# Serum Lipoproteins in Overweight/Obese Postmenopausal Women: A One-Year Exercise Trial

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*[1]Fred Hutchinson Cancer Research Center, Seattle, WA; [2]Department of Epidemiology, University of Washington, Seattle, WA; [3]Department of Epidemiology and Public Health, Yale University, New Haven, CT; [4]Department of Public Health Sciences, University of Alberta, Edmonton, CANADA; [5]Channing Laboratory, Brigham and Women's Hospital and Harvard Medical School, Boston, MA; and [6]Department of Medicine, University of Washington, Seattle, WA*

ABSTRACT

MOHANKA, M., M. IRWIN, S. R. HECKBERT, Y. YASUI, B. SORENSEN, J. CHUBAK, S. S. TWOROGER, C. M. ULRICH, and A. MCTIERNAN. Serum Lipoproteins in Overweight/Obese Postmenopausal Women: A One-Year Exercise Trial. *Med. Sci. Sports Exerc.*, Vol. 38, No. 2, pp. 231–239, 2006. **Introduction:** This analysis was conducted to study the effect of a 1-yr moderate-intensity aerobic exercise program on serum lipoproteins among overweight/obese postmenopausal women. **Methods:** We randomized 173 sedentary (mean $\dot{V}O_{2max}$ = 20.2 mL·kg$^{-1}$·min$^{-1}$), overweight/obese women (body mass index (BMI) 25.0–42.0 kg·m$^{-2}$ or body fat > 33% if BMI 24.0–25.0) aged 50–75 yr, not using hormone therapy, living in the Seattle area, to an exercise intervention or stretching control group. The exercise intervention included facility and home-based exercise (45 min, 5 d·wk$^{-1}$ of moderate-intensity sports or recreational exercise). Total cholesterol (TC), triglycerides, and high-density lipoprotein (HDL) were determined by chemical assay; low-density lipoprotein (LDL) was then calculated. **Results:** Of the 173 women, 170 (98.3%) completed the study with exercisers averaging 176 (SD 91) min·wk$^{-1}$ of moderate- to vigorous-intensity (60%–75% HRmax) exercise, expending approximately 3828 kJ·wk$^{-1}$ (SD 2053). Exercisers, compared with stretchers, significantly increased their $\dot{V}O_{2max}$ (+11%, $P < 0.001$) and lost more body weight (−1.4 kg, $P < 0.05$), DEXA-% total body fat (−1.0, $P < 0.005$), $L_{4-5}$ computed tomography intraabdominal fat (−8.6 cm$^2$, $P < 0.05$) and subcutaneous abdominal fat (−28.8 cm$^2$, $P < 0.005$) after 12 months. Mean (SD) baseline lipoprotein levels for TC, LDL, HDL, and triglycerides were 231 (39), 152 (39), 52 (12), and 135 (65) mg·dL$^{-1}$, respectively. We observed no significant change in serum lipoprotein levels among exercisers compared with stretchers at either 3 or 12 months postrandomization. We did not observe a statistically significant effect of exercise on serum lipoproteins in subgroups that changed their intraabdominal and subcutaneous fat, percent total body fat, or $\dot{V}O_{2max}$ the most, or that adhered to the exercise regimen the most over 1 yr. **Conclusion:** It appears that a year long moderate-intensity exercise program alone does not significantly alter serum lipoprotein levels among overweight/obese postmenopausal women. **Key Words:** LIPID, PHYSICAL ACTIVITY, CHOLESTEROL, RANDOMIZED TRIAL, OBESITY, ELDERLY

The NIH Consensus Statement (29) has recommended that all adults perform moderate-intensity physical exercise for about 30 min on most or all days of the week, but only about 22% of adults in the United States engage in this level of regular exercise (29). Physically inactive persons are often overweight (37) and have greater amounts of visceral fat than those who are active (31); both of these characteristics have been associated with an adverse lipid profile (16,27,30,39). Observational studies have reported an association of physical activity with a favorable lipid profile (9,11,21).

Few randomized exercise trials have studied the effects of exercise alone on lipid levels in postmenopausal women. King et al. (19) reported significant increases in HDL with both a home-based, high-intensity and a more frequent, but low-intensity, exercise regimen compared with a high-intensity, facility-based exercise regimen in a 2-yr trial. Stefanick et al. (33), however, observed no changes in serum lipoproteins from exercise alone in a 1-yr factorial trial of facility and home-based exercise and diet. Binder

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et al. (3) demonstrated a decrease in total cholesterol (TC) and LDL cholesterol with 45-min, thrice weekly vigorous exercise regimen for 11 months. Fahlman et al. (10) compared aerobic and resistance training with controls in a 10-wk trial. Both aerobic and resistance training resulted in a significant increase in HDL and a decrease in triglycerides. Resistance training also resulted in significantly lower LDL and total cholesterol, compared with the control group. Cauley et al. (6) and Asikainen et al. (2) did not observe significant changes in lipoproteins in walking trials up to 2 yr. Thus, the effects of exercise on serum lipoproteins have been inconsistent and have been studied inadequately, particularly in overweight/obese postmenopausal women.

This analysis was done to study the effects of a 1-yr home- and facility-based, moderate-intensity physical exercise program on lipoproteins among sedentary overweight/obese postmenopausal women. We hypothesized that, with exercise, HDL would increase and LDL would decrease, thereby improving the lipid profile. We earlier reported from this trial that this regimen of moderate-intensity exercise improved aerobic fitness and decreased total body and abdominal fat (17). Other studies suggested that high levels of intraabdominal fat (4,16,27,30,39) and low aerobic fitness (8) are associated with adverse lipid profiles. Therefore, in this study we expected to see an improved lipid profile, particularly in those with the greatest decreases in total body fat, intraabdominal and subcutaneous fat, and the greatest increase in aerobic fitness.

## METHODS

Details of the study rationale and design have been reported (23). Briefly, this was a randomized trial comparing the effects of a 1-yr moderate-intensity aerobic exercise intervention versus stretching control among sedentary and overweight/obese postmenopausal women. We recruited 173 sedentary (exercising <60 min·wk$^{-1}$) and overweight/obese (BMI 25.0–42.0 kg·m$^{-2}$, or body fat > 33% if BMI 24.0–25.0) postmenopausal women aged 50–75 yr from the greater Seattle, WA area through a combination of mass mailings and media placements. Diabetes, current smoking, alcohol intake over two drinks a day, use of hormone replacement therapy in the past 6 months, diet drugs, structured program, or surgery for weight loss were among the exclusion criteria. Women with comorbidity likely to limit exercise were also excluded from the study. Women were assigned to exercise or control groups using random number generation in a BMI-stratified randomization process (<27.5 kg·m$^{-2}$ vs ≥27.5 kg·m$^{-2}$). Written informed consent was obtained from the subjects, and the institutional review board at the Fred Hutchinson Cancer Research Center approved this research study.

