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Resistance Exercise and Lipoproteins in Postmenopausal Women

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Abstract

The specific aims of this study were to quantify the effects of 12 weeks of resistance training, as well as a single session of resistance exercise on lipids and lipoproteins in obese, postmenopausal women. Twenty-one obese, postmenopausal women, not on hormone replacement therapy (age = 65.9 ± 0.5 yr; BMI = 32.7 ± 0.8 kg/m²), were randomly assigned to control (n=12) and exercise (n=9) groups matched for age and BMI. For 12 weeks, 3 days/week, the exercise group performed 10 whole body resistance exercises (3 sets at 8-RM). Fasting (10 hr) blood samples were collected immediately prior to and 24 hr after the first and last exercise and control session. Serum was assayed for concentrations of total cholesterol, triglycerides, LDL-C, HDL-C, HDL₂-C, HDL₃-C, non-HDL-C and TC:HDL and LDL:HDL ratios. The exercise group exhibited a significant ($P < 0.01$) improvement in muscular strength, but no change in BMI, body mass or body composition post-training. Total cholesterol, LDL-C and non-HDL-C were significantly ($P < 0.05$) lower in the exercise compared to the control group following the 12 weeks of resistance training. Whole body resistance training provides obese, postmenopausal women a non-pharmacological approach for the reduction of lipid and lipoprotein-cholesterol concentrations.

Keywords

cholesterol; exercise; menopause; obesity

INTRODUCTION

Recently, postmenopausal status has been identified as an independent risk factor for metabolic syndrome [8]. Cho *et al.* reported that after controlling for age and body mass index (BMI), postmenopausal women had a greater prevalence of abdominal obesity, high fasting glucose and triglyceride (Tg) concentrations, and hypertension during the 10 to 14 yr following menopause, which led to an increased risk for metabolic syndrome and cardiometabolic diseases such as type 2 diabetes and dyslipidemia [29,33].

Aerobic exercise is commonly prescribed as a non-pharmacological approach for the treatment of obesity and dyslipidemia; however, the effectiveness of resistance exercise training as a strategy for treating obesity and dyslipidemia remains unresolved. Resistance

exercise training has resulted in inconsistent effects on body mass index, fat mass and percent body fat in obese adults with type 2 diabetes [23]. Similarly, resistance exercise training has been shown to lower serum concentrations of total cholesterol (TC), Tg, low-density lipoprotein cholesterol (LDL-C), and non-high-density lipoprotein cholesterol (non-HDL-C) and raise high-density lipoprotein cholesterol (HDL-C) concentrations [5,7,15,24,26,27]; whereas, several authors have reported no changes in lipids and lipoproteins after resistance exercise training [3,13,26,35]. Inconsistencies in previous findings may be due to differences in the duration of training, types of resistance training (e.g., circuit-machines, free-weights or elastic bands), total resistance exercise volume and intensity of work performed, as well as the participant characteristics.

Few studies have examined the responses of lipid and lipoprotein-cholesterol concentrations to resistance exercise training in postmenopausal women. Fahlman *et al.* [15] examined the responses of lipids and lipoproteins in older (age 70 to 87 yrs), overweight, postmenopausal women randomly assigned to two groups, either an aerobic (3 days/week, 20 to 50 min at 70% heart rate reserve) or resistance (3 days/week, 1 to 3 sets of 8 exercises at 8-repetition maximum (RM)) exercise group for 10 weeks. Following aerobic training, an increase in TC and HDL-C concentrations with a concomitant reduction in LDL-C and Tg concentrations were observed, whereas the resistance training group favorably reduced TC, LDL-C and Tg and increased HDL-C concentrations. In contrast, Elliott *et al.* [13] examined the effects of resistance training (3 days/week, 3 sets of 6 exercises at 80% of 10-RM) for 8 weeks in circulating lipids and lipoproteins, as well as muscular strength in postmenopausal women age 49 to 62 yrs. Despite significant increases in muscular strength, no significant changes in serum concentrations of lipids and lipoproteins were observed post-training. Failure to account for a number of factors including use of hormone replacement therapy (HRT) and post-exercise changes in plasma volume likely contribute to these contradictions in findings.

