Influence of Fitness Status on Very–Low-Density Lipoprotein Subfractions and Lipoprotein(a) in Men and Women

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The purpose of this study was to examine the influence of the physical activity level of men and women on the very–low-density lipoprotein (VLDL) subfractions and lipoprotein(a) [Lp(a)]. Fifty-four men (n = 30) and women (n = 24) aged 30 to 53 years were recruited based on their level of activity over the past 2 years, and formed three groups: sedentary (S), no routine activity; recreational exercise (R), routine moderate exercise three to five times per week; and trained (T), competition-based, high-volume aerobic training five to seven times per week. Each subject underwent a maximal oxygen consumption ($\dot{V}O_2max$) test and was measured for body composition (skinfolds) and waist to hip ratio (WHR). Following a prescribed 24-hour diet and abstinence from activity, a blood sample was obtained from each subject and the plasma was analyzed for cholesterol and triglycerides (TGs) in $VLDL_1$, $VLDL_2$, and $VLDL_3$ subfractions. High-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C) and Lp(a) also were analyzed. Total VLDL-C was higher in men than in women, but no gender differences were observed in VLDL subfractions. $VLDL_1$-TG and $VLDL_2$-TG were elevated in the S group compared with groups R and T, even though total VLDL-TG, LDL-C, and HDL-C values were not different among the groups. Values for Lp(a) were not significantly different between men and women or among the groups. The two exercising groups were not different on any lipoprotein variable or WHR. $VLDL_1$-TG was inversely correlated with $\dot{V}O_2max$ and HDL-C. These results suggest that life-style activity is associated with a favorable VLDL subfraction pattern and WHR, but not Lp(a). In addition, long-term recreational activity is associated with a lipoprotein profile and WHR similar to those obtained with higher-volume exercise training.
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THE ASSOCIATION BETWEEN elevations in very–low-density lipoprotein (VLDL)-triglycerides (TGs) and an increased risk of cardiovascular disease (CVD) has not been consistently documented in the scientific literature.[1,2] However, the significance of the heterogeneity of VLDL has only recently received attention.[1,3] Each VLDL subfraction may have a unique association with the progression of atherosclerosis.[4] Elevation in the less dense subfraction, $VLDL_1$, has been associated with diabetes,[5] obesity,[6] and hypercholesteremia.[7] An indirect role of $VLDL_1$ in CVD risk may relate to its exchange of cholesterol and TG with high-density lipoprotein (HDL) through the action of cholesteryl ester transfer protein (CETP).[7,8] The role of $VLDL_1$ as a precursor in the formation of the more atherogenic low-density lipoprotein (LDL) subfraction also has been suggested.[9,10] A higher TG content of $VLDL_1$ also may contribute to decreased thrombolytic activity.[11] On the other hand, the apoprotein C-III content of the more dense $VLDL_3$ may cause this subfraction to be atherogenic.[4]

The effect of exercise training on VLDL subfraction distribution has received little attention. Endurance-trained individuals have lower total VLDL-TG and VLDL-cholesterol (VLDL-C) levels.[11] Williams et al[12] reported that 1 year of moderate exercise training in previously sedentary adults did not significantly affect small or large VLDL mass. No other studies were found that examined the association of VLDL subfractions with fitness levels.

Another apoprotein B–containing particle, lipoprotein(a) [Lp(a)], appears to be an independent risk factor for atherosclerosis,[13] although not all studies have demonstrated this association.[14] Most intervention trials and cross-sectional comparisons have demonstrated that Lp(a) is not associated with activity level.[15-18] However, a recent prospective study demonstrated an increase in this lipoprotein after 1 year of aerobic training.[19] Like VLDL-TG, Lp(a) may interfere with fibrinolytic function and promote thrombosis.[13]

We are not aware of any previous investigations examining the relationship among VLDL subfractions, Lp(a), and physical activity levels. Therefore, the purpose of this study was to examine the influence of the habitual physical activity level of healthy men and women on VLDL subfractions and Lp(a). We hypothesized that an inverse dose-response would exist between activity level and $VLDL_1$ and Lp(a) concentrations, with the most highly trained individuals having the lowest levels of these lipoproteins.

