysis, the general consensus identifies that women with IAAT above 110 cm^2 were more likely to have increased risk of CVD, while those women with IAAT below 40 cm^2 were at minimal risk. Frequently studies have designed interventions in obese participants for 12–16 weeks, but these subjects were still obese or overweight after weight loss.[7,9–11,15,17] It is important to determine risk of elevated IAAT and risk factors for CVD in participants who achieve normal weight, a body mass index (BMI) <25 kg/m^2 following weight loss induced by diet with and without exercise training.

Thus, the aims of this study were: 1) to test the effects of a very-low calorie diet with and without exercise training (AT or RT) on IAAT and CVD risk factors after achieving a BMI <25 kg/m^2 and; 2) to evaluate CVD risk factors before and after weight loss in overweight premenopausal women using previously established IAAT cut-points.

MATERIALS AND METHODS

Participants

Sixty European Americans and 62 African Americans healthy premenopausal women (BMI: 27–30 kg/m^2 and age ranged between 23 and 46 years) participated in this study which is a secondary analysis of our JULIET study that was designed to determine what effects exercise training has on body weight maintenance after weight loss. The women were nonsmokers, did not take medications known to metabolism-altering or oral contraceptives, experienced regular menstrual cycles, and normal glucose tolerance (documented by 2-h postprandial blood glucose levels after an oral glucose load). Procedures were approved by the local Institutional Review Board for Human Subjects. All participants provided their informed consent prior to participation in the study.

Study design

Participants were randomly assigned to three different groups of weight loss: diet only (Diet); diet and aerobic exercise training (Diet + AT); or diet and resistance exercise training (Diet + RT). The diet consisted of 800 kcal/day (58–62% carbohydrate, 20–22% fat and 18–

22% protein) and all food was furnished during the weight loss. Participants were instructed to pick up the food at the General Clinical Research Center (GCRC) twice weekly during the weight loss and remain on the 800 kcal/day diet until a BMI of <25 kg/m^2 was reached.

All participants maintained weight-stable state for 4 weeks before evaluations. During the period of weight-stable state, body weight measurements were made 3 days a week for the first 2 weeks, and 5 days a week for the last 2 weeks. All food were provided by the GCRC to ensure stability of weight, i.e., a variation of <1% from initial weight, also maintaining daily macronutrient intake during the final two weeks. Participants were admitted to the GCRC at follicular phase of the menstrual cycle during 4 days for measurements of body composition/fat distribution, blood lipids and maximal oxygen consumed test ($\dot{V}O_{2max}$).

Aerobic training

The AT consisted of continuous walking/or jogging on a treadmill. Each training session started with 3 minutes of warm-up followed by 3–5 minutes of stretching. Participants performed continuous exercise for 20 min during the first week of training at 67% maximum heart rate (HR). Following the 1st week, duration and intensity increased each week. Participants were exercising continuously at 80% of maximum HR on the 8th week for 40 min. Intensity was increased (either speed or grade) when average exercise HR was below 80% of maximum. Participants cooled down after each session for 3–5 min with gradually decreasing exercise intensity.

Resistance training

A warm-up on the bike ergometer or treadmill for 5 minutes and stretching for 3–5 minutes preceded each RT session. RT included elbow flexion, bench press, lateral pull-down, triceps extension, military press, squats, bent-leg sit-ups, leg extension, lower-back extension and leg curl. Following 1 week of training with a light weight (familiarization), strength was measured (one repetition maximum, acronym 1RM). According to the 1RM tests during the 1st week, participants performed one set of 10 repetitions at 65% 1RM. The percentage of 1RM increased on subsequent weeks until 80% 1RM on week 4. On week 5, two sets of 10 repetitions were performed at 80% 1RM for each exercise. Participants rested 2-min between sets. Tests of 1RM was performed every 5 weeks, and adjustments in training resistance were made. In both the Diet + AT and Diet + RT groups, participants trained 3 days/week.