Similar to findings from resistance training studies, the effects of a single session of resistance exercise on lipids and lipoproteins have been inconsistent as well. In healthy men, Wallace *et al.* [36] reported significant reductions in Tg (20%) and an increase in HDL-C (11%) and HDL₃-C (12%) at 24 hr following a high volume, moderate intensity (7 sets of 7 exercises at 8 to 12-RM) session of resistance exercise. Although, when the participants completed a single bout of low volume, high intensity (4 sets of 7 exercises at 1 to 5-RM) resistance exercise, no significant changes in lipids and lipoproteins were observed. Hill *et al.* [22] observed a significant increase in HDL-C immediately following a single bout of high-intensity (3 sets of 8 exercise at 10-RM) resistance exercise in healthy men; however, the change in HDL-C concentration may have been due to exercise-induced changes in plasma volume post-exercise since plasma volume adjustments were not performed in this investigation.

To our knowledge, no one has examined the effects of a single session of resistance exercise in addition to resistance training on lipids and lipoproteins in obese, postmenopausal women. Therefore, the purpose of this study was to determine the responses of lipid and lipoprotein-cholesterol concentrations to a single session of resistance exercise and 12 weeks of training in obese, postmenopausal women.

METHODS

Participants

Participants recruited for this investigation were sedentary, obese (BMI 30 – 40 kg/m²), postmenopausal women (n=24) between the ages of 60 and 70 years. Postmenopausal was defined as not experiencing a menstrual cycle for five or more years as a result of natural or surgical menopause. Sedentary was defined as not participating in a regular exercise

program for the past six months prior to entry into this investigation. Participants were excluded from participation if they were on HRT or selective estrogen receptor modulators, drugs for lipids and lipoprotein-cholesterol or type 2 diabetes, had severe arthritis, any central or peripheral nervous system disorder, an acute or chronic infection, a major affective disorder, HIV or auto-immune disorders, metabolic disorders, bedridden within three months of the study, experienced a stroke, used tobacco, used oral steroids, or had surgery within the previous six months. Participants were required to be weight stable for at least the previous three months.

After obtaining approval from their personal physician, each potential participant was given a medical screening by our study physician. The medical screening included a review of their medical history and a physical exam, which included an upper and lower extremity examination to identify musculoskeletal or flexibility limitations, and a screening for dementia. Potential participants completed a submaximal treadmill stress test while blood pressure and ECG were monitored with a physician present. The treadmill test began with a 3 min warm-up at 2 mph. The workload was increased by approximately one MET every 2 min until heart rate reached 85% of the age-predicted maximum. Informed consent was obtained from each potential participant prior to participation. This study was approved by the Institutional Review Boards at Texas Christian University and John Peter Smith Hospital. In addition, this study has been conducted in accordance with the ethical principles of the Declaration of Helsinki [21].

Exercise and Control Protocols

Participants cleared for participation were randomized, matched for age and BMI into either an exercise group (n=12) or a control (n=12) group. Three exercise participants had to withdraw from the study due to time constraints of the training sessions. Nine exercise participants completed the training study. Both groups completed a week-long acclimation period to the following resistance exercises: chest press, “lat” pull-down, shoulder press, seated rows, leg abduction, leg adduction, chest flies, leg press, leg curl, and leg extension. They performed three acclimation exercise sessions on non-consecutive days. On acclimation day 1, participants were taught proper lifting techniques for each of the ten exercises to minimize the risk of injury. In addition, each participant’s 8-RM was assessed for each of the 10 resistance exercises. On the second acclimation day, participants performed 3 sets of each exercise at 50% of their estimated 1-RM. During the first two sets, participants performed eight repetitions and the last set was completed to volitional fatigue. Eight-RM for all exercises was reassessed on acclimation day 3. All acclimation and exercise sessions were preceded by 5-10 min of cycle ergometry or walking. All acclimation sessions and resistance exercise sessions were completed in the weight room at the Texas Christian University’s Recreation Center using Nautilus (Vancouver, WA) and Cybex equipment (Medway, MA) under the supervision of trained technicians.