The exercise intervention consisted of at least 45 min of moderate-intensity exercise, 5 d·wk$^{-1}$ for 12 months. Participants were required to attend the three offered supervised sessions per week at a study facility (University of Washington or a commercial gym) during months 1 through 3 and to exercise 2 d·wk$^{-1}$ at home. For months 4 through 12, they were required to attend at least one of the three offered sessions per week at a study facility and to exercise 4 d·wk$^{-1}$, either at home or at the facility. Because the sample of women recruited into the study were physically inactive with low aerobic fitness levels, and because our goal was to promote moderate-intensity physical activity, the training program started at 40% of observed maximal HR for 16 min per session and gradually increased to 60–75% of maximal HR for 45 min per session by week 8, where it was maintained for the duration of the study (15). Participants wore Polar HR monitors during exercise sessions. Facility sessions consisted of treadmill walking and stationary bicycling. Strength training to fatigue, consisting of 2 sets of 10 repetitions of leg extension, leg curls, leg press, chest press, and seated dumbbell row, was recommended, but not required, to decrease risk of injury and maintain joint stability. A variety of home exercises were suggested and encouraged, including walking, aerobics, and bicycling. Participants were encouraged to wear their heart-rate monitors when exercising at home. Participant adherence was assessed via daily activity logs, on which participants reported the type and duration of exercise they performed.

Control participants attended one weekly 60-min stretching and relaxation session for the 12 months and were asked not to change other exercise habits. Both exercisers and stretchers were asked not to modify their dietary intake for the duration of the study. After their assessments at the conclusion of the study, control women were offered three training sessions with the exercise physiologist.

### Data Collection

We collected detailed information on demographics, medical history, medication use, and self-reported dietary intake at the baseline and at 3 and 12 months. Anthropometric and physical measurements, including height, weight, waist and hip circumferences, blood pressure, and resting HR were taken in duplicate and averaged. The exercise-intervention participants kept daily activity logs (type of exercise and duration) of all sports or recreational activities they performed throughout the trial. These logs were reviewed by exercise trainers for completeness and clarity every week. The compendium of physical activity (1) was used to assess energy expenditure for each activity. Only sports and recreational activities of at least 3.0 METs were included in the assessment of caloric expenditure. A self-administered adaptation of the Minnesota physical activity questionnaire (34) was used to assess current physical activity every 3 months. Dietary habits were assessed using a 120-item food frequency questionnaire (FFQ) at baseline, and at 3 and 12 months. At baseline and at 12 months, total body fat using DEXA whole-body scanner (Hologic QDR 1500, Hologic Inc, Waltham, MA), and intraabdominal and subcutaneous fat compartments at the $L_{4-5}$ level using a single slice of computed tomography (CT) (General Electric model CT 9800

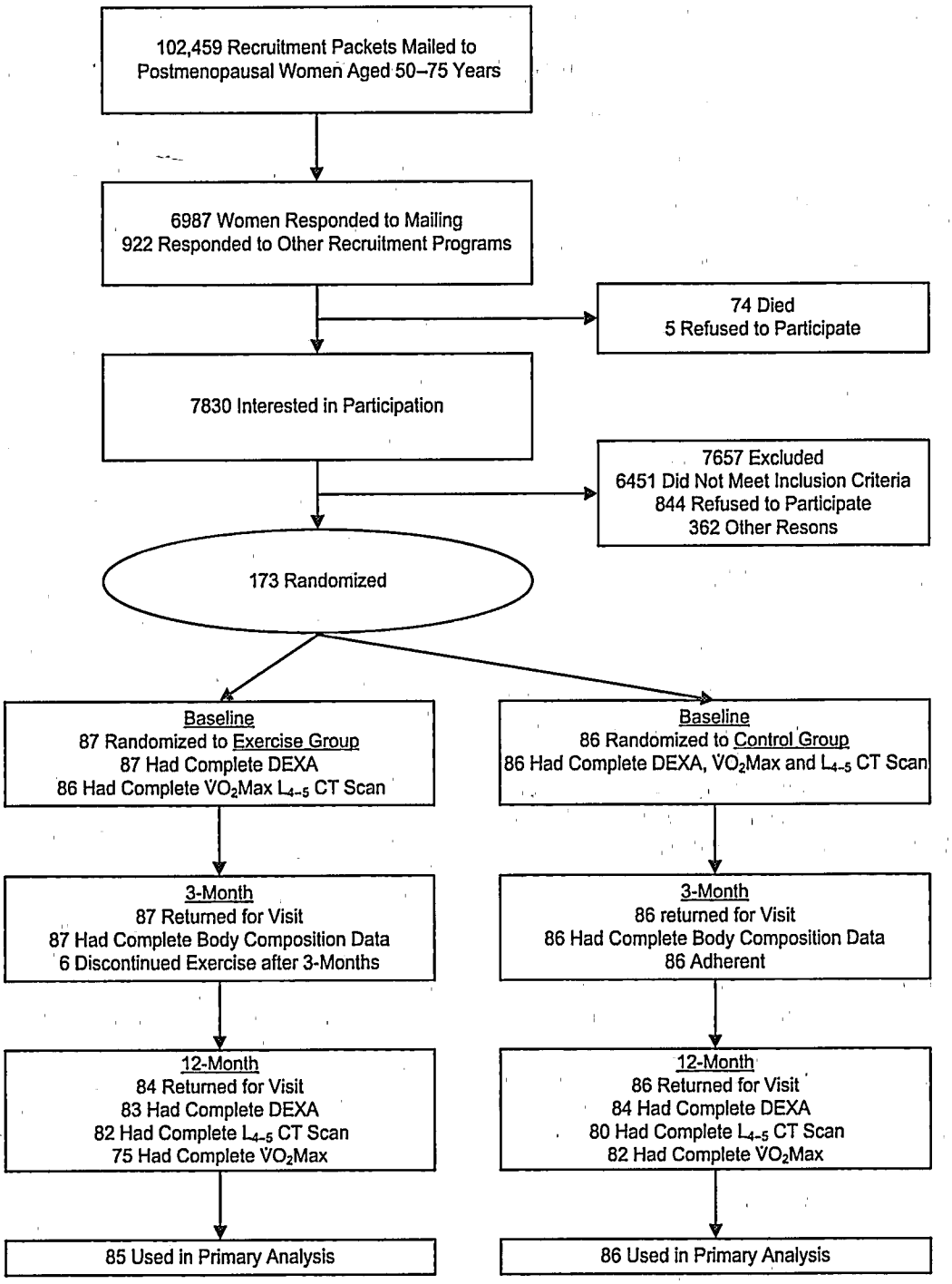


**FIGURE 1—Recruitment and follow-up of participants.**

scanner, Waukesha, WI) (18) were measured. $\dot{V}O_{2max}$ was assessed at baseline and at 12 months using a Medgraphics automated metabolic cart (Medgraphics, St. Paul, MN), where participants completed a maximal-graded treadmill test. The test was started at 3.0 mph and 0% slope grade. Speed was raised to 3.5 mph at 2 min, and thereafter slope grade was increased by 2% every 2 min until the participant reached volitional fatigue. No treadmill tests were terminated for reasons other than volitional fatigue. A 12-h fasting blood sample was collected, processed, and stored at −70°C. Participants were asked not to exercise for 24 h before having blood drawn.