Dual-energy x-ray absorptiometry

A dual-energy x-ray absorptiometry (GE Medical Systems Lunar, Madison, WI) was used to estimate the percentage of fat mass (%FM), using the ADULT software, LUNAR DPX-L version 1.35 (GE Medical Systems Lunar) for scans analysis.

Computerized tomography

A computerized tomography was used to estimate the cross-sectional area of IAAT. The IAAT was measured at L4-L5 vertebrae taking a 5-mm scan for 2 s with the use of HiLight/HTD Advantage scanner (General Electric, Milwaukee, WI) set at 120 kVp (peak kilovoltage) and 40 mA. The range of attenuation for adipose tissue between −30 to −190

Hounsfield units was used.[22] IAAT was separated from subcutaneous fat by encircling the muscle wall surrounding the abdominal cavity with a cursor. A coefficient of variation of <2% for repeated measures of IAAT and an *r* value of 0.99 for both intra- and inter-observer reliability following 20 scans were found.

Blood lipids profile

An Ektachem DT II system was used to measure total cholesterol (TC), HDL-C and TG. LDL-C was estimated using the Friedewald formula.[23] A calibration was performed for the DT II every 6 months with reagents supplied by the manufacturer.

Maximal volume of oxygen consumed

The $\dot{V}O_{2max}$ was measured by a modified Bruce protocol.[24] Oxygen uptake and carbon dioxide production were measured using a MAX-II metabolic cart (Physiodyne Instrument Corporation, Quogue, NY, USA). Certified gases were used to calibrate the gas analyzers. A POLAR Vantage XL HR monitor (Polar Electro Inc., Gays Mills, WI, USA) was used to measure the maximal HR. Standard criteria for plateauing, respiratory exchange ratio (RER) (RER >1.2) and HR (HR within 10 bpm of estimated maximum) were used to verify achievement of $\dot{V}O_{2max}$. A coefficient of variation of <3% for repeated measures of $\dot{V}O_{2max}$ was found in our laboratory.

Statistical analysis

One-way analysis of variance *F* test (ANOVA) or Kruskal-Wallis *H* test was used to compare age, height and days to goal between treatments groups. Also, we used the Chi-squared x^2 test to verify the homogeneous distribution of European Americans and African Americans participants in the treatments groups. Two (time) by three (group) ANOVA with repeated measures for time, were run for weight, BMI, CVD risk factors and fat distribution. Data not normally distributed for ANOVA with repeated measures were transformed to log10. Relative prevalence of participants with elevated CVD risk according to previous IAAT cut-points (<40 cm^2; 40–110cm^2 and >110 cm^2)[21] were determined. In addition, we used partial correlation of Pearson or Spearman to verify associations between delta of CVD risk factors and delta of IAAT (overweight – normal weight) adjusted for race, delta %FM and delta $\dot{V}O_{2max}$. The significance level for statistical analysis was set at a 0.05 (SPSS version 22.0 - IBM SPSS Statistics for Windows, Armonk, NY, USA).

RESULTS

Subject characteristics are presented in Table 1. No significant difference between groups was observed for age, height and race prior to weight loss. Additionally, no significant group effects or interaction of time by group were observed for weight, BMI, and blood lipids. No group effects were also observed for %FM and $\dot{V}O_{2max}$. However, significant time-effects, demonstrating decreases in weight, BMI, %FM, and improvements in $\dot{V}O_{2max}$ and blood lipid profile were observed. A time by group interaction was observed for %FM and $\dot{V}O_{2max}$.

IAAT decreased (significant time effect, Figure 1A) after weight loss. In addition the percentage participants showing an IAAT <40 cm^2 increased from 9.0 to 40.2%, while the percentage of participants with an IAAT between 40–110 cm^2 decreased from 77.0 to 57.4% and those >110 cm^2 decreased from 13.9 to 2.5 % (Figure 1B). However, no significant group-effects, or time x group interaction were observed, demonstrating similar effectiveness for all strategies in improving IAAT.