Exercise training was completed for 12 weeks on non-consecutive days (3 days/week, 10 exercises for 2 sets at 8-RM and a 3rd set to failure). If the participant was able to perform more than 12 repetitions on the third set, weight was increased on the following workout day. Following the 12-weeks, both the exercise and control group completed an 8-RM test for all exercises. Two participants from the exercise group and one participant from the control group were unable to complete the final 8-RM test due to personal reasons. Participants assigned to the control group did not undergo supervised exercise sessions, but reported to the lab twice per week for social interaction or education classes (e.g., stretching, talks on books, health education). All participants were asked to maintain their “normal” diet during the intervention period and to consume no alcohol the days prior to any blood sampling. Subjects recorded all food consumed during the 24 hr prior to the pre-training blood sample which was to represent a “normal” day’s diet. After the 12 week intervention,

they were given a copy of that food log and asked to record and consume the same meals during the 24 hr prior to the post-training blood sample.

Blood Collection

For the experimental trials, participants reported to the lab after an overnight fast (10 hr) between 0530 and 0730. Prior to all blood collection, participants were asked to rest in a seated position for 20 min. Blood samples were collected into EDTA treated and untreated vacutainer tubes (BD Diagnostics, Franklin Lakes, NJ) immediately before and 24 hr following both the first and last control and exercise sessions. Blood collection was performed by a trained technician. Hematocrit and hemoglobin concentration were quantified immediately following collection of blood using the microcapillary microcentrifugation method and cyanamethemoglobin method, respectively. Serum and plasma were separated from the blood by centrifugation (5000×g, 10 min, 4°C) and stored at -80°C until analysis.

Body Composition

To assess the effects of resistance training on body mass and body composition, measurements were performed on all participants prior to the first and following the last exercise or control session. Body mass was measured to the nearest 0.1 kg with a calibrated digital scale and height was measured to the nearest 1 mm with a stadiometer. Body mass index was then calculated by dividing body weight (kg) by height squared (m²). In addition, body density was estimated using a seven-site skinfold procedure [25], and percent body fat was estimated from body density using the Siri equation [30]. Skinfold measurements were performed by the same trained technician for both pre- and post-training trials.

Analytical Methods

Total cholesterol and Tg concentrations were determined using standard enzymatic colorimetric techniques (Kit #TR13421 and #TR22321, respectively; ThermoFisher Scientific, Pittsburgh, PA). The serum concentration of HDL-C and HDL₃-C were determined according to methods by Warnick and Albers [37] and Gidez et al. [18]. Serum concentration of HDL₂-C was calculated as the difference between HDL-C and HDL₃-C. The Friedewald equation was used to estimate the concentration of LDL-C [16]. Non-HDL-C concentration was calculated by subtracting HDL-C from TC. The coefficients of variation for TC, Tg, HDL-C, and HDL₃-C were 4.8%, 6.8%, 2.9%, and 3.1%, respectively.

Statistical Analysis

A $2 \times 2 \times 2$ (group $\times$ training status $\times$ time) MANOVA with repeated measures on the second and third factors was employed to identify significant main effects and interactions. A MANOVA was chosen because the lipid and lipoprotein-cholesterol variables were interrelated. The independent factors were group (exercise and control), training status (pre-training; post-training), and time of blood sampling (immediately before and 24 hr following the first and last exercise and control session). The dependent variables were TC, Tg, LDL-C, HDL-C, HDL₂-C, HDL₃-C and non-HDL-C concentrations, and TC:HDL and LDL:HDL ratios. When the MANOVA detected statistical differences the Student Newman-Keuls'-test was used to determine where the differences existed. A 2×2 (group $\times$ time) ANOVA with repeated measures was used to assess significant changes in muscular strength (8-RM), BMI, body mass and percent body fat following the 12-week study. The criterion reference for statistical significance was set at $P < 0.05$.