## Lipoprotein Assays

Total cholesterol, HDL, and triglycerides were measured enzymatically using the Hitachi 917 autoanalyzer at the Northwest Lipid Research Laboratories, University of Washington, Seattle, directed by Dr. Santica Marcovina. The HDL assay was done using methods standardized to the Center for Disease Control and Prevention reference methods (38), and the assay for triglycerides used the Boehringer Mannheim reagent. The intra- and interassay CV were 1.0 and 1.9% for TC, 0.9 and 2.0% for triglycerides, and 1.4 and 2.1% for HDL. We used the Friedewald's equation method (14) to calculate LDL.

TABLE 1. Baseline characteristics of exercisers and stretchers: physical activity for total health (PATH) study.

| Variable | Exercisers ($N$ = 87) Mean (SD) or % | Stretchers ($N$ = 86) Mean (SD) or % |
|---|---|---|
| Age (yr) | 60.6 (6.6) | 60.5 (6.7) |
| Weight (kg) | 81.6 (14) | 81.7 (12) |
| Race: white | 85% | 87% |
| Systolic blood pressure (BP) (mm Hg) | 124.6 (15.5) | 127.1 (14.5) |
| Diastolic BP (mm Hg) | 79.2 (9.1) | 79.9 (9.6) |
| Self-reported hypertension | 24% | 23% |
| Ever smoked >100 cigarettes | 47% | 46% |
| Heart disease in any blood relatives | 69.8% | 82.0% |
| Alcohol intake (>0.35 $g \cdot d^{-1}$) | 66% | 59.3% |
| Lipid-lowering medication use* | 10% | 3% |
| Beta-blocker use | 4% | 7% |
| Thyroid supplement | 17% | 17% |
| **Baseline serum lipoprotein ($mg \cdot dL^{-1}$)** | | |
| Total cholesterol | 230.7 (37.8) | 232.4 (41.4) |
| HDL cholesterol | 51.9 (11.5) | 52.6 (13.3) |
| LDL cholesterol | 152.3 (36.3) | 152.5 (41.8) |
| Triglycerides | 133.6 (58.5) | 136.4 (71.8) |
| **Variables for stratification** | | |
| $\dot{V}O_{2max}$ ($mL \cdot min^{-1} \cdot kg^{-1}$) | 20.1 (3.5) | 20.4 (2.9) |
| Percent body fat | 47.6 (4.8) | 47.4 (4.6) |
| Intraabdominal fat ($cm^2$) | 147.6 (60) | 147.6 (56) |
| Subcutaneous abdominal fat ($cm^2$) | 389.1 (124) | 371.1 (107) |

* $P < 0.05$ for lipid-lowering medication use at baseline.

For 87.2% of subjects, blood samples from the three time points (baseline, and 3 and 12 months after randomization) were assayed in the same batch. This was done to make the chemical assay more sensitive to changes in lipoprotein fractions by reducing the interbatch variability. For the remaining samples, batching was broken at least at one time point.

## Statistical Analysis

Under the principle of intent-to-treat, we studied the primary effect of the exercise intervention on serum lipoproteins using generalized estimating equations (GEE) (22), which account for within-subject correlation of lipoproteins over time (Stata version 8.0). We used baseline values as the reference to compare changes in lipoproteins between exercisers and stretchers at 3 and 12 months. After an unadjusted primary analysis, we adjusted for dietary cholesterol or saturated fat intake, use of lipid-lowering medications and seasonal variation (28). We defined seasons by starting dates as autumn (Sept 23), winter (Dec 22), spring (Mar 21), and summer (June 22). Exclusion of subjects using lipid-lowering medications from the analysis did not change our results. Because batching was broken for 22 subjects during lipoprotein assays, we conducted a subanalysis, excluding the subjects if their baseline blood sample was assayed separately. We also excluded the specific time-points when 3- or 12-month blood samples were assayed separately. Another subanalysis excluded three subjects with triglyceride levels >400 $mg \cdot dL^{-1}$ because Friedewald's calculation method has been reported (25) to be inaccurate for LDL calculation over triglyceride levels of 400 $mg \cdot dL^{-1}$. These exclusions did not change our results and, therefore, we present the results including all subjects.

Within the limits of statistical power afforded by the study, as a secondary analysis we studied if the effect of exercise on lipoproteins was particularly evident in subgroups of exercisers defined by quartiles of baseline or 1-yr change in total body fat, intraabdominal fat, subcutaneous fat, level of aerobic fitness, or by self-reported adherence. We introduced these variables as effect modifiers in the GEE models, keeping the randomized design intact, except for exercise adherence. Subgroup analysis for adherence was conducted for exercisers only (exercising <135, 136 to 195, and >95 $min \cdot wk^{-1}$ of exercise), with stretchers as the reference group.

We defined quartiles of change in CT-measured intra-abdominal fat (gain >12 $cm^2$, gain <12 $cm^2$, loss <18 $cm^2$, and loss >18 $cm^2$) and subcutaneous abdominal fat (gain >30 $cm^2$, gain <30 $cm^2$, loss <45 $cm^2$, and loss >45 $cm^2$). DEXA-measured percent total body fat was stratified as an increase >0.5%, an increase or a decrease <0.5%, a decrease ≤2.0%, or a decrease >2.0%. Change in $\dot{V}O_{2max}$

TABLE 2. Serum lipoprotein changes among exercisers and stretchers.

| | Baseline | | 3 Month | | | 12 Month | | |
|---|---|---|---|---|---|---|---|---|
| Lipoprotein ($mg \cdot dL^{-1}$) Group | Mean | 95% CI | Mean | 95% CI | $P$* | Mean | 95% CI | $P$* |
| Total cholesterol | | | | | | | | |
| Exerciser† | 230.7 | 222–239 | 221.4 | 213–229 | 0.053 | 225.2 | 216–233 | 0.83 |
| Stretcher† | 232.4 | 223–241 | 227.0 | 218–235 | | 225.1 | 216–233 | |
| Triglycerides | | | | | | | | |
| Exerciser | 133.6 | 121–146 | 133.9 | 121–146 | 0.26 | 129.6 | 117–142 | 0.68 |
| Stretcher | 136.4 | 121–151 | 142.8 | 124–161 | | 132.9 | 117–148 | |
| LDL cholesterol | | | | | | | | |
| Exerciser | 152.3 | 144–160 | 146.4 | 139–153 | 0.43 | 146.6 | 139–154 | 0.43 |
| Stretcher | 152.5 | 143–161 | 142.0 | 135–149 | | 147.1 | 138–155 | |
| HDL cholesterol | | | | | | | | |
| Exerciser | 51.9 | 49–54 | 49.1 | 46–51 | 0.08 | 52.2 | 49–55 | 0.28 |
| Stretcher | 52.6 | 49–55 | 51.8 | 48–54 | | 51.4 | 48–54 | |
| Total HDL‡ | | | | | | | | |
| Exerciser | 4.48 | 4.2–4.7 | 4.57 | 4.3–4.8 | 0.74 | 4.34 | 4.0–4.6 | 0.16 |
| Stretcher | 4.48 | 4.1–4.8 | 4.39 | 4.0–4.6 | | 4.43 | 4.1–4.6 | |
| LDL HDL‡ | | | | | | | | |
| Exerciser | 2.88 | 2.6–3.1 | 2.97 | 2.7–3.1 | 0.40 | 2.80 | 2.5–3.0 | 0.23 |
| Stretcher | 2.88 | 2.6–3.1 | 2.74 | 2.5–3.0 | | 2.85 | 2.6–3.1 | |

