Published in final edited form as:
Atherosclerosis. 2009 December ; 207(2): 530. doi:10.1016/j.atherosclerosis.2009.05.009.

Effect of Exercise Training on Cardiometabolic Risk Markers among Sedentary, but Metabolically Healthy Overweight or Obese Postmenopausal Women with Elevated Blood Pressure

Benoit J. Arsenault, PhD[1,2], **Mélanie Côté, MSc**[1,2], **Amélie Cartier, MSc**[1,2], **Isabelle Lemieux, PhD**[1], **Jean-Pierre Després, PhD, FAHA**[1,3,*], **Robert Ross, PhD**[4], **Conrad P. Earnest, PhD**[5], **Steven N. Blair, PED**[6], and **Timothy S. Church, MD, MPH, PhD**[5]

[1] Centre de recherche de l'Institut universitaire de cardiologie et de pneumologie de Québec, Canada

[2] Department of Anatomy and Physiology, Faculty of Medicine, Université Laval, Québec, Canada

[3] Division of Kinesiology, Department of Social and Preventive Medicine, Université Laval, Québec, Canada

[4] School of Kinesiology and Health Studies, Queen's University, Kingston, Canada

[5] Pennington Biomedical Research Center, Louisiana State University System, Baton Rouge, USA

[6] Arnold School of Public Health, University of South Carolina, Columbia, USA

Abstract

Objective—To investigate the effect of exercise training on markers of the lipoprotein-lipid profile and inflammatory markers in postmenopausal overweight/obese women with a moderately elevated systolic blood pressure.

Methods—A total of 267 women [mean body mass index (BMI) =32.0±5.7 kg/m^2 and mean age=57.3±6.6 years] underwent a 6-month exercise intervention program. Exercise training was performed 3 to 4 times per week at a targeted heart rate corresponding to 50% of the maximal oxygen consumption.

Results—Compared to baseline values, mean change in relative VO_2 max (the primary endpoint) was of 1.18±2.25 mL/min*kg ($p<0.0001$), mean weight loss was of 1.4±3.3 kg ($p<0.0001$), mean reduction in waist circumference was of 2.4±6.9 cm ($p<0.0001$) and systolic blood pressure did not change significantly (−1.2±13.0 mmHg, NS). No changes were observed in markers of the lipoprotein-lipid profile. No changes were observed for plasma levels of C-reactive protein, interleukin-6, tumor necrosis factor-α and adiponectin. Changes in VO_2 max were negatively associated with changes in body weight ($r=-0.26$, $p<0.0001$) and waist circumference ($r=-0.16$, $p=0.01$), but not with changes in cardiometabolic risk markers.

*Address for correspondence: Jean-Pierre Després, PhD, FAHA, Director of Research, Cardiology, Centre de recherche de l'Institut universitaire de cardiologie et de pneumologie de, Québec, Pavilion Marguerite-D'Youville, 4th Floor, 2725, chemin Sainte-Foy, Québec, (QC) CANADA G1V 4G5, Tel: 418-656-4863 Fax: 418-656-4610, jean-pierre.despres@criucpq.ulaval.ca.

Conclusion—Although exercise training significantly increased cardiorespiratory fitness in these sedentary, but metabolically healthy obese/overweight women with a moderately elevated systolic blood pressure, no significant improvements were observed in their cardiometabolic risk profile.

Keywords

Exercise training; Cardiometabolic risk; Obesity; Postmenopausal Women

Introduction

Obesity, and particularly its deleterious form, visceral obesity, is associated with insulin resistance and with a constellation of metabolic abnormalities which include the presence of an atherogenic dyslipidemia characterized by elevated apolipoprotein B and triglyceride levels, decreased apolipoprotein A1 and high-density lipoprotein (HDL) cholesterol levels and an increased preponderance of small, dense low-density lipoprotein (LDL) and HDL particles.[1, 2] Other features of the visceral obesity include a pro-inflammatory profile characterized by increased levels of C-reactive protein (CRP), interleukin-6 (IL-6), tumor-necrosis factor-α (TNF-α) and reduced levels of adiponectin, a potentially anti-diabetic and anti-atherogenic adipokine.[3, 4] Several prospective studies have shown that physically active individuals have increased levels of cardiorespiratory fitness and are therefore at lower risk of developing insulin resistance and cardiovascular disease (CVD)-related mortality.[5, 6] Moreover, studies have also shown that increased cardiorespiratory fitness levels might reduce the CVD risk associated with either obesity or the metabolic syndrome.[7] Although studies have reported that exercise-training had beneficial impact on cardiometabolic risk markers, few studies have investigated the effects of exercise-training on cardiometabolic risk markers in overweight/obese post-menopausal women.