RESULTS

Descriptive characteristics of the participants are in Table 1. No significant differences were observed between the control and exercise groups with respect to age, height, weight, or percent body fat prior to the experimental trials. Despite matching participants for age and BMI at the beginning of the study, BMI was significantly ($P = 0.005$) different between the control and exercise group due to participant drop-out ($n=3$) in the exercise group during the course of the investigation. Percent body fat and body mass were not significantly different between groups. At the end of the 12 weeks, no significant change in body weight ($P = 0.112$), BMI ($P = 0.118$) or percent body fat ($P = 0.985$) were observed for either the control or exercise group. While no significant changes in BMI, body mass or body composition occurred post-training, for each of the exercises performed we observed significant ($P < 0.01$) increases in muscular strength (Table 2).

No significant changes in plasma volume were observed for either the control ($P = 0.342$) or exercise ($P = 0.464$) group across time; however, adjustments in post-exercise plasma volume were performed for all lipid and lipoprotein-cholesterol concentrations. It has been shown that small non-significant post-exercise changes in plasma volume can have physiological and statistical relevance for lipids and lipoproteins [38].

Baseline lipid and lipoprotein-cholesterol concentrations were normally distributed. Mean (SE) baseline lipid and lipoprotein-cholesterol concentrations ($\text{mmol}\cdot\text{L}^{-1}$; $n=21$) for TC, Tg, LDL-C, HDL-C, HDL₂-C, HDL₃-C and non-HDL-C were 5.25 (0.25), 1.25 (0.10), 32.51 (0.23), 1.42 (0.08), 0.93 (0.06), 0.50 (0.04) and 3.82 (0.23), respectively. The mean (SE) baseline TC:HDL ratio was 3.8 (0.2) and LDL:HDL ratio was 2.4 (0.2). Post-exercise responses of lipid and lipoprotein-cholesterol concentrations and ratios are shown in Table 3. The repeated measures MANOVA revealed a significant ($P < 0.05$) group by training interaction for TC, LDL-C, non-HDL-C and HDL₃-C concentrations. Post hoc analyses revealed that TC, LDL-C, non-HDL-C and HDL₃-C concentrations were significantly ($P < 0.05$) lower in the exercise group than the control group post-training (Figure 1). In addition, post-training TC, LDL-C and non-HDL-C concentrations were significantly ($P < 0.05$) reduced 16.0%, 23.0% and 20.2% in the exercise group when compared to pre-training concentrations. A significant ($P = 0.011$) time effect revealed a reduction in Tg concentration. No significant effects or interactions were observed for HDL-C or HDL₂-C concentrations, or for TC:HDL and LDL:HDL ratios 24 hr after resistance exercise.

DISCUSSION

The key findings of our study were that a single bout of resistance exercise did not modify circulating lipid and lipoprotein-cholesterol concentrations 24 hr post-exercise; however, TC, LDL-C, non-HDL-C and HDL₃-C concentrations were 23.6%, 28.5%, 27.0% and 24.1%, respectively lower in the exercise group when compared to the control group following 12 weeks of resistance training in obese, postmenopausal women. In addition, the exercise group experienced significant increases in muscular strength. Interestingly, despite significant differences between groups in lipid and lipoprotein-cholesterol concentrations post-training, 12 weeks of resistance training provided no significant changes in BMI, body mass or body composition. This is in contrast to previous observations that reductions in TC, LDL-C and non-HDL-C are dependent on reductions in body mass or percent body fat [12].