* $P$ values test whether exercisers change differently compared with stretchers from baseline to a given time-point.
† Number of exercisers and stretchers were 85 and 86. Maximal number of missing values for any lipoprotein fraction is 5 and 6, respectively.
‡ Geometric means of total:HDL and LDL:HDL ratio used for analysis.

TABLE 3. Serum lipoprotein changes among exercisers and stretchers across strata of change in intraabdominal fat (computed tomography at $L_{4-5}$ level).

| Lipoprotein ($mg \cdot dL^{-1}$) (Change in Intraabdominal Fat) | Exercisers N† Baseline Mean (95% CI) | Exercisers N† 3 Month Mean (95% CI) | Exercisers N† 12 Month Mean (95% CI) | Stretchers N† Baseline Mean (95% CI) | Stretchers N† 3 Month Mean (95% CI) | Stretchers N† 12 Month Mean (95% CI) |
|---|---|---|---|---|---|---|
| **Total cholesterol** | | | | | | |
| Gain >12 $cm^2$ | 227 (208–246) | 226 (206–246) | 222 (203–241) | 225 (201–249) | 229 (207–251) | 224 (204–244) |
| Gain ≤12 $cm^2$ | 224 (198–251) | 207 (182–233) | 220 (194–246) | 232 (220–244) | 235 (218–252) | 224 (210–238) |
| Loss ≤18 $cm^2$ | 234 (218–251) | 225 (210–240) | 229 (213–245) | 242 (219–264) | 228 (207–250) | 239 (220–259) |
| Loss >18 $cm^2$ | 225 (210–239) | 213 (200–227) | 220 (204–236) | 229 (217–241) | 212 (201–223) | 210 (198–222) |
| **Triglycerides** | | | | | | |
| Gain >12 $cm^2$ | 139 (115–163) | 142 (116–168) | 142 (111–173) | 122 (96–147) | 137 (108–165) | 135 (105–165) |
| Gain ≤12 $cm^2$ | 141 (99–184) | 134 (99–168) | 123 (83–163) | 139 (106–171) | 131 (89–174) | 119 (86–151) |
| Loss ≤18 $cm^2$ | 128 (105–152) | 134 (110–158) | 122 (102–142) | 136 (111–162) | 135 (110–160) | 126 (108–145) |
| Loss >18 $cm^2$ | 122 (98–146) | 124 (100–148) | 126 (102–151) | 159 (108–210) | 174 (112–236) | 150 (102–197) |
| **LDL cholesterol** | | | | | | |
| Gain >12 $cm^2$ | 147 (127–168) | 149 (128–170) | 144 (124–163) | 147 (121–172) | 134 (122–146) | 144 (124–165) |
| Gain ≤12 $cm^2$ | 151 (124–178) | 136* (110–162) | 141 (114–169) | 149 (136–161) | 152 (135–169) | 142 (129–155) |
| Loss ≤18 $cm^2$ | 154 (139–169) | 148 (135–161) | 149 (135–162) | 164 (144–183) | 147 (129–164) | 164 (145–183) |
| Loss >18 $cm^2$ | 147 (133–161) | 140 (129–150) | 144 (131–156) | 149 (131–167) | 134 (119–149) | 134 (118–149) |
| **HDL cholesterol** | | | | | | |
| Gain >12 $cm^2$ | 50.5 (45–55) | 48.1 (42–53) | 50.1 (44–55) | 51.0 (45–56) | 52.3 (46–57) | 49.1 (43–54) |
| Gain ≤12 $cm^2$ | 47.1 (39–54) | 44.4 (38–50) | 48.8 (42–55) | 55.8 (49–62) | 56.6 (48–64) | 57.7 (50–64) |
| Loss ≤18 $cm^2$ | 53.9 (49–58) | 51.2 (46–55) | 52.8 (48–57) | 51.5 (45–57) | 49.4 (43–55) | 50.0 (45–54) |
| Loss >18 $cm^2$ | 54.7 (47–61) | 50.8 (42–59) | 55.5 (46–64) | 49.4 (42–56) | 46.6 (39–53) | 47.8 (41–54) |

* $P < 0.05$; $P$ value tests if exercisers change differently from stretchers from baseline across strata of change in intraabdominal fat.
† Number of subjects gaining >12 $cm^2$; gaining <12 $cm^2$; losing <18 $cm^2$; and losing >18 $cm^2$ were 17, 12, 29, and 22 among exercisers and 25, 20, 20, and 18 among stretchers. Maximal number of missing values for any lipoprotein fraction is 11.

was stratified as a decrease in $\dot{V}O_{2max}$, an increase of 1% to 10%, and an increase of >10%.

Lastly, with all the participants pooled, independent of intervention and thereby breaking randomization, we studied if serum lipoprotein changes were associated with changes in subcutaneous or intraabdominal fat, or with changes in percent total body fat.

## RESULTS

Of the 173 women, 170 (98.3%) women completed the study with exercisers averaging 176 (SD 91) $min \cdot wk^{-1}$ of exercise at 60–75% HRmax, expending approximately 3828 kJ (SD 2053) per week. The most common sports or recreational activities reported at both the exercise facility and at home were walking and bicycling. Three exercisers dropped out after 3 months. Self-reported adherence was available for all the exercisers. No significant difference was seen in physical activity beside the intervention between the exercisers and controls as assessed by the physical activity questionnaire. Completeness of data for $\dot{V}O_{2max}$, DEXA, and CT is shown in Figure 1, and the baseline characteristics of subjects are presented in Table 1.

Blood was available for 165, 159, and 162 subjects at baseline, and at 3 and 12 months after randomization, respectively. Baseline distributions of all the lipoprotein fractions were approximately normally distributed except for triglycerides, which, as expected, was slightly right-skewed. Mean (SD) baseline lipoprotein levels for TC, LDL, HDL, and triglycerides were 231 (39), 152 (39), 52 (12), and 135 (65) $mg \cdot dL^{-1}$, respectively.