SUBJECTS AND METHODS

Fifty-four men and women aged 30 to 53 years were selected based on their activity level over the previous 2 years. The physical activity status of each subject was determined by an activity questionnaire and interview, and the groups formed were to sedentary (S), recreational exercise (R), and competitively trained (T). The questionnaire sought information on the frequency, duration, intensity (pace and heart rate), longevity, and type of activity. From these answers, weekly caloric expenditure was estimated.[20] The criteria for each group were as follows: S, no regular exercise, or exercise at a frequency of no more than one time per week for the past 2 years; R, regular, noncompetitive aerobic exercise three to five times per week (actual, 4.2 ± 0.9) for at least the past 2 years; and T, competition-based, intense aerobic exercise training five to seven times per week (actual, 5.5 ± 0.8) for at least 2 years. The fitness level of each individual was determined by measurement of maximal aerobic capacity during an exhaustive treadmill run. The groups were distinctly different in both activity level and aerobic capacity (Table 1). Each subject provided informed consent as approved by the University Health Sciences Institutional Review Board.

Each subject was screened using a health history questionnaire and

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Table 1. Subject Characteristics for Males and Females by Group

Characteristic	Sedentary			Recreational Exercise			Trained		
	M	F	M + F	M	F	M + F	M	F	M + F
Age (yr)	42.1 ± 8.3	40.5 ± 1.1	41.4 ± 6.0	42.7 ± 6.3	39.7 ± 4.2	41.4 ± 5.6	39.0 ± 4.3	39.0 ± 6.8	39.0 ± 5.3
Body weight (kg)	77.7 ± 7.2	61.7 ± 5.6	70.2 ± 10.4	81.2 ± 7.0	61.4 ± 7.4	72.7 ± 12.2	75.2 ± 6.8	59.4 ± 8.1	68.3 ± 10.8
TG ($mg \cdot dL^{-1}$)	80.1 ± 31.2	70.7 ± 29.5	74.8 ± 29.0	62.5 ± 22.8	61.6 ± 21.4	62.1 ± 21.6	60.2 ± 25.3	50.9 ± 8.4	57.1 ± 19.9
TC ($mg \cdot dL^{-1}$)	178.0 ± 29.6	153.3 ± 17.6	166.4 ± 27.1	164.5 ± 13.6	153.2 ± 11.1	159.6 ± 13.5	149.6 ± 11.9	163.2 ± 27.2	155.6 ± 20.5
$\dot{V}O_2max$ ($mL \cdot kg^{-1} \cdot min^{-1}$)	34.0 ± 4.8	29.0 ± 5.4	31.5 ± 5.6[a]	45.3 ± 3.0	36.5 ± 4.6	41.5 ± 5.8[b]	53.2 ± 6.6	46.2 ± 6.7	50.2 ± 7.4[c]
Training ($kcal \cdot wk^{-1}$)				1,800 ± 759	1,844 ± 1,140	1,818 ± 931[a]	4,479 ± 1,600	3,590 ± 979	4,113 ± 1,692[b]

NOTE. Values are the mean ± SD. M + F means with different superscripts for the same variable are statistically different ($P < .05$).
Abbreviations: M, male; F, female; TC, total cholesterol.

eliminated from the study if he/she had more than one major CVD risk factor or any disease symptom. All female participants were premenopausal non–oral contraceptive users, and were not on any estrogen therapy. A screening plasma analysis was performed for the purpose of selecting participants with normal plasma lipid status. Body composition was estimated using three skinfold measurements.[21,22] Waist to hip ratio (WHR) was assessed by taking triplicate measurements at the minimum circumference of the waist and the maximum circumference of the buttocks.[23] Subject characteristics are provided in Table 1. Before the testing, each subject maintained a 4-day dietary record, and these records were analyzed using the Food Processor IV software (Esha, Salem, OR).