When quantifying either the acute effects or training effects of exercise on the concentration of lipids and lipoproteins, it is critical to adjust for changes in plasma volume in order to distinguish between changes in lipoprotein concentration and adjustments in vascular volume. In addition, HRT has been shown to alter lipids and lipoproteins [31,32], as well as

redistribute body fat to the abdomen [31]. Failure to account for possible shifts in plasma volume and the use of HRT when examining the effects of resistance training in postmenopausal women may account for the inconsistencies in results among previous studies. In the present study, we made adjustments in post-exercise plasma volume and excluded participants who were taking HRT to control for HRT induced changes in lipids and lipoproteins, which is a clear strength of our study.

Based on the National Cholesterol Education Program's Adult Treatment Panel III classification [1], 47.6% of the participants had borderline-high or high TC ($5.2 \text{ mmol}\cdot\text{L}^{-1}$), 42.9% had borderline-high or high LDL-C ($3.4 \text{ mmol}\cdot\text{L}^{-1}$), 33.3% of the participants had low HDL-C ($1.3 \text{ mmol}\cdot\text{L}^{-1}$), and 14.3% had high Tg ($1.7 \text{ mmol}\cdot\text{L}^{-1}$) concentrations. When the baseline lipid data from our participants were compared to U.S. national averages for women of similar age from the National Health and Nutrition Examination Survey III 1999-2002 data [6], the mean serum concentrations observed in the current study appear similar or slightly lower for TC (5.2 vs. 5.8 $\text{mmol}\cdot\text{L}^{-1}$), LDL-C (3.3 vs. 3.4 $\text{mmol}\cdot\text{L}^{-1}$), HDL-C (1.5 vs. 1.5 $\text{mmol}\cdot\text{L}^{-1}$), and Tg (1.3 vs. 1.6 $\text{mmol}\cdot\text{L}^{-1}$). In comparison to previous studies with postmenopausal women performing resistance training [4,9,13,15], the participants in our study had a greater BMI. In addition, our participants had similar baseline serum concentrations of TC, LDL-C, Tg and HDL-C when compared to lipid profiles reported by Fahlman *et al.* [15] and lower lipid profiles than reported by Bemben *et al.* [4], Colado *et al.* [9] and Elliott *et al.* [13].

In overweight, postmenopausal women Fahlman *et al.* [15] reported that following 12 weeks of resistance training, TC and LDL-C concentrations were reduced by 5.1% and 23.2%, respectively. Similarly, following 12 weeks of resistance training in the current study, TC and LDL-C concentrations in the exercise group were 23.6% and 28.5% lower than the control group, respectively. The difference in TC and LDL-C between groups at post-training can be attributed to reduced TC (16.0%) and LDL-C (23.0%) concentrations in the exercise group from pre- to post-training. In contrast to our findings, several studies using traditional resistance exercise techniques, as well as elastic bands and aquatic resistance exercises have not demonstrated any changes in TC and LDL-C concentrations after training for up to 24 weeks. Elliott *et al.* [13] had overweight, postmenopausal women train for 8 weeks, performing 6 exercises, 3 times per week at 80% of 10-RM. It is likely that the training duration and volume of work performed was insufficient to modify TC and LDL-C concentrations.

Recently, non-HDL-C concentration has become a powerful predictor of CVD [17,26]. This is largely due to the fact that non-HDL-C contains all known atherogenic lipid particles (lipoprotein (a), LDL, intermediate density lipoprotein, and very low density lipoprotein). Recent research has suggested that non-HDL-C may be a better predictor of CVD morbidity [28] and mortality [10] than LDL-C concentration. In the present study, a significant group by training interaction was observed for non-HDL-C concentration, which revealed a 27.0% lower non-HDL-C concentration in the exercise group compared to the control group post-training. The 20.2% reduction in the exercise group from pre- to post-training best explains the observed difference between groups. Interestingly, a meta-analysis of randomized clinical trials examining the responses of lipid and lipoproteins to resistance training, reported that the mean percent reduction in TC, LDL-C and non-HDL-C concentrations post-training were 2.7%, 4.6%, and 5.6%, respectively [26]. In comparison, the large percent reductions in TC, LDL-C and non-HDL-C concentrations observed in the present study may have a clinical significance.