Over 12 months, exercisers significantly increased their $\dot{V}O_{2max}$ (+11%; 95% CI, 6.9% to 15.1%, $P < 0.001$), compared with stretchers. At the end of 12 months, exercisers lost more body weight (−1.4 kg; 95% CI, −2.5 to −0.3 kg, $P < 0.05$), % total body fat (−1.0%; 95% CI, −1.6% to −0.4%, $P < 0.005$), intraabdominal fat (−8.6 $cm^2$; 95% CI −17.8 to 0.9 $cm^2$, $P < 0.05$) and subcutaneous abdominal fat (−28.8 $cm^2$; 95% CI, −47.5 to −10.0 $cm^2$, $P < 0.005$) compared with stretchers (17). At 1 yr, exercisers compared with the stretchers did not significantly change their dietary intake of total calories (1645 to 1608 vs 1724 to 1608 $kcal \cdot d^{-1}$, $P = 0.34$), cholesterol (247 to 252 vs 253 to 252 $mg \cdot d^{-1}$, $P = 0.83$), saturated fatty acid (24.3 to 24.4 vs 25.8 to 24.1 $g \cdot d^{-1}$, $P = 0.37$), or percent calories from saturated fatty acids (12.5 to 13.0% vs 13.3 to 13.2%, log-transformed when analyzed, $P = 0.67$).

In our primary analysis, we did not observe a statistically significant change in serum lipoprotein levels among exercisers compared with the stretchers at either 3 or 12 months (Table 2). The small increase in HDL among exercisers compared with stretchers was not statistically significant ($P = 0.28$).

More exercisers (9, 7, and 9) compared with stretchers (2, 2, and 5) reported using lipid-lowering medications at baseline, and at 3 and 12 months, respectively, and these were significantly associated ($P < 0.01$) with a lower LDL and TC. Excluding subjects using these medications from the analysis did not change the results. Alcohol intake, however, was significantly associated ($P < 0.05$) with a higher HDL. Adjustments for lipid-lowering medications, alcohol intake, cholesterol, and saturated fat intake and seasons did not change the results of the primary analysis appreciably.

Secondary analysis was conducted to determine if changes in serum lipoproteins by exercise intervention were evident in subgroups that maximally changed their subcutaneous and intraabdominal fat (Table 3), percent total body fat (Table 4), $\dot{V}O_{2max}$ (Table 5), or in subgroups of highest self-reported minutes per week of exercise over 1 yr (data not shown). We did not observe a statistically significant effect of exercise on serum lipoproteins in the above subgroups.

Breaking randomization and pooling participants together, intraabdominal fat changes were not consistently related to lipoprotein levels changes. We, however, observed a significant association of greater loss of DEXA-assessed percent total body fat with increase in HDL ($P < 0.01$) and decrease in triglyceride ($P < 0.01$) levels (Table 6).

## DISCUSSION

Our trial suggests that, among sedentary and overweight/obese postmenopausal women, participation in a 1-yr moderate-intensity aerobic exercise regimen alone does not significantly change serum lipoprotein levels. We did not observe a significant improvement in serum lipoprotein fractions at 3 or 12 months in exercisers compared with stretchers in subgroups that decreased their intraabdominal fat, subcutaneous fat, or percent total body fat the most, or increased their aerobic fitness ($\dot{V}O_{2max}$) the most. We, however, did find a significant association of decrease in percent total body fat with an increase in HDL and a decrease in triglycerides.

The main strengths of study were the single-blinded, randomized design, large sample size, and 1-yr duration of

TABLE 4. Serum lipoprotein changes among exercisers and stretchers across strata of change in percent total body fat (DEXA).

| | Exercisers N* | | | Stretchers N* | | |
|---|---|---|---|---|---|---|
| | Baseline | 3 Month | 12 Month | Baseline | 3 Month | 12 Month |
| Lipoprotein (mg·dL$^{-1}$) (Change in Percent Total Body Fat) | Mean (95% CI) | Mean (95% CI) | Mean (95% CI) | Mean (95% CI) | Mean (95% CI) | Mean (95% CI) |
| **Total cholesterol** | | | | | | |
| Gain >0.5% | 240 (218–261) | 239 (222–257) | 237 (219–256) | 225 (204–246) | 223 (205–242) | 220 (203–236) |
| No change | 232 (211–253) | 217 (189–245) | 228 (204–252) | 229 (213–246) | 224 (206–242) | 223 (208–239) |
| Lost 0.5–2.0% | 234 (220–248) | 229 (215–243) | 228 (212–243) | 240 (224–256) | 237 (222–253) | 236 (221–251) |
| Lost >2.0% | 217 (200–234) | 201 (188–214) | 212 (196–227) | 236 (214–257) | 216 (185–248) | 213 (182–244) |
| **Triglycerides** | | | | | | |
| Gain >0.5% | 128 (92–164) | 135 (103–167) | 135 (100–170) | 133 (103–164) | 157 (118–195) | 144 (110–179) |
| No change | 131 (98–164) | 118 (75–161) | 125 (90–160) | 145 (102–188) | 153 (102–204) | 141 (108–175) |
| Lost 0.5–2.0% | 138 (118–158) | 148 (130–166) | 139 (118–161) | 126 (104–149) | 121 (96–146) | 116 (92–139) |
| Lost >2.0% | 131 (103–159) | 126 (99–152) | 115 (92–138) | 154 (122–187) | 137 (108–166) | 114 (86–142) |
| **LDL cholesterol** | | | | | | |
| Gain >0.5% | 163 (144–182) | 163 (147–178) | 165 (151–179) | 150 (127–172) | 133 (122–144) | 144 (126–162) |
| No change | 149 (130–168) | 141 (117–166) | 146 (125–168) | 147 (131–163) | 136 (122–150) | 144 (128–160) |
| Lost 0.5–2.0% | 156 (142–170) | 153 (140–166) | 144 (130–159) | 159 (144–174) | 157 (141–173) | 157 (142–172) |
| Lost >2.0% | 138 (120–155) | 126 (114–138) | 135 (122–149) | 155 (135–175) | 143 (118–168) | 133 (104–163) |
| **HDL cholesterol** | | | | | | |
| Gain >0.5% | 49.4 (43–55) | 46.6 (41–51) | 48.4 (43–53) | 49.1 (43–54) | 47.8 (42–52) | 46.6 (41–51) |
| No change | 56.5 (45–67) | 55.2 (39–71) | 56.5 (44–68) | 53.6 (48–58) | 52.6 (45–59) | 51.4 (46–56) |
| Lost 0.5–2.0% | 50.4 (46–54) | 47.9 (43–52) | 50.2 (45–54) | 54.5 (47–61) | 55.0 (49–60) | 54.5 (48–60) |
| Lost >2.0% | 54.3 (49–59) | 49.8 (45–54) | 54.7 (49–59) | 55.2 (44–65) | 54.0 (41–66) | 60.5 (48–72) |

* Number of subjects gaining >0.5%, remaining same, losing 0.5–2.0%, and losing >2% of body fat were 16, 11, 29, and 25 among exercisers and 27, 21, 25, and 11, among stretchers. Maximal number of missing values for any lipoprotein fraction is 11. None of the values differed significantly in exercisers compared with stretchers in any of the strata.