Each subject underwent a maximal oxygen consumption ($\dot{V}O_2max$) test on a treadmill. After a warm-up period, the speed was set at a moderate jogging pace and then increased 0.5 mph/min for 4 minutes, and then the elevation was raised 2%/min until exhaustion. After the $\dot{V}O_2max$ test, each subject was given instructions to prepare for the blood collection session. The subject was instructed to follow a normal 24-hour diet followed by a 12-hour overnight fast. To ensure similar fasting conditions, subjects were asked to eat a snack exactly 12 hours before the morning blood collection and thereafter to refrain from all food and liquid intake except water. Vigorous exercise was not permitted for 36 hours prior to blood collection. Each female subject was scheduled for blood collection during the luteal phase of the menstrual cycle to avoid any large fluctuations in estrogen levels, which may affect VLDL-TG levels.[24]

During the blood collection session, the subject rested in a chair for 5 minutes, and then a 30-mL blood sample was collected from an arm vein into vacutainer tubes containing EDTA. The vacutainer was inverted gently six to eight times and immediately placed in ice water. Plasma was separated by centrifugation a 4°C at 3,500× g for 15 minutes in a Beckman TJ-6R centrifuge (Palo Alto, CA). Plasma was transferred to freezer tubes and stored frozen at −70°C until analyzed within 1 month.

TG levels were measured enzymatically using a reagent kit (Sigma #339; Sigma Diagnostics, St Louis, MO); cholesterol level also was measured using a reagent kit (Sigma #352). Both were quantified on a Beckman spectrophotometer (Beckman Instruments, Fullerton, CA). Interassay coefficients of variation were 2% and 5%, respectively. For all assays, samples from each group were measured in a given assay. In addition, a known standard was measured with each assay to ensure quality control.

HDL-C was analyzed enzymatically (Sigma #352-4). The HDL fraction was separated as follows: 0.1 mL precipitating reagent (PTA/$MgCl_2$) was added to .5 mL plasma and then mixed with a vortex to precipitate VLDL and LDL fractions. The samples were centrifuged for 5 minutes at 3,000× g. The supernatant was assayed for cholesterol. The coefficient of variation was 2.7%.

LDL separation was achieved using the density gradient ultracentrifugation procedure of Swinkels et al.[25] The LDL density range was 1.021 to 1.063. The LDL fraction was analyzed for cholesterol using cholesterol reagent (Boehringer-Mannheim, Indianapolis, IN) at 500 nm wavelength. The coefficient of variation was 3.2%.

VLDL subfractions (1 = flotation rate Sf 100 to 400, 2 = 60 to 100, and 3 = 20 to 60) were isolated from total plasma by density gradient ultracentrifugation. Plasma (2.5 mL adjusted to 1.2 g/mL with NaBr) was placed in the bottom of a polyallomer tube. This was overlayered with three solutions of decreasing density: 1.5 mL 1.063-g/mL, 3 mL 1.019-g/mL, and 4.5 mL distilled water. The tubes were centrifuged in a Beckman SW41 rotor for 156 minutes at 20°C and 160,000 × g in a Beckman L7-65 Ultracentrifuge. The separate VLDL subfractions were isolated by puncturing the bottom of the tube and pumping the fractions out. The fraction collector was computer-controlled. A 1-mL wash was performed after all fractions were removed and combined with the $VLDL_1$ fraction. Each VLDL fraction was assayed for cholesterol and TG. The coefficient of variation for TG in the subfractions was 5.0%, 8.0%, and 8.0% for subfractions 1, 2, and 3, respectively, and the recovery for VLDL-TG was 94% ± 4%.

The Lp(a) level was measured on a Roche Cobas Mira autoanalyzer using an immunoturbidometric kit from Incstar (Stillwater, MN). The coefficient of variation was 5.3%. The Cardiovascular Research Laboratory participates in the CDC/NIH lipid standardization program.

Subject characteristics, dietary results, VLDL subfractions, and Lp(a) differences among groups were analyzed using a 3 × 2 (group × gender) ANOVA. Significant F ratios ($P < .05$) were followed by Tukey tests to determine which pairs of groups were different ($P < .05$). Selected Pearson correlations also were determined for VLDL subfractions and Lp(a) with other variables.