We therefore measured an expanded panel of cardiometabolic risk markers in overweight or obese post-menopausal women who participated to the Dose-Response to Exercise in postmenopausal Women (DREW) before and after a 6-month exercise-training program.

Materials and Methods

Study Design

A complete description of DREW design and methods has been previously described.[8, 9] The study was a randomized, dose-response exercise trial with a no-exercise control group and 3 exercise groups with incrementally higher doses of energy expenditure (4,8 or 12kcal/kg/wk [KKW]). The results of the primary endpoints of this protocol have been previously published.[9] They included changes in aerobic fitness, changes in anthropometric parameters such as body weight, body fat percentage and waist circumference and changes in basic plasma lipoprotein-lipid levels. The research protocol was reviewed and approved annually by the Cooper Institute's institutional review board, and written informed consent was obtained from all participants prior to their inclusion in the study.

Study Participants

A total of 4545 telephone screening interviews between April 2001 and June 2005 were conducted and written informed consent was obtained from 464 eligible postmenopausal women aged 45 to 75 years. Women who were sedentary (not exercising >20 minutes on ≥3 d/wk and taking <8000 steps/d assessed over the course of 1 week), overweight or obese [body mass index (BMI) of 25.0–43.0 kg/m^2], and who had a systolic blood pressure (SBP) ranging from 120.0 to 159.9 mmHg were randomly assigned to 1 of the 4 groups. Women in the no-exercise control group were asked to maintain their level of activity during the 6-month study

period. Exclusion criteria included history of stroke, heart attack, or any serious medical condition that prevented participants from adhering to the protocol or exercising safely. Participants were recruited using a wide variety of techniques, including newspaper, radio, television, mailers, community events, and e-mail distributions.

Exercise Training

Exercising women participated in 3 or 4 training sessions each week for 6 months with training intensity at the heart rate corresponding to 50% of each woman's peak VO_2. A computer-controlled exercise training management system allows for input of relevant data points on each woman (week of exercise, KKW dose according to group assignment, heart rate associated with 50% VO_2, training heart rate zone, body weight and number of visits per week). The computer then provides the appropriate power output for the cycle ergometer and the correct speed and grade for the treadmill that will elicit the programmed heart rate. Knowing the exact power output for the cycle ergometer and the treadmill, the total kilocalories expended each minute and the time needed to reach the target energy expenditure for the exercise session or for the week can then be calculated. The duration of each individual session depended on the number of visits required to reach the target KKW. During the first week, each group expended 4 KKW. Those assigned to that level continued to expend 4 KKW per week for 6 months. All the other groups increased their energy expenditure by 1 KKW a week until they reached level required for their group. All exercise sessions were performed under observation and supervision in an exercise laboratory with complete and strict monitoring of the amount of exercise completed in each session. Among women who completed the trial, those in the 4 KKW groups had a mean exercise time of 72.2±12.3 minutes per week. Women in the 8 KKW and 12 KKW groups has a mean exercise time of 135.8±19.5 and 191.7±33.7. respectively. Two exercise training facilities were used in this study: one in North Dallas and the other in Oak Cliff (South Dallas), Texas. Participants were weighed each week and their weight was multiplied by their exercise dosage to determine the number of calories to be expended for the week. Women in the exercise groups alternated training sessions on semi-recumbent cycle ergometers and treadmills. Cardiorespiratory fitness was measured using a Lode Excalibur Sport cycle ergometer as previously described.[9]

Participant Retention and Adherence

To reduce participant dropout and maintain adherence, several strategies were used including a 2-week prerandomization run-in period, behavioral contracts, and consistent support from staff members. Participants were reimbursed $150 ($75 each) for completion of baseline and follow-up assessments. Participants could earn another $350 in incentives based on adherence. For the control group, adherence was based on returning monthly step-count forms and medical symptoms questionnaires. For each month missed, $50 was deducted from the $350 incentive. For the exercise groups, the $350 was reduced by $50 for each week of missed sessions beyond the 90% adherence target. Although this incentive is a substantial amount, it was considered appropriate because the study objective was to evaluate the dose-response effects of exercise. For this reason, excellent adherence to both intervention and measurement was necessary. If DREW were testing the effectiveness of an exercise intervention as a public health strategy, such a payment would not be appropriate. However, DREW was not testing whether financial incentives encourage individuals to exercise; rather, it was evaluating specific responses to various doses of exercise.