TABLE 5. Serum lipoprotein changes among exercisers and stretchers across strata of percent change in aerobic fitness ($\dot{V}O_{2max}$).

| | Exercisers N† | | | Stretchers N† | | |
|---|---|---|---|---|---|---|
| | Baseline | 3 Month | 12 Month | Baseline | 3 Month | 12 Month |
| Lipoprotein (mg·dL$^{-1}$) (Change in Aerobic Fitness) | Mean (95% CI) | Mean (95% CI) | Mean (95% CI) | Mean (95% CI) | Mean (95% CI) | Mean (95% CI) |
| **Total cholesterol** | | | | | | |
| Same or decreased | 238 (216–261) | 235 (220–251) | 234 (213–256) | 231 (218–245) | 229 (216–242) | 227 (215–239) |
| Gain of 1–10% | 236 (219–254) | 232 (209–256) | 221 (199–243) | 227 (212–242) | 220 (208–233) | 218 (205–231) |
| Gain of >10% | 219 (215–237) | 207 (204–224) | 222 (213–234) | 239 (219–262) | 221 (203–251) | 216* (201–252) |
| **Triglycerides** | | | | | | |
| Same or decreased | 147 (111–183) | 148 (113–183) | 160 (113–207) | 144 (119–168) | 150 (121–179) | 141 (117–165) |
| Gain of 1–10% | 123 (99–146) | 135 (106–164) | 122 (100–144) | 138 (109–167) | 139 (103–176) | 123 (92–154) |
| Gain of >10% | 126 (116–150) | 124 (114–145) | 122 (109–138) | 106 (90–134) | 121 (109–143) | 111* (102–140) |
| **LDL cholesterol** | | | | | | |
| Same or decreased | 164 (143–186) | 159 (144–175) | 159 (141–177) | 152 (137–166) | 139 (130–148) | 149 (136–161) |
| Gain of 1–10% | 155 (136–175) | 153 (130–176) | 138 (118–158) | 145 (130–159) | 139 (128–151) | 139 (126–153) |
| Gain of >10% | 141 (137–158) | 135 (132–149) | 145 (136–155) | 162 (143–182) | 148 (129–173) | 142 (127–175) |
| **HDL cholesterol** | | | | | | |
| Same or decreased | 47.6 (41–53) | 47.0 (40–53) | 48.6 (40–56) | 49.7 (45–53) | 49.6 (45–54) | 48.7 (44–52) |
| Gain of 1–10% | 54.9 (48–61) | 50.1 (42–57) | 53.7 (46–61) | 56.8 (50–62) | 56.1 (49–62) | 55.8 (50–61) |
| Gain of >10% | 52.5 (48–55) | 48.6 (45–52) | 52.0 (49–56) | 55.9 (48–63) | 51.3 (45–59) | 53.2 (45–61) |

* $P < 0.01$; $P$ value tests if exercisers change differently from stretchers from baseline across strata of percent change in aerobic capacity ($\dot{V}O_{2max}$).
† Number of subjects gaining <1% or decreasing, gaining 1–10%, and gaining >10% of $\dot{V}O_{2max}$ were 14, 18, and 41 among exercisers and 46, 23, and 13 among stretchers. Maximal number of missing values for any lipoprotein fraction is 11.

the intervention, combined with excellent adherence to the exercise regimen and near complete follow-up of study subjects. One limitation of the study was that, for some participants, not all samples from the same woman were assayed at the same time for triglycerides. We expect that this would lead to a nondifferential measurement error that may attenuate the risk estimates (36). Results excluding these women were similar, however, to those including them. Some studies (35) have suggested that acute exercise effects on blood lipids can extend beyond 48 h, and that maximal acute effects occur at 24 h. Because we asked subjects not to exercise for 24 h before their blood was drawn, we are unable to rule out the possibility that training did change serum lipoproteins independent of its acute effects. In addition, with the relatively small number of subjects in various subgroups, our study was presumably underpowered for the subgroup analysis.

Our trial has studied the effects of exercise exclusively among overweight/obese postmenopausal women. Participants in our trial also had borderline high levels of TC and LDL cholesterol at baseline, according to the NCEP ATP III classification. The mean cholesterol level of women in our study was similar to U.S. national averages for women in similar age group from NHANES III (1999–2000 data)

TABLE 6. Trends in serum lipids changes stratified by changes in DEXA-measured percent total body fat and by changes in CT-measured intraabdominal fat, regardless of exerciser or stretcher status.

| | | Change in Total Cholesterol* | | Change in LDL* | | Change in HDL* | | Change in Triglycerides* | |
|---|---|---|---|---|---|---|---|---|---|
| Change in Body Fat Measure | N† | Mean | 95% CI | Mean | 95% CI | Mean | 95% CI | Mean | 95% CI |
| **Intraabdominal fat** | | | | | | | | | |
| Gain >12 cm$^2$ | 42 | −2.2 | −12, 8 | −2.8 | −11, 6 | −1.2 | −3.5, 0.9 | 8.9 | −5, 23 |
| Gain <12 cm$^2$ | 32 | −7.0 | −13, −1 | −5.9 | −11, −1 | 2.0 | −0.4, 4.4 | −19.2 | −34, −4 |
| Loss <18 cm$^2$ | 48 | −4.0 | −15, 7 | −1.7 | −12, 9 | −1.1 | −2.7, 0.4 | −6.3 | −18, 6 |
| Loss >18 cm$^2$ | 40 | −10.8 | −19, −2 | −10.0 | −18, −2 | 0.6 | −1.5, 2.7 | −1.8 | −14, 11 |
| | | Trend | $P = 0.21$ | Trend | $P = 0.46$ | Trend | $P = 0.73$ | Trend | $P = 0.80$ |
| **Percent total body fat** | | | | | | | | | |
| Gain >0.5 | 43 | −4.5 | −11, 2 | −4.4 | −10, 2 | −1.9 | −3.5, 0.3 | 9.6 | −2, 22 |
| Gain or loss of 0.5 | 32 | −5.3 | −14, 3 | −3.1 | −11, 5 | −1.4 | −3.7, 0.8 | −4.2 | −19, 10 |
| Lost 0.5–2.0 | 54 | −5.5 | −16, 5 | −4.8 | −14, 4 | 0.22 | −1.4, 1.8 | −4.3 | −16, 8 |
| Loss >2.0 | 35 | −9.9 | −21, 2 | −8.2 | −20, 4 | 2.8 | 0, 5.6 | −21.1 | −35, −6 |
| | | Trend | $P = 0.20$ | Trend | $P = 0.25$ | Trend | $P < 0.01$ | Trend | $P < 0.01$ |

* Lipoprotein changes in milligrams per deciliter.
† For any lipoprotein fraction, the maximum number of missing blood samples is 10. $P$ values indicate overall trend in lipoprotein fraction changes in strata of changes in intraabdominal fat and percent total body fat.