In clinical trials examining effects of statins on the risk for coronary heart disease morbidity and mortality, an approximate 30-40% reduction in risk is observed with the majority of the

effect directly related to the absolute reduction in LDL-C [2,14]. Moreover, a meta-analysis of 14 randomized trials of statin therapy concluded that each 1 mmol·L⁻¹ reduction in LDL-C was matched by a 4.4% reduction in risk of a nonfatal coronary heart event. In this study, 12 weeks of resistance exercise training reduced LDL-C by 0.77 mmol·L⁻¹, which may translate to a 3.4% reduction in risk of a nonfatal coronary heart event in this group of women.

In the present study, resistance training did not reduce Tg concentration. This observation is similar to results reported by Elliott *et al.* [13] and Bembien *et al.* [4]. Participants in these studies were hypertriglyceridemic and overweight. In contrast, Fahlman *et al.* [15] observed a 22.5% reduction in Tg concentration in overweight, but non-obese postmenopausal women with fasting Tg concentration similar to participants in the present study. Based on these observations, hypertriglyceridemia or obesity may attenuate the Tg lowering effects of resistance training in postmenopausal women.

Total energy expenditure has been associated with changes in lipid and lipoproteins in a dose-response relationship between the number of calories expended and the percent change in lipid and lipoprotein-cholesterol concentrations [12]. Recently, Dalleck *et al.* [11] reported a dose-response relationship between acute aerobic exercise duration and increases in HDL-C concentration, as well as reductions in body mass and percent fat in postmenopausal women. In contrast to our findings where resistance training did not change HDL-C or HDL-C subfractions, 16 to 24 weeks of resistance training using elastic bands significantly increased HDL-C by 12-13% in postmenopausal women despite that the participants were hypercholesterolemic [4,9]. In addition, Fahlman *et al.* [15] reported a 7.7% increase in HDL-C after 12 weeks of traditional resistance training. Interestingly, the changes in HDL-C concentration may be independent of changes in fat mass and may depend on caloric expenditure to a greater extent. Only Colado *et al.* [9] observed reductions in BMI and fat mass with concomitant increases in HDL-C concentration post-training. Despite a significant difference in HDL₃-C between groups post-training in the present study, this difference is most likely due to a non-significant 27.4% increase in HDL₃-C in the control group from pre- to post-training and not a resistance exercise training effect. This is evident by the 5.3% difference between pre- and post-training HDL₃ concentration in the exercise group. It remains unclear as to why the resistance training in the present study did not change HDL-C, HDL₂-C or HDL₃-C concentrations, except that the caloric expenditure was below the caloric threshold necessary to increase HDL-C.

In the present study, a single session of resistance exercise did not significantly influence circulating lipid and lipoprotein-cholesterol 24 hr post-exercise for either the first or last bout of exercise. To our knowledge this is the first study that has quantified the effects of acute resistance exercise on lipids and lipoproteins in obese, postmenopausal women. The majority of studies examining the acute responses of lipids and lipoproteins have used aerobic exercise [12]. A single session of aerobic exercise has been reported to reduce Tg and increase HDL-C concentrations at 24 and 48 hr post-exercise [19]. The reduction in Tg and increase in HDL-C concentrations are generally attributed to an increased lipoprotein lipase (LPL) activity [19]. The increase in LPL activity may in part be due to an effort to restore intramuscular Tg stores acutely depleted during aerobic exercise. In contrast to aerobic exercise, resistance exercise has been characterized by an increased utilization of anaerobic energy sources (e.g., ATP-phosphagenic cycle and glycolysis). However, heavy volume resistance exercise has been demonstrated to reduce intramuscular Tg levels [20].