Measurement of Cardiometabolic Risk Markers

Plasma levels of apolipoprotein B, apolipoprotein A1 and CRP were measured by a highly sensitive immunoassay that used monoclonal antibodies coated with polystyrene particles. The assay was performed with a Behring BN-100 nephelometer (Dade Behring) according to the

methods described by the manufacturer.[10] LDL and HDL particle size were measured by nondenaturing polyacrylamide gradient gel electrophoresis (2–16% for LDL and 4–30% for HDL) as previously described.[11, 12] Plasma glucose was measured enzymatically, whereas plasma insulin was measured by electrochemiluminescence.[13] ELISAs were used to measure plasma adiponectin (B-Bridge International, Inc., San Jose, CA), IL-6 and TNF-α (R&D Systems Inc., Mineapolis, Minnesota).

Statistical Analysis

Data are presented as mean ± SD. Spearman correlations were used to quantify associations between changes in anthropometric parameters and changes in cardiometabolic risk markers. Paired t-tests were performed to compare baseline and achieved levels of cardiometabolic risk markers and anthropometric parameters. Because the changes in the markers of the lipoprotein-lipid profile and in inflammatory markers were similar across the 3 intervention groups, women of the 3 intervention groups were pooled together. All statistical analyses were performed with the SAS package (SAS Institute, Cary, NC).

Results

A complete lipoprotein-lipid profile was obtained and markers of the glucose-insulin homeostasis and inflammatory markers were measured in 349 women. A total of 82 women were on the control group (Table 1), 117 women were on the 4 KKW energy deficit group, 70 women were on the 8 KKW energy deficit group and 80 women were on the 12 KKW energy deficit group for a total of 267 women with an exercise prescription. At baseline, mean age of women was of 57.3 ± 6.6 years and 46.5% of them used hormone replacement therapy. Women were all either overweight or obese (mean BMI = 32.0 ± 5.7 kg/m^2) and had an elevated SBP (mean SBP = 138.6 ± 13.0 mmHg).

Anthropometric and metabolic characteristic of women are presented in Table 2 for women of the control group and women who had the exercise intervention separately before and after the intervention period. Although the BMI of control subjects decreased by an average of 0.4 kg/m^2 and SBP decreased by an average of 3.0 mmHg, no changes were observed in cardiorespiratory fitness. Moreover, no changes were found in the cardiometabolic risk profile of women who did not exercise. In the 267 women of the intervention group, body weight and waist circumference significantly decreased. These women also improved their maximal oxygen consumption as their peak absolute oxygen consumption increased by an average of 7.8% ($p<0.001$). Besides small but significant decreases in fasting plasma insulin and glucose levels, the cardiometabolic risk profile of women who exercised did not change significantly. Although the peak absolute oxygen consumption increased in a dose-dependant manner across the 3 intervention groups, the magnitude of changes of the cardiometabolic risk profile was similar in these 3 groups (not shown). For that reason, women of the 3 intervention groups were pooled together in Table 2.

Finally, changes in VO_2 max were negatively associated with changes in body weight ($r=-0.26$, $p<0.0001$) and waist circumference ($r=-0.16$, $p=0.01$). However, neither changes in VO_2 max nor changes in body weight or waist circumference were associated with changes in cardiometabolic markers.

Discussion

In this prospective, randomized, controlled exercise trial, we found that exercise training had no major impact on the cardiometabolic risk profile of overweight or obese, post-menopausal women with moderately elevated SBP, despite considerable improvements in maximal oxygen consumption. Over the past years, a few studies have measured the effects of exercise-training

