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Lipoprotein Subfractions of Runners and Sedentary Men

Paul T. Williams, Ronald M. Krauss, Peter D. Wood, Frank T. Lindgren, Christine Giotas, and Karen M. Vranizan

From the Stanford Center for Research in Disease Prevention, Stanford University School of Medicine, Stanford, Calif., Dormer Laboratory of Medical Physics, Lawrence Berkeley Laboratory, University of California, Berkeley, Calif., Department of Medicine. University of California, San Francisco

Abstract

Serum concentrations of lipoprotein mass by flotation rate were measured in 12 long-distance runners and 64 sedentary men by analytic ultracentrifugation. The runners had significantly lower serum mass concentrations of the smaller, denser low-density lipoprotein particles of flotation rates $S_f 0-7$ (including the LDL-II, LDL-III, and LDL-IV subspecies), very-low-density lipoprotein (VLDL) particles of $S_f 20-400$, and high-density lipoprotein (HDL) particles of flotation rates $F_{1.20} 0-1.5$ (predominately the HDL3 subspecies), and higher serum mass concentrations of HDL particles with flotation rates between $F_{1.20} 2.0-9.0$ (including HDL_{2a} and HDL_{2b} and less dense particles belonging to HDL₃) than did sedentary men. Lipoprotein lipase activity was higher, and hepatic lipase activity was lower in runners than in the sedentary men. Thus, the effects of endurance exercise training to lower LDL may be specific to the smaller, denser LDL particle region. Similarities in the lipoprotein mass profiles of the runners and the low-risk profiles of sedentary, middle-aged women suggest the effects of common metabolic factors possibly leading to reduced risk of coronary artery disease.

Long-distance running and other endurance activities are reported to elevate plasma concentrations of high-density lipoprotein (HDL) cholesterol, and to decrease plasma concentrations of low-density (LDL) and very-low-density lipoprotein (VLDL) cholesterol in men and women [1–7]. Previous studies of lipoproteins in relation to exercise have focused primarily on the measurements of the total cholesterol and various protein components of the HDL, LDL, and VLDL particles. These variables may not adequately portray the relationship of endurance exercise training to lipoprotein concentrations since there is much evidence to suggest that multiple subspecies of lipoprotein particles exist for these three lipoprotein species [8–12]. The technique of analytic ultracentrifugation may be used to estimate the serum mass concentrations of lipoprotein subspecies, and to measure directly the mass concentrations of finer subdivisions of very-low-density (14 intervals), intermediate-density (4 intervals), low-density (11 intervals), and high-density lipoproteins (15 intervals) by particle flotation rate. Differences in the serum lipoprotein mass concentrations of runners and sedentary men have not been previously reported for LDL and HDL subspecies, nor for individual flotation intervals within HDL, LDL, and VLDL particle regions.

LIPOPROTEIN HETEROGENEITY

The traditional measurement LDL-cholesterol is a composite measurement that includes the cholesterol content of both the LDL ($S_f 0-12$) and intermediate density lipoprotein (IDL, $S_f 12-20$) particles. Further heterogeneity within the LDL particle region ($S_f 0-12$) is evident from the presence of distinct isopycnic banding regions when LDL is subjected to density gradient ultracentrifugation, from the multiple bands that appear for gradient polyacrylamide gel electrophoresis of LDL, and from correlational analyses of the relationships of peak flotation rates from analytic ultracentrifugation [8–10]. At least four major subspecies of LDL particles exist within the flotation interval of $S_f 0-12$ as assessed by the analytic ultracentrifuge: LDL-I

(approximately S_f 7.5-10.5), LDL-II (S_f 5.7-7.5), LDL-III (S_f 4.1-5.7), and LDL-IV (S_f 0-4.1) [8,9].

Two major HDL subspecies are recognized: a more-dense HDL₃ subspecies and a less-dense HDL₂ subspecies. Serum concentrations of HDL₃ are most frequently approximated from analytic ultracentrifugation as the total lipoprotein mass for flotation (F) rates between $F_{1.20}$ 0-3.5 and those of HDL₂ as the total mass for $F_{1.20}$ 3.5-9.0 [9], or alternatively, by a computer algorithm that fits separate distributions to estimate HDL_{2a}, and HDL_{2b}, and HDL₃ particle distributions [10,11]. Their partial separation is also achieved by preparative centrifugation, density gradient ultracentrifugation, gradient polyacrylamide gel electrophoresis, and zonal ultracentrifugation [13].

CARDIOVASCULAR DISEASE RISK

Much evidence suggests that high-, low-, intermediate-, and very-low-density lipoproteins and their subspecies have different relationships to coronary heart disease (CHD). High serum concentrations of the total cholesterol [14], total apolipoprotein (apo) A-1 [15], and the total lipoprotein mass [16] of HDL particles appear to provide protection against CHD. Low concentrations of either the HDL₂, HDL₃, or both the HDL₂ and HDL₃ subspecies of HDL are associated with increased CHD risk and severity. Two studies have compared the level of coronary disease with HDL subspecies in cross-sectional samples of men who received coronary arteriography: Miller et al [