Significant correlations were observed between delta CVD risk factors with delta IAAT, showing the improvements in CVD risk factors after weight loss were related to decreases of IAAT, even after adjusting for race, changes in %FM and changes in $\dot{V}O_{2max}$ (Table 2). However, no significant correlation was observed between delta HDL-C and delta IAAT.

DISCUSSION

Diet without exercise elicited similar decreases in IAAT and CVD risk factors as diet with exercise (either AT or RT), suggesting that weight loss is a very potent stimulus for improving metabolic health in overweight pre-menopausal women. However, addition of exercise did not have an added effect on reducing CVD risk factors. Interestingly, after achieving normal weight (BMI averaged about 24 kg/m^2), the prevalence of women above the previously established upper cut-point for metabolic disease,[21] IAAT >110 cm^2, were observed (decreasing from 13.9 to 2.5%), while the prevalence of women below the cut-point showing no risk for CVD,[21] IAAT <40 cm^2, increased (from 9.0 to 40.2%), suggesting the majority of women were no longer at risk of being over fat.

It has previously been shown by Ross et al.,[8] that for every kilogram of weight loss induced by diet, the IAAT decreased ~3 to 4 cm^2 or ~2 to 3%. Similarly, we observed a decrease of ~38% of IAAT after ~12 kg weight loss. After adding AT or RT to diet, effects on IAAT were similar, suggesting exercise training does not influence fat distribution changes during energy restriction. Other studies are consistent with this finding.[7,9–11,15,17] It must be acknowledged that the participants in this study underwent a very low calorie diet, so it is possible if not probable the extreme calorie restriction may have influence the results. Consistent with that premise several studies have shown that exercise training induces visceral fat loss despite no weight loss.[7,14,25] It is important to point out that although fat distribution is not favorably affected by exercise training during diet induced weight loss, resistance training conserves fat-free mass during weight loss.[26] In addition, both resistance training and aerobic training prevent regain of any IAAT for one year following weight loss.[27]

A weight loss of ~12 kg in the present study significantly influenced a reduction of the CVD risk factors, improved blood lipids, which appeared to be at least partially mediated by the corresponding reductions in IAAT. In addition, ratios of TC/HDL-C and LDL-C/HDL-C decreased after weight loss to a level considered minimal risk of CVD.[28] However, adding AT or RT to the diet also did not induce greater improvements in CVD risk factors. A similar study, dieting with or without AT or RT induced an ~10 kg weight loss, also demonstrated no exercise effects CVD risk after 16 weeks.[10]

Although exercise training does not seem to affect visceral fat loss during weight loss when using a very low calorie diet, it does not mean that exercise training may not be beneficial. It has already been shown that RT conserve fat-free mass and resting energy expenditure,[26] decreases heart rate during exercise tasks after weight loss,[29] prevents regain of visceral fat[27] and conserves insulin sensitivity[30] for 1 year following weight loss. Thus, considering the importance of exercise on morphological and physiological improvements, it is suggested that exercise training be included for all, even during periods of weight loss.

Strengths of the present study include: all meals were provided through the GCRC during the intervention program ensuring similar diets for all participants. In addition, all participants reduced to a similar BMI, 24–25 kg/m^2.

In conclusion, a diet alone or combined with AT or RT elicit similar improvements on IAAT and CVD risk factors after weight loss. These results are supportive of the prevailing recommendation, that a BMI of less than 25 kg/m^2 is associated with healthful visceral fat levels and thus favorable body composition in pre-menopausal women. It is also supportive of the concept that reduced visceral fat is related to improved CVD risk.