on inflammatory markers in women and conflicting results have been observed. For instance, Fairey et al.[14] have shown that plasma CRP concentrations decreased by an average of 1.39 mg/L in 25 post-menopausal women who survived breast cancer and exercised on a cycle ergometer 3 times per week for 15 weeks. In the present study, although baseline CRP values were similar, we could not find an effect of exercise training on plasma CRP levels. In another study, Olson et al.[15] have tested the effect of moderate resistance training in 16 slightly overweight women aged approximately 39 years. Although no changes were observed in the lipoprotein-lipid profile, in markers of the glucose-insulin homeostasis and in circulating adhesion molecules, they reported a significant decrease in CRP concentrations despite no significant decrease in IL-6 levels. Plasma adiponectin concentrations were also significantly increased. Another randomized control trial has investigated the effect of aerobic exercise on plasma levels of several inflammatory markers in 60 women who exercised for an average of 2 years.[16] This trial was performed in middle-aged women with a more severe degree of obesity than women of the present study. It was found that CRP levels were slightly decreased in response to this intervention program. They also reported decreases in fasting insulin and IL-6 levels and increases in adiponectin levels. However, women in that study lost a substantial amount of body weight (14 kg) compared to our study (1.6 kg).

In the present study, we have measured an expanded panel of inflammatory markers that included specific markers of the lipoprotein-lipid profile and emerging markers associated with either insulin resistance or low-grade inflammation. We had an excellent exercise adherence and low drop out rate.[9] However, our study sample was limited to healthy overweight/obese women with moderately elevate blood pressure. Otherwise, their cardiometabolic profile was normal as reflected, for instance, by a very low baseline cholesterol/HDL cholesterol ratio (<3.7). It is therefore very likely that our exercise protocol would have had an impact on our panel of cardiometabolic risk markers in men or women with a more atherogenic/diabetogenic cardiometabolic risk profile. For instance, less than 6% of our study subjects had impaired fasting glucose (fasting glucose equal or above 6.1 mmol/L) and less than 13% of women were characterized by the high-triglyceride, low-HDL cholesterol atherogenic dyslipidemia (simultaneous presence of triglyceride levels ≥1.7 mmol/L and HDL cholesterol <1.29 mmol/L). It is therefore reasonable to believe that women of the DREW trial were “metabolically” healthy despite having an important excess body weight. In this context, results from our laboratory have previously shown that an expanded waistline is not necessarily predictive of a deteriorated cardiometabolic risk profile, especially in obese women.[17] Women of the present study were therefore likely to have an increased accumulation of subcutaneous adipose tissue rather than carrying an important amount of visceral fat, which is more associated with impaired levels of cardiometabolic risk markers. Based on these observations, one could hypothesize that equally obese women with a more atherogenic body fat distribution might benefit more from such an intervention. Further studies, which are currently underway.[18] are needed to investigate the effect of exercise-training in overweight/obese post-menopausal women with a deteriorated cardiometabolic risk profile. Moreover, the fact that exercise training was performed at low intensity (50% of maximal oxygen consumption) rather than at high intensity could also explain these findings.

In conclusion, although a 6-month exercise training period significantly increased cardirespiratory fitness of healthy overweight or obese post-menopausal women with slightly elevated SBP, we found no effects of this intervention program on their cardiometabolic risk profile. This finding is likely to be attributable to the fact that these women were already “metabolically” healthy at baseline despite having an important excess body weight.

Acknowledgments

The authors would like to thank the participants and the staff and the Scientific Advisory Board of the Cooper Clinic Institute. Benoit J. Arsenault is recipient of a training scholarship from Hôpital Laval Research Centre. Mélanie Côté is supported by a scholarship from the Fonds de la Recherche en Santé du Québec (FRSQ). Amélie Cartier is supported by the training program in obesity of the Merck Frosst Canadian Institutes of Health Research research chair in obesity. Dr. Després is Scientific Director of the International Chair on Cardiometabolic Risk, which is supported by an unrestricted grant from Sanofi Aventis awarded to Université Laval.