(12). Notably, women in trials discussed earlier (2,3,6,10,19,32,33) have been leaner and had a relatively favorable lipid profile compared with those in our study at baseline. Thus, it is possible that the effects of exercise on serum lipoproteins might be different among overweight/obese women when compared with leaner women or among women with adverse lipid profiles compared with better lipid profiles. Donnelly et al. (7), studying the effects of intermittent versus continuous exercise for 18 months among moderately obese women, reported an increase in HDL cholesterol compared with baseline among both the groups. The trial lacked a control group. Women in our study expended about 3828 kJ (915 kcal) from the exercise intervention, which is lower than the threshold of 1200–1600 kcal·wk$^{-1}$ suggested by Kokkinos and Fernhall (20). We, however, did not observe greater changes in lipoproteins among women who adhered most to the exercise regimen. A randomized trial by King et al. (19) suggested that a more frequent exercise regimen and a longer duration of 2 yr, although home based and low intensity, might be required to increase HDL. Miller (24) reported reductions in total cholesterol (−25 mg·dL$^{-1}$, $P < 0.001$), LDL (−18 mg·dL$^{-1}$, $P = 0.005$), and HDL (−5 mg·dL$^{-1}$, $P < 0.001$) after a 9-wk combined diet, exercise, and weight loss intervention in overweight women. Therefore, greater weekly energy expenditure from exercise, longer duration of exercise regimen, and a combination of diet with exercise need to be studied in randomized exercise trials. We, however, were not able to address in our study whether longer-duration, higher-intensity exercise, combined with diet, or the same exercise regimen among leaner women, would significantly change serum lipoproteins.

Nicklas et al. (26) reported an association of higher HDL, a lower LDL:HDL ratio and triglyceride level with an intraabdominal fat level below a threshold of 105 cm$^2$, compared with higher levels. Whereas, Brochu et al. (5) reported similar improvements in HDL and cholesterol: HDL ratio among women who decreased their intra-abdominal fat below threshold of 110 cm$^2$ compared with those who did not, after a 1-yr, nonrandomized prospective weight loss program. In our study, although exercisers lost significantly greater amounts of total body fat, intra-abdominal, and subcutaneous abdominal fat compared with stretchers, we did not observe greater changes in serum lipoproteins among exercisers compared to stretchers even in the subgroups that lost maximal fat. All the women in this study had high amounts of baseline visceral fat (mean 147.6 cm$^2$, SD 58.1 cm$^2$), and it is possible that they did not reduce their visceral fat sufficiently to substantively affect their lipid profile, if a specific threshold must be attained to alter lipid profiles.

Our intervention was sufficiently intense to cause biological effects, as attested by significant changes in insulin and leptin (13) in exercisers compared with controls. We feel, therefore, that our conclusion, that moderate-intensity aerobic exercise alone is insufficient to cause significant changes in lipid profile is justified.

Although our data did not demonstrate significant evidence for a beneficial effect of exercise on serum lipoproteins in the intent-to-treat analysis, in the pooled analysis of subjects from both treatment groups, we observed a significant association of greater loss of percent total body fat with increased HDL and decreased triglyceride levels. These findings could reflect the possibility that total body fat is an intermediate in the pathway between exercise and serum lipoproteins or could also result from confounding.

In a cross-sectional study with older men ($N = 53$) and women ($N = 63$), Dvorak et al. (8) reported that individuals with greater cardiorespiratory fitness ($\dot{V}O_{2max}$), regardless of physical activity, had lower triglycerides, TC, DL, and total:HDL ratio compared with those with lower cardiorespiratory fitness. We analyzed whether serum lipoprotein changes were evident in subgroups defined by the greatest increase from baseline (>10%) in aerobic fitness ($\dot{V}O_{2max}$), but did not observe consistent trends.

In conclusion, it appears that participation in a 1-yr moderate-intensity aerobic exercise regimen alone does not significantly change serum lipoprotein levels among sedentary and overweight/obese postmenopausal women. Future research should focus on randomized exercise trials studying effects of longer durations of exercise at varying intensities and frequencies in combination with diet among postmenopausal women with varying BMI and other cardiovascular risk profiles (diabetes, smoking, hyperlipidemia). These should assess changes in serum lipoproteins and their subfractions, and make accurate measures of intraabdominal fat and $\dot{V}O_{2max}$ to assess whether changes in these measures are associated with changes in lipoprotein fractions.

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## REFERENCES

1. Ainsworth, B. E., W. L. Haskell, M. C. Whitt, et al. Compendium of physical activities: an update of activity codes and MET intensities. *Med. Sci. Sports Exerc.* 32:S498–S504, 2000.
2. Asikainen, T. M., S. Miilunpalo, K. Kukkonen-Harjula, et al. Walking trials in postmenopausal women: effect of low doses of exercise and exercise fractionization on coronary risk factors. *Scand. J. Med. Sci. Sports.* 13:284–292, 2003.
3. Binder, E. F., S. J. Birge, and W. M. Kohrt. Effects of endurance exercise and hormone replacement therapy on serum lipids in older women. *J. Am. Geriatr. Soc.* 44:231–236, 1996.