ACKNOWLEDGMENTS

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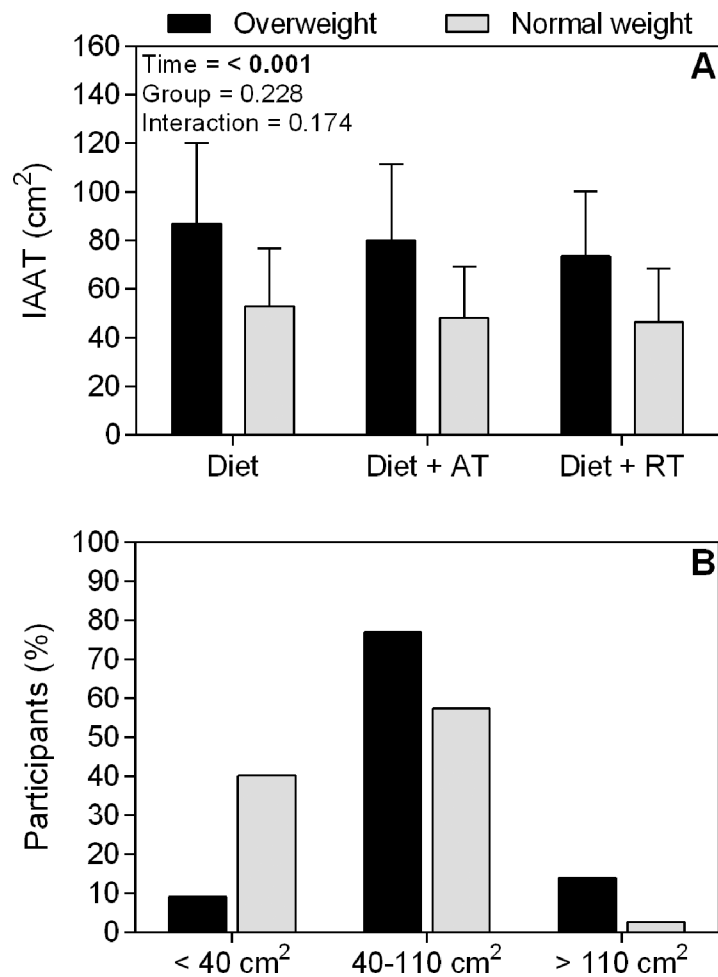
Figure 1.
A) Intra-abdominal adipose tissue (IAAT) during overweight and after achieving normal weight induced by diet only (Diet); diet and aerobic exercise training (Diet + AT), or diet and resistance exercise training (Diet + RT). Values in boldface indicate significant differences ($P < 0.05$). B) Relative number of participants at cut-points of IAAT to predict risk of cardiovascular disease during overweight and after achieving normal weight.

Table 1.

Characteristics, physical fitness and blood lipid profile of the participants during overweight and after achieving normal weight (Mean ± SD)