Table 2. Body Composition of Males and Females by Group

Parameter	Sedentary			Recreational Exercise			Trained		
	M	F	M + F	M	F	M + F	M	F	M + F
WHR	0.99 ± 0.11	0.78 ± 0.07	0.86 ± 0.11[a]	0.88 ± 0.06	0.73 ± 0.04	0.81 ± 0.09[ab]	0.83 ± 0.03	0.76 ± 0.05	0.80 ± 0.06[b]
Body mass index	25.2 ± 2.3	22.3 ± 1.6	23.7 ± 2.5	24.9 ± 1.9	22.4 ± 2.0	24.1 ± 2.3	22.3 ± 2.5	21.7 ± 2.8	22.0 ± 2.6
Body fat (%)	21.2 ± 5.4	26.6 ± 4.2	23.7 ± 5.5[a]	17.6 ± 3.5	24.0 ± 2.6	20.3 ± 4.5[b]	12.4 ± 2.1	18.6 ± 5.1	15.1 ± 4.8[c]

NOTE. Values are the mean ± SD. M + F means with different superscripts for the same variable are statistically different ($P < .05$).

Table 3. Subject Dietary Characteristics by Group

Variable	Sedentary	Recreational Exercise	Trained
Fat	30.9 ± 4.9[a]	29.6 ± 4.7[ab]	25.9 ± 5.1[b]
SFA	10.4 ± 1.8[a]	10.1 ± 1.4[a]	8.5 ± 2.2[b]
MFA	11.3 ± 2.1	10.8 ± 2.0	9.5 ± 2.3
PUFA	7.0 ± 3.0	6.5 ± 2.3	6.8 ± 1.6
Carbohydrate	53.5 ± 4.7[a]	55.5 ± 4.4[ab]	59.4 ± 5.3[b]
Protein	15.6 ± 3.7	15.0 ± 2.0	14.7 ± 2.9
Energy intake (kcal)	1,953 ± 372[a]	2,400 ± 664[b]	2,717 ± 885[b]

NOTE. Values are the mean ± SD. Except for energy intake, all values are %. Means with different superscripts for the same variable are statistically different ($P < .05$).

Abbreviations: SFA, saturated fatty acids; MFA, monounsaturated fatty acids; PUFA, polyunsaturated fatty acids.

RESULTS

As expected, men and women across all groups were different in body weight and $\dot{V}O_2max$ (Table 1). However, there were no gender × group interactions for any of the variables in the study. Characteristics of the subjects in the three activity groups were different for $\dot{V}O_2max$ and weekly training energy expenditure (Table 1). Men and women were significantly different on each body composition variable. WHR was different between groups S and T but not between group R and either of the other groups (Table 2). Although body mass index was not different among the groups, body fat values followed a rank-order by activity group.

Dietary intake was different among the groups, with trained individuals consuming a lower percentage of fat in the diet, a higher percentage of carbohydrate, and more total calories than sedentary individuals (Table 3). No gender differences were found for most of the VLDL subfractions, except that total VLDL-C was higher in men (15.5 ± 8.9 mg/dL) than in women (10.6 ± 4.6 mg/dL). The S group exhibited significantly higher $VLDL_1$-C than the R and T groups, and higher $VLDL_2$-C than the T group. $VLDL_3$-C and total VLDL-C were not significantly different among the groups (Table 4).

LDL-C levels were not significantly different between the genders, nor was there a significant difference among activity groups (Table 4). Although women (57.3 ± 12.0 mg/dL) exhibited higher HDL-C values than men (45.6 ± 8.4 mg/dL), no significant differences were observed for HDL-C among the activity groups (Table 4). Men (34.4 ± 22.7 mg/dL) had a considerably higher mean Lp(a) value than women (18.5 ± 16.4 mg/dL), but the difference did not achieve statistical significance, due to the high variability. Values for Lp(a) were not significantly different among activity groups (Table 4).

No gender differences or gender × group interactions were noted for any of the VLDL-TG subfractions (Table 5). $VLDL_1$-TG and $VLDL_2$-TG were elevated in the S group compared with groups R and T (Fig 1). $VLDL_3$-TG and total VLDL-TG were not significantly different among the groups, and groups R and T did not differ on any of the VLDL-TG subfractions (Fig 1).

Selected correlation coefficients are presented in Table 6. As expected, the VLDL subfractions were highly intercorrelated. $VLDL_1$- and $VLDL_2$-TG were inversely related to HDL-C values. $VLDL_1$-TG was inversely related to $\dot{V}O_2max$. None of the VLDL values were significantly correlated with Lp(a), which was correlated only with total cholesterol and WHR.