References

1. Després JP, Lemieux I. Abdominal obesity and metabolic syndrome. Nature 2006;444:881–887. [PubMed: 17167477]
2. Sam S, Haffner S, Davidson MH, D'Agostino RB Sr, Feinstein S, Kondos G, Perez A, Mazzone T. Relationship of abdominal visceral and subcutaneous adipose tissue with lipoprotein particle number and size in type 2 diabetes. Diabetes 2008;57:2022–2027. [PubMed: 18469202]
3. Rana JS, Nieuwdorp M, Jukema JW, Kastelein JJ. Cardiovascular metabolic syndrome - an interplay of, obesity, inflammation, diabetes and coronary heart disease. Diabetes Obes Metab 2007;9:218–232. [PubMed: 17391148]
4. Yudkin JS, Kumari M, Humphries SE, Mohamed-Ali V. Inflammation, obesity, stress and coronary heart disease: is interleukin-6 the link? Atherosclerosis 2000;148:209–214. [PubMed: 10657556]
5. Blair SN, Kampert JB, Kohl HW 3rd, Barlow CE, Macera CA, Paffenbarger RS Jr, Gibbons LW. Influences of cardiorespiratory fitness and other precursors on cardiovascular disease and all-cause mortality in men and women. JAMA 1996;276:205–210. [PubMed: 8667564]
6. LaMonte MJ, Barlow CE, Jurca R, Kampert JB, Church TS, Blair SN. Cardiorespiratory fitness is inversely associated with the incidence of metabolic syndrome: a prospective study of men and women. Circulation 2005;112:505–512. [PubMed: 16009797]
7. Katzmarzyk PT, Church TS, Janssen I, Ross R, Blair SN. Metabolic syndrome, obesity, and mortality: impact of cardiorespiratory fitness. Diabetes Care 2005;28:391–397. [PubMed: 15677798]
8. Morss GM, Jordan AN, Skinner JS, Dunn AL, Church TS, Earnest CP, Kampert JB, Jurca R, Blair SN. Dose Response to Exercise in Women aged 45–75 yr (DREW): design and rationale. Med Sci Sports Exerc 2004;36:336–344. [PubMed: 14767260]
9. Church TS, Earnest CP, Skinner JS, Blair SN. Effects of different doses of physical activity on cardiorespiratory fitness among sedentary, overweight or obese postmenopausal women with elevated blood pressure: a randomized controlled trial. JAMA 2007;297:2081–2091. [PubMed: 17507344]
10. Ledue TB, Weiner DL, Sipe JD, Poulin SE, Collins MF, Rifai N. Analytical evaluation of particle-enhanced immunonephelometric assays for C-reactive protein, serum amyloid A and mannose-binding protein in human serum. Ann Clin Biochem 1998;35 (Pt 6):745–753. [PubMed: 9838988]
11. Arsenault BJ, Lemieux I, Després JP, Wareham NJ, Luben R, Kastelein JJ, Khaw KT, Boekholdt SM. Cholesterol levels in small LDL particles predict the risk of coronary heart disease in the EPIC-Norfolk prospective population study. Eur Heart J 2007;28:2770–2777. [PubMed: 17947216]
12. Pérusse M, Pascot A, Després JP, Couillard C, Lamarche B. A new method for HDL particle sizing by polyacrylamide gradient gel electrophoresis using whole plasma. J Lipid Res 2001;42:1331–1334. [PubMed: 11483636]
13. Richterich R, Dauwalder H. Determination of plasma glucose by hexokinase-glucose-6-phosphate dehydrogenase method. Schweiz Med Wochenschr 1971;101:615–618. [PubMed: 5576952]
14. Fairey AS, Courneya KS, Field CJ, Bell GJ, Jones LW, Martin BS, Mackey JR. Effect of exercise training on C-reactive protein in postmenopausal breast cancer survivors: a randomized controlled trial. Brain, behavior, and immunity 2005;19:381–388.
15. Olson TP, Dengel DR, Leon AS, Schmitz KH. Changes in inflammatory biomarkers following one-year of moderate resistance training in overweight women. Int J Obes (Lond) 2007;31:996–1003. [PubMed: 17299382]
16. Esposito K, Pontillo A, Di Palo C, Giugliano G, Masella M, Marfella R, Giugliano D. Effect of weight loss and lifestyle changes on vascular inflammatory markers in obese women: a randomized trial. JAMA 2003;289:1799–1804. [PubMed: 12684358]

17. Lemieux I, Drapeau V, Richard D, Bergeron J, Marceau P, Biron S, Mauriege P. Waist girth does not predict metabolic complications in severely obese men. Diabetes Care 2006;29:1417–1419. [PubMed: 16732038]

18. Thompson AM, Mikus CR, Rodarte RQ, Distefano B, Priest EL, Sinclair E, Earnest CP, Blair SN, Church TS. Inflammation and exercise (INFLAME): study rationale, design, and methods. Contemporary clinical trials 2008;29:418–427. [PubMed: 18024231]

Table 1

Baseline characteristics of the 349 women of the study.