Previous studies that have examined the responses of lipid and lipoprotein-cholesterol concentrations to a single session of resistance exercise have reported significant increases in HDL-C and HDL₃-C concentrations 24 hr following whole-body, high volume (7

exercises of 7 sets at 8 to 12-RM) exercises in healthy, trained men [36]. Recently, Tsekouras *et al.* [34] reported a significant reduction in Tg following a single bout of whole-body isokinetic resistance exercise (12 exercise of 3 sets at 10 reps at 80% of maximum peak torque) in healthy, untrained men. In the present study, a significant time effect for Tg was observed; however, this was attributed to non-significant changes at 24 hr for both pre- and post-training trials in both the control and exercise groups. When examining the changes of the exercise group, Tg concentration was reduced by 13.5% and 16.6% at 24 hr for the pre- and post-training trials, respectively. Triglyceride concentration in the control group was -7.4% and -9.5% lower at 24 hr for the pre- and post-training trials, respectively. Given the reductions in Tg concentration were observed in both groups, it is difficult to determine if these changes were exercise-induced or due to normal day-to-day variations.

A limitation of this study was that dietary consumption of the participants was not strictly monitored or recorded, but participants were instructed and consistently encouraged to maintain their “normal” diet during training and prior to blood collection. Based on the observation that the changes in lipid and lipoproteins observed post-training were exclusive to the exercise group, this limitation does not appear to be a critical factor in the outcomes of this study. A second limitation of this study was the significant difference in BMI between the control and exercise groups at baseline. The small difference (8.8%) between groups may have had an influence on the post-training responses of the lipids and lipoproteins; however, this is unlikely since BMI remained unchanged and both groups were obese despite the significant difference. In addition, percent body fat was not different between groups. Another potential limitation in this study was that body composition was estimated by the skinfold technique. To ensure test-retest reliability of the measures, the same trained technician performed all skinfold measures. Since body composition was not used to define obesity in this study we do not feel that it is a critical factor in the outcomes of this study.

In summary, the primary findings in our study of obese, postmenopausal women were that a single bout of resistance exercise of 10 exercises at an 8-RM intensity did not modify lipids and lipoproteins; however, 12 weeks of resistance training resulted in significant reductions in TC, LDL-C and non-HDL-C concentrations and increased the muscular strength of the participants. In addition, the relatively large, percent reductions in TC, LDL-C and non-HDL-C concentrations following the 12 weeks of resistance training may provide a significant reduction in nonfatal coronary heart disease events. Interestingly, in this study the effect of resistance training on lipid and lipoprotein-cholesterol concentrations was independent of changes in BMI and body composition. Furthermore, these results provide support for the use of whole body resistance training in obese, postmenopausal women as a non-pharmacological approach for the reduction of lipid and lipoprotein-cholesterol concentrations.

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Figure 1.

Table 1

Descriptive characteristics of the participants.

	Control (n=12)	Exercise (n=9)	<i>P</i>
Age (yr)	67.0 ± 0.6	64.4 ± 0.7	0.879
Height (cm)	158.0 ± 1.7	163.8 ± 2.2	0.534
Weight (kg)	85.0 ± 3.7	83.5 ± 3.3	0.108
BMI (kg/m ²)	34.0 ± 1.3	31.0 ± 0.5	0.005
Body fat (%)	36.3 ± 0.9	34.4 ± 0.9	0.283

Note. Data are mean ± SE.

Table 2

Percent change in muscular strength following 12 weeks of resistance exercise training.

	Exercise (n=7)*	Control (n=11)	P
Chest Press	35.6 ± 6.8	5.2 ± 2.6	<0.001
Lat pull-down	21.1 ± 7.4	3.3 ± 1.8	0.011
Shoulder press	47.4 ± 11.2	0.1 ± 2.6	<0.001
Seated rows	31.0 ± 5.7	0.6 ± 1.7	<0.001
Leg abduction	35.7 ± 12.1	3.9 ± 2.3	0.005
Leg adduction	58.1 ± 16.3	2.5 ± 3.0	<0.001
Chest flies	48.8 ± 12.1	1.9 ± 2.6	<0.001
Leg press	29.2 ± 5.7	-0.5 ± 2.2	<0.001
Leg curl	69.4 ± 22.5	8.6 ± 4.5	0.004
Leg extension	41.6 ± 8.8	8.8 ± 3.1	<0.001

Note. Values are mean ± SE in %.