DISCUSSION

The mean values for subject characteristics (Table 1) indicate that the groups were relatively homogeneous except for the expected differences in activity level (kilocalories per week) and $\dot{V}O_2max$. Dietary intake was not substantially different among the groups, although the T group averaged less dietary fat intake and more carbohydrate and energy (caloric) intake than the S group (Table 2).

One of the most consistent findings reported in the exercise literature is the lower plasma TG level in trained individuals.[11] Less information is available related to VLDL-TG and VLDL-C with training, but several investigators have observed lower values in trained individuals.[26] In the present study, total VLDL-TG and VLDL-C were lower in the exercising groups, but the differences were not significant compared with the S group, perhaps because individuals in the S group exhibited relatively low VLDL-TG and VLDL-C values. Individuals engaged in training programs generally exhibit decreased LDL-C and elevated HDL-C versus sedentary individuals.[26] However, this was not the case in the present study, although the trends were in the expected direction. The lack of significance may be the result of the relatively healthy lipoprotein profile observed in the S group (Table 4).

The primary finding of this study was the lower $VLDL_1$- and $VLDL_2$-TG in the two groups in which exercise was part of the life-style. Results from other investigations indicate that the large VLDL subfraction may be associated with diabetes,[5] obesity,[6] hypercholesterolemia,[7] and conversion of macrophages to foam cells.[1] In addition, the large VLDL appears to be a better substrate for CETP, enhancing the transfer of choles-

Table 4. VLDL-C, LDL-C, HDL-C, and Lp(a) for Males and Females by Group

Variable	Sedentary M	Sedentary F	Sedentary M + F	Recreational Exercise M	Recreational Exercise F	Recreational Exercise M + F	Trained M	Trained F	Trained M + F
$VLDL_1$-C	7.3 ± 6.1	3.5 ± 2.5	5.5 ± 5.0[a]	2.5 ± 2.0	2.4 ± 1.6	2.5 ± 1.8[b]	2.8 ± 1.5	1.4 ± 0.8	2.2 ± 1.4[b]
$VLDL_2$-C	3.7 ± 2.3	2.0 ± 1.1	2.9 ± 2.0[a]	1.7 ± 1.2	1.7 ± 1.0	1.7 ± 1.1[ab]	1.9 ± 1.3	1.0 ± 0.8	1.5 ± 1.2[b]
$VLDL_3$-C	10.4 ± 4.4	7.0 ± 1.5	8.8 ± 3.7	7.8 ± 3.4	6.8 ± 2.7	7.3 ± 3.1	14.3 ± 6.3	8.1 ± 3.2	7.9 ± 4.2
Total VLDL-C	21.4 ± 11.8	12.5 ± 4.9	17.2 ± 10.0	11.9 ± 6.1	10.9 ± 4.9	11.5 ± 5.5	14.3 ± 6.3	8.1 ± 3.2	11.6 ± 5.9
LDL-C	122.9 ± 32.5	93.0 ± 22.2	108.8 ± 31.3	101.5 ± 13.2	87.3 ± 19.3	95.4 ± 17.2	88.7 ± 14.9	97.4 ± 36.7	92.5 ± 26.0
HDL-C	40.7 ± 6.5	55.0 ± 13.6	47.4 ± 12.5	48.5 ± 9.1	55.8 ± 11.6	51.6 ± 11.1	46.5 ± 7.9	62.0 ± 11.1	53.3 ± 12.0
Lp(a)	29.0 ± 25.1	19.6 ± 14.2	24.3 ± 20.3	33.3 ± 23.0	12.6 ± 13.2	24.4 ± 21.7	25.7 ± 19.9	25.0 ± 23.0	25.4 ± 20.6

NOTE. Values are the mean ± SD (in $mg \cdot dL^{-1}$). M + F means with different superscripts for the same variable are significantly different ($P < .05$).