	Diet (n = 29)			Diet + AT (n = 41)			Diet + RT (n = 52)			P-value		
	Overweight	Normal weight	%	Overweight	Normal weight	%	Overweight	Normal weight	%	Time	Group	Interaction
Age (y)	36.1 ± 5.0	-	-	35.2 ± 6.8	-	-	33.9 ± 6.2	-	-	-	0.292	-
Race (EA/AA)	16/13	-	-	20/21	-	-	24/28	-	-	-	0.737	-
Height (cm)	165.6 ± 6.0	-	-	164.6 ± 6.4	-	-	166.3 ± 6.8	-	-	-	0.472	-
Days to goal	-	146.8 ± 48.7	-	-	163.3 ± 74.6	-	-	153.7 ± 75.4	-	-	0.733	-
Weight (kg)	77.8 ± 7.3	65.3 ± 6.4	-16.0	77.0 ± 6.5	64.7 ± 5.8	-16.0	77.8 ± 7.8	66.0 ± 6.5	-15.1	**< 0.001**	0.778	0.205 *
BMI (kg/m²)	28.2 ± 1.4	23.7 ± 1.1	-15.7	28.4 ± 1.5	23.9 ± 1.1	-15.9	28.0 ± 1.1	23.8 ± 1.1	-15.0	**< 0.001**	0.712	0.268 *
%FM	42.7 ± 3.5	33.2 ± 4.7	-22.4	44.1 ± 3.8	34.1 ± 4.6	-22.8	43.2 ± 3.8	32.4 ± 4.6	-25.1	**< 0.001**	0.269	**0.055**
$\dot{V}O_{2max}$ (ml/kg/min)	27.7 ± 3.8	31.9 ± 4.8	14.9	28.1 ± 4.1	33.9 ± 5.6	20.9	29.1 ± 3.7	33.5 ± 4.7	15.2	**< 0.001**	0.302	**0.029**
TC (mg/dl)	163.6 ± 33.0	152.2 ± 26.2	-5.6	160.7 ± 28.0	160.8 ± 29.2	0.9	153.7 ± 31.9	148.1 ± 28.6	-2.6	**0.008**	0.191	0.136 *
HDL-C (mg/dl)	40.0 ± 10.5	45.0 ± 12.9	12.9	39.9 ± 12.1	48.0 ± 12.7	23.6	37.5 ± 10.8	44.1 ± 12.3	21.5	**< 0.001**	0.389	0.238 *
LDL-C (mg/dl)	104.2 ± 33.3	93.2 ± 24.7	-8.0	102.1 ± 25.1	99.1 ± 23.8	-1.5	97.9 ± 28.5	90.3 ± 23.1	-5.6	**< 0.001**	0.407	0.239 *
TG (mg/dl)	96.8 ± 55.7	70.1 ± 28.2	-17.9	93.4 ± 47.1	68.3 ± 32.1	-20.1	91.3 ± 47.2	68.1 ± 30.9	-18.1	**< 0.001**	0.907	0.951 *
TC/HDL-C	4.4 ± 1.5	3.6 ± 1.0	-15.5	4.3 ± 1.4	3.5 ± 0.9	-16.3	4.4 ± 1.3	3.5 ± 0.9	-16.7	**< 0.001**	0.982	0.844 *
LDL-C/HDL-C	2.8 ± 1.2	2.2 ± 0.9	-17.2	2.8 ± 1.2	2.2 ± 0.8	-17.7	2.8 ± 1.0	2.2 ± 0.8	-18.6	**< 0.001**	0.999	0.821 *

Abbreviations: Diet, diet only; Diet + AT, diet and aerobic exercise training; EA, European-American; AA, African-American; BMI, body mass index; %FM, percentage of fat mass; V̇O2max, maximal oxygen consumption; TC, total cholesterol; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; TG, triglycerides; TC/HDL-C, total cholesterol / high-density lipoprotein cholesterol; LDL-C/HDL-C, low-density lipoprotein cholesterol / high-density lipoprotein cholesterol;

Values in boldface indicate significant differences ($P < 0.05$).

*
Statistical analysis was performed using transformed values in log10.

Table 2.

Partial correlation between CVD risk factors and IAAT adjusted by race, %FM and $\dot{V}O_{2max}$

CVD risk factors	IAAT	
	r	*P*-value
TC	0.20	**0.031**
HDL-C	−0.14	0.120
LDL-C	0.24	**0.008**
TG	0.28	**0.002**
TC/HDL-C	0.31	**0.001**
LDL-C/HDL-C	0.31	**0.001**

Abbreviations: IAAT, delta of intra-abdominal adipose tissue; CVD risk factors, delta of cardiovascular disease risk; TC, delta of total cholesterol; HDL-C, delta of high-density lipoprotein cholesterol; LDL-C, delta of low-density lipoprotein cholesterol; TG, triglycerides; TC/HDL-C, delta of total cholesterol / high-density lipoprotein cholesterol; LDL-C/HDL-C, delta of low-density lipoprotein cholesterol / high-density lipoprotein cholesterol; %FM, delta of percentage of fat mass; $\dot{V}O_{2max}$, delta of maximal oxygen consumption. Values in boldface indicate significant correlations ($P < 0.05$).