* Two participants from the exercise group and one participant from the control group were unable to complete the last 8-RM test due to personal reasons.

Table 3

Serum lipid and lipoprotein-cholesterol concentrations before and after resistance exercise training.

	Control (n = 12)				Exercise (n = 9)			
	Pre-Training		Post-Training		Pre-Training		Post-Training	
	Pre-	+24 hr	Pre-	+24 hr	Pre-	+24 hr	Pre-	+24 hr
Tg	1.24 ± 0.13	1.14 ± 0.16	1.48 ± 0.23	1.34 ± 0.16	1.26 ± 0.16	1.09 ± 0.05	1.25 ± 0.28	1.04 ± 0.14
TC	5.21 ± 0.37	5.44 ± 0.32	5.75 ± 0.64	5.89 ± 0.64	5.30 ± 0.34	5.31 ± 0.40	4.63 ± 0.54	4.26 ± 0.41
LDL-C	3.21 ± 0.35	3.46 ± 0.29	3.51 ± 0.54	3.69 ± 0.57	3.30 ± 0.30	3.39 ± 0.33	2.70 ± 0.47	2.45 ± 0.30
HDL-C	1.42 ± 0.11	1.46 ± 0.10	1.56 ± 0.09	1.59 ± 0.12	1.43 ± 0.11	1.42 ± 0.14	1.36 ± 0.13	1.34 ± 0.13
HDL2-C	0.94 ± 0.08	0.90 ± 0.07	0.93 ± 0.09	0.89 ± 0.12	0.91 ± 0.09	0.87 ± 0.12	0.82 ± 0.12	0.86 ± 0.11
HDL3-C	0.49 ± 0.06	0.55 ± 0.05	0.63 ± 0.04	0.70 ± 0.04	0.51 ± 0.04	0.55 ± 0.05	0.53 ± 0.04	0.48 ± 0.05
non-HDL-C	3.78 ± 0.34	3.99 ± 0.28	4.19 ± 0.61	4.30 ± 0.58	3.88 ± 0.31	3.89 ± 0.33	3.27 ± 0.46	2.92 ± 0.34
TC:HDL	3.8 ± 0.3	3.8 ± 0.2	3.7 ± 0.4	3.8 ± 0.3	3.8 ± 0.3	3.9 ± 0.3	3.5 ± 0.3	3.3 ± 0.3
LDL:HDL	2.4 ± 0.3	2.4 ± 0.2	2.3 ± 0.3	2.3 ± 0.3	2.4 ± 0.2	2.5 ± 0.3	2.3 ± 0.3	2.3 ± 0.3

Note. Values are mean ± SE in mmol·L⁻¹. To convert Tg from mmol·L⁻¹ to mg·dL⁻¹ divide by 0.01129. To convert cholesterol from mmol·L⁻¹ to mg·dL⁻¹ divide by 0.02586. Pre-Training, the first exercise or control session before the 12 weeks of training; Post-Training, the last exercise or control session after the 12 weeks of training; Pre-, immediately prior to the exercise or control session; +24 hr, 24 hr following the exercise or control session; Tg, triglyceride; TC, total cholesterol; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; HDL2-C, high-density lipoprotein cholesterol fraction 2; HDL3-C, high-density lipoprotein cholesterol fraction 3; non-HDL-C, non-HDL cholesterol; TC:HDL, total cholesterol to high-density lipoprotein cholesterol ratio; low-density lipoprotein cholesterol to high-density lipoprotein cholesterol ratio.