Number of women	Control group 82	Intervention group 267
Age, years	57.2 ± 6.1	57.3 ± 6.6
Hormone replacement therapy, %	53.8	46.5
Weight, kg	85.0 ± 12.4	84.2 ± 11.8
Body mass index, kg/m^2	31.9 ± 3.8	32.0 ± 5.7
Waist circumference, cm	101.7 ± 12.0	100.1 ± 11.3
Systolic blood pressure, mmHg	142.4 ± 12.4	138.6 ± 13.0
Diastolic blood pressure, mmHg	81.1 ± 8.0	80.7 ± 8.4
Peak absolute VO_2, L/min	1.31 ± 0.28	1.28 ± 0.26
Peak relative VO_2, mL/Kg	15.5 ± 2.9	15.4 ± 3.1

Table 2

Anthropometric and metabolic characteristic of women of the control and intervention groups before and after the 6-month intervention.

	Control 82		Intervention 267	
Number of women	**Before**	**After**	**Before**	**After**
Weight, kg	85.0 ± 12.4	83.8.1 ± 12.6†	84.2 ± 11.8	82.8 ± 11.8*
Body mass index, kg/m^2	31.9 ± 3.8	31.5 ± 4.0†	32.0 ± 5.7	31.1 ± 3.8*
Waist circumference, cm	101.7 ± 12.0	101.7 ± 12.1	100.1 ± 11.3	97.7 ± 10.8*
Systolic blood pressure, mmHg	142.4 ± 12.4	139.4 ± 11.9†	138.6 ± 13.0	137.4 ± 14.0
Diastolic blood pressure, mmHg	81.1 ± 8.0	80.5 ± 7.6	80.7 ± 8.4	81.0 ± 8.6
Peak absolute, VO_2 L/min	1.31 ± 0.28	1.28 ± 0.28	1.28 ± 0.26	1.36 ± 0.27*
Peak relative VO_2, mL/kg	15.5 ± 2.9	15.4 ± 3.1	15.4 ± 3.1	16.6 ± 2.9*
Total cholesterol, mmol/L	5.28 ± 0.81	5.37 ± 0.96	5.18 ± 6.63	5.18 ± 0.79
VLDL cholesterol, mmol/L	0.67 ± 0.33	0.65 ± 0.26	0.65 ± 0.30	0.63 ± 0.30
LDL cholesterol, mmol/L	3.11 ± 0.68	3.23 ± 0.87	3.03 ± 0.70	3.07 ± 0.72
HDL cholesterol, mmol/L	1.51 ± 0.36	1.49 ± 0.31	1.50 ± 0.37	1.48 ± 0.34
Total cholesterol/HDL cholesterol	3.66 ± 0.88	3.73 ± 0.96	3.62 ± 0.90	3.65 ± 0.89
Triglycerides, mmolL	1.46 ± 0.72	1.41 ± 0.57	1.43 ± 0.66	1.37 ± 0.67
Apolipoprotein B, g/L	0.96 ± 0.25	0.98 ± 0.28	0.97 ± 0.23	0.97 ± 0.23
Apolipoprotein A1, g/L	1.75 ± 0.36	1.75 ± 0.32	1.79 ± 0.36	1.74 ± 0.35
LDL particle size, Å	260.2 ± 4.1	260.5 ± 4.4	260.3 ± 4.7	260.6 ± 4.9
HDL particle size, Å	80.1 ± 1.0	79.9 ± 1.0	79.8 ± 1.1	79.8 ± 1.2
Insulin, pmol/L	73.6 ± 50.4	75.8 ± 54.1	72.9 ± 39.4	69.7 ± 43.4†
Glucose, mmol/L	5.27 ± 0.75	5.35 ± 0.53	5.25 ± 0.49	5.17 ± 0.48†
Adiponectin, ug/mL	7.15 ± 3.60	7.03 ± 3.29	6.90 ± 3.28	6.80 ± 3.14
C-reactive protein, mg/L	5.59 ± 5.62	5.44 ± 4.90	5.36 ± 4.93	5.46 ± 5.67
Tumor necrosis factor-alpha, pg/mL	1.63 ± 0.50	1.58 ± 0.47	1.76 ± 0.76	1.83 ± 0.95
Interleukin-6, pg/L	2.54 ± 1.33	2.53 ± 1.30	2.70 ± 1.99	2.82 ± 2.79

VLDL, very low-density lipoprotein; LDL, low-density lipoprotein, HDL, high-density lipoprotein.

† $p<0.05$.

* $p<0.001$.