Table 5. VLDL-TG Subfractions for Males and Females by Group

Subfraction	Sedentary M	Sedentary F	Recreational Exercise M	Recreational Exercise F	Trained M	Trained F
$VLDL_1$-TG	29.3 ± 23.7	22.4 ± 31.5	10.7 ± 7.3	11.7 ± 7.5	12.0 ± 7.2	6.0 ± 4.4
$VLDL_2$-TG	20.0 ± 9.8	13.9 ± 5.7	10.5 ± 5.2	11.4 ± 6.8	13.0 ± 5.3	9.0 ± 5.3
$VLDL_3$-TG	17.4 ± 8.8	15.4 ± 5.3	14.0 ± 6.1	15.2 ± 6.9	18.4 ± 6.1	12.1 ± 5.9
Total VLDL-TG	66.6 ± 40.7	51.6 ± 39.8	35.2 ± 16.7	38.3 ± 18.7	43.4 ± 17.4	27.1 ± 13.1
Total TG	80.1 ± 31.2	70.7 ± 29.5	62.5 ± 22.8	61.6 ± 21.4	60.2 ± 25.3	50.9 ± 8.4

NOTE. Values are the mean ± SD ($mg \cdot dL^{-1}$). Group × gender interactions were not significantly different ($P > .05$).

terol esters from HDL to VLDL, thus reducing HDL-C levels.[7,8] These cholesterol esters may then serve as a pool for LDL-C.[8,9] In the present study, $VLDL_1$-TG also was inversely associated with $\dot{V}O_2$max and HDL-C, indicating an inverse relationship with cardiovascular fitness.

The lack of correlation between the VLDL subfractions and WHR was surprising. Williams et al[12] found that changes in large and small VLDL mass were correlated with changes in WHR following 1 year of aerobic exercise. The relatively small spread of values among subjects in the present study may account for the lack of correlation. An elevated WHR (android obesity) has been associated with CVD risk.[27] This variable was lowest in T subjects, which is consistent with other reports that have demonstrated a link between physical activity and WHR.[12,28]

Lp(a) values were not significantly different among the groups. Although Lp(a) has been identified as an independent risk factor for CVD,[13] the data have not been unanimous.[14] Attempts to decrease Lp(a) with life-style alterations have not been successful. For example, several cross-sectional studies have found no differences between trained and sedentary populations.[16,17,29] Likewise, results from at least two intervention studies indicated no significant effect of exercise training or detraining on Lp(a).[18,30] On the other hand, Holme et al[19] reported increased Lp(a) levels in individuals in an aerobic exercise program for 1 year. In the present study, Lp(a) was significantly correlated only with total cholesterol and the WHR. Other investigators[16,17] also observed little relationship between Lp(a) and other CVD variables.

One of the proposed mechanisms for Lp(a) as a CVD risk factor is enhanced thrombogenesis.[13] A similar mechanism has been proposed for VLDL-TG.[11] We found no statistical association between Lp(a) and any of the VLDL-TG subfractions (Table 6). This lack of relationship may be due to the relatively homogeneous nature of the lipoprotein profile of this subject pool. TG and Lp(a) also may affect thrombogenesis independently.

The apparent difference ($P = .06$) for Lp(a) between men and women is interesting. Most of the epidemiological studies have measured Lp(a) levels in men only. MacAuley et al[29] observed inconsistent differences in Northern Ireland between men and women depending on the age group. For all age groups

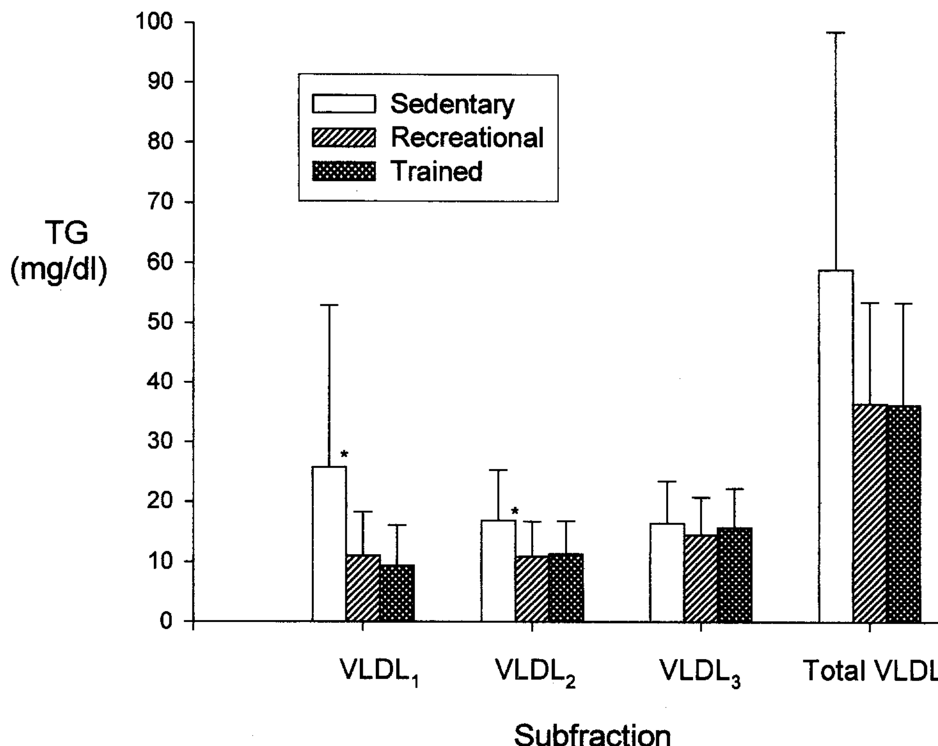

Fig 1. VLDL-TG subfractions by group: Values are the mean ± SD. *Significantly different among the groups.

Table 6. Selected Pearson Product Correlation Coefficients for VLDL-TG Subfractions and Lp(a)

	$VLDL_1$-TG	$VLDL_2$-TG	$VLDL_3$-TG	VLDL-TG	Lp(a)
$VLDL_1$-C	.864*	.699*	.669*	.877*	−.092
$VLDL_2$-C	.766*	.894*	.727*	.879*	−.018
$VLDL_3$-C	.524*	.552*	.827*	.665*	.177
VLDL-C	.759*	.741*	.836*	.862*	.043
$VLDL_1$-TG	—	.709*	.604*	.949*	−.116
$VLDL_2$-TG	.709*	—	.689*	.861*	−.145
$VLDL_3$-TG	.604*	.689*	—	.791*	.062
VLDL-TG	.949*	.861*	.791*	—	−.094
LDL-C	.258	.226	.134	.250	.307
TC	.141	.147	.154	.162	.484*
HDL-C	−.371*	−.378*	−.310	−.402*	.144
Lp(a)	−.116	−.145	.062	−.094	—
WHR	.291	.184	.203	.277	.330*
$\dot{V}o_2max$	−.340*	−.208	−.008	−.267	.147
% Body fat	.276	.261	.056	.252	−.141

*$P < .05$, 1-tailed test.

combined (N = 1,600), Lp(a) values were not significantly different between the sexes. Likewise, Israel et al[16] reported similar mean Lp(a) values in 126 men and 76 women. If the trend we observed is accurate, the higher Lp(a) values in men could account for part of their greater risk of CVD at a given age.

One of the most surprising findings in this study is the lack of difference in lipoprotein profiles between the recreational and competitively trained groups. We had hypothesized that the quantity of activity would influence the lipoprotein profile and especially the VLDL subfractions. Except for $\dot{V}o_2max$ and percent body fat, the R and T groups were similar on the variables measured in this study, including VLDL subfractions. On the other hand, there was a substantial difference in the amount of activity incorporated into the life-style of these two groups over the previous 2 years, and the $\dot{V}o_2max$ values paralleled the activity difference (Table 1). This apparently similar benefit of moderate and vigorous exercise programs is consistent with epidemiological data indicating that health benefits are derived from mild/moderate activity[31] and moderate levels of fitness.[32] However, many of the trends in the data were in the direction of more positive effects in the T group. In addition, this group could not be considered an elite group of endurance athletes. Thus, we cannot rule out the possibility that additional benefits are derived from a higher volume and/or intensity of training.

The results of this study suggest that life-style physical activity is associated with a more favorable VLDL subfraction pattern and WHR. In addition, life-style activity has little influence on the level of Lp(a). Finally, moderate recreational activity over an extended time may provide nearly as much benefit for many CVD risk parameters as higher-volume training.

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