4. Brochu, M., A. Tchernof, I. J. Dionne, et al. What are the physical characteristics associated with a normal metabolic profile despite a high level of obesity in postmenopausal women? *J. Clin. Endocrinol. Metab.* 86:1020–1025, 2001.
5. Brochu, M., A. Tchernof, A. N. Turner, P. A. Ades, and E. T. Poehlman. Is there a threshold of visceral fat loss that improves the metabolic profile in obese postmenopausal women? *Metabolism* 52:599–604, 2003.
6. Cauley, J. A., A. M. Kriska, R. E. LaPorte, R. B. Sandler, and G. Pambianco. A two year randomized exercise trial in older women: effects on HDL-cholesterol. *Atherosclerosis* 66:247–258, 1987.
7. Donnelly, J. E., D. J. Jacobsen, K. S. Heelan, R. Seip, and S. Smith. The effects of 18 months of intermittent vs. continuous exercise on aerobic capacity, body weight and composition, and metabolic fitness in previously sedentary, moderately obese females. *Int. J. Obes. Relat. Metab. Disord.* 24:566–572, 2000.
8. Dvorak, R. V., A. Tchernof, R. D. Starling, P. A. Ades, L. DiPietro, and E. T. Poehlman. Respiratory fitness, free living physical activity, and cardiovascular disease risk in older individuals: a doubly labeled water study. *J. Clin. Endocrinol Metab.* 85:957–963, 2000.
9. Eaton, C. B., K. L. Lapane, C. A. Garber, A. R. Assaf, T. M. Lasater, and R. A. Carleton. Sedentary lifestyle and risk of coronary heart disease in women. *Med. Sci. Sports Exerc.* 27: 1535–1539, 1995.
10. Fahlman, M. M., D. Boardley, C. P. Lambert, and M. G. Flynn. Effects of endurance training and resistance training on plasma lipoprotein profiles in elderly women. *J. Gerontol. A. Biol. Sci. Med. Sci.* 57:B54–B60, 2002.
11. Folsom, A. R., D. K. Arnett, R. G. Hutchinson, F. Liao, L. X. Clegg, and L. S. Cooper. Physical activity and incidence of coronary heart disease in middle-aged women and men. *Med. Sci. Sports Exerc.* 29:901–909, 1997.
12. Ford, E. S., A. H. Mokdad, W. H. Giles, and G. A. Mensah. Serum total cholesterol concentrations and awareness, treatment, and control of hypercholesterolemia among US adults: findings from the National Health and Nutrition Examination Survey, 1999 to 2000. *Circulation* 107:2185–2189,2003.
13. Frank, L. L., K. B. Rajan, Y. Yasui, S. S. Tworoger, C. M. Ulrich, and A. McTiernan. Effects of physical activity on metabolic risk variables in overweight postmenopausal women. A randomized clinical trial. *Obes. Res.* 13:615–625, 2005.
14. Friedewald, W. T., R. I. Levy, and D. S. Fredrickson. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. *Clin. Chem.* 18:499–502, 1972.
15. Guidelines for Physical Activity Testing and Prescription, 4th ed. *American College of Sports Medicine.* Philadelphia: Lea and Febiger, 1991, pp. 3–268.
16. Hernandez-Ono, A., G. Monter-Carreola, J. Zamora-Gonzalez, et al. Association of visceral fat with coronary risk factors in a population-based sample of postmenopausal women. *Int. J. Obes. Relat. Metab. Disord.* 26:33–39, 2002.
17. Irwin, M. L., Y. Yasui, C. M. Ulrich, et al. Effect of exercise on total and intra-abdominal body fat in postmenopausal women: a randomized controlled trial. *JAMA* 289:323–330, 2003.
18. Jensen, M. D., J. A. Kanaley, J. E. Reed, and P. F. Sheedy. Measurement of abdominal and visceral fat with computed tomography and dual-energy x-ray absorptiometry. *Am. J. Clin. Nutr.* 61:274–278, 1995.
19. King, A. C., W. L. Haskell, D. R. Young, R. K. Oka, and M. L. Stefanick. Long-term effects of varying intensities and formats of physical activity on participation rates, fitness, and lipoproteins in men and women aged 50 to 65 years. *Circulation* 91:2596–2604, 1995.
20. Kokkinos, P. F., and B. Fernhall. Physical activity and high density lipoprotein cholesterol levels: what is the relationship? *Sports Med.* 28:307–314, 1999.
21. LaCroix, A. Z., S. G. Leveille, J. A. Hecht, L. C. Grothaus, and E. H. Wagner. Does walking decrease the risk of cardiovascular disease hospitalizations and death in older adults? *J. Am. Geriatr. Soc.* 44:113–120, 1996.
22. Liang, K. Y., and S. L. Zeger. Longitudinal data analysis using generalized linear models. *Biometrika* 73:13–22, 1986.
23. McTiernan, A., C. M. Ulrich, D. Yancey, et al. The Physical Activity for Total Health (PATH) Study: rationale and design. *Med. Sci. Sports Exerc.* 31:1307–1312, 1999.
24. Miller 3rd, E. R., T. P. Erlinger, D. R. Young, et al. Results of the Diet, Exercise, and Weight Loss Intervention Trial (DEW-IT). *Hypertension* 40:612–8, 2002.
25. Nauck, M., G. R. Warnick, and N. Rifai. Methods for measurement of LDL-cholesterol: a critical assessment of direct measurement by homogeneous assays versus calculation. *Clin. Chem.* 48:236–254, 2002.
26. Nicklas, B. J., B. W. Penninx, A. S. Ryan, D. M. Berman, N. A. Lynch, and K. E. Dennis. Visceral adipose tissue cutoffs associated with metabolic risk factors for coronary heart disease in women. *Diabetes Care* 26:1413–1420, 2003.
27. Obisesan, T. O., M. J. Toth, P. A. Ades, and E. T. Poehlman. Central markers of body fat distribution are important predictors of plasma lipids in elderly men and women. *Exp. Gerontol.* 32: 643–651, 1997.
28. Ockene, I. S., D. E. Chiriboga, E. J. Stanek 3rd, et al. Seasonal variation in serum cholesterol levels: treatment implications and possible mechanisms. *Arch. Intern. Med.* 164:863–870, 2004.
29. Physical activity and cardiovascular health. *NIH Consens Statement* 13:1–33, 1995.
30. Rendell, M., U. L. Hulthen, C. Tornquist, L. Groop, and I. Mattiasson. Relationship between abdominal fat compartments and glucose and lipid metabolism in early postmenopausal women. *J. Clin. Endocrinol. Metab.* 86:744–749, 2001.
31. Riechman, S. E., R. E. Schoen, J. L. Weissfeld, F. L. Thaete, and A. M. Kriska. Association of physical activity and visceral adipose tissue in older women and men. *Obes. Res.* 10:1065–1073, 2002.
32. Schuit, A. J., E. G. Schouten, T. P. Miles, W. J. Evans, W. H. Saris, and F. J. Kok. The effect of six months training on weight, body fatness and serum lipids in apparently healthy elderly Dutch men and women. *Int. J. Obes. Relat. Metab. Disord.* 22:847–853, 1998.
33. Stefanick, M. L., S. Mackey, M. Sheehan, N. Ellsworth, W. L. Haskell, and P. D. Wood. Effects of diet and exercise in men and postmenopausal women with low levels of HDL cholesterol and high levels of LDL cholesterol. *N. Engl. J. Med.* 339:12–20, 1998.
34. Taylor, H. L., D. R. Jacobs Jr., B. Schucker, J. Knudsen, A. S. Leon, and G. Debacker. A questionnaire for the assessment of leisure time physical activities. *J. Chronic Dis.* 31:741–755, 1978.
35. Thompson, P. D., S. F. Crouse, B. Goodpaster, D. Kelley, N. Moyna, and L. Pescatello. The acute versus the chronic response to exercise. *Med. Sci. Sports Exerc.* 33:S438–S445; discussion S452–S453, 2001.
36. Tworoger, S. S., Y. Yasui, L. Chang, F. Z. Stanczyk, and A. McTiernan. Specimen allocation in longitudinal biomarker studies: controlling subject-specific effects by design. *Cancer Epidemiol. Biomarkers Prev.* 13:1257–1260, 2004.
37. Visser, M., L. J. Launer, P. Deurenberg, and D. J. Deeg. Total and sports activity in older men and women: relation with body fat distribution. *Am. J. Epidemiol.* 145:752–761, 1997.
38. Warnick, G. R. Enzymatic methods for quantification of lipoprotein lipids. *Methods Enzymol.* 129:101–123, 1986.
39. Weltman, A., J. P. Despres, J. L. Clasey, et al. Impact of abdominal visceral fat, growth hormone, fitness, and insulin on lipids and lipoproteins in older adults. *Metabolism* 52:73–80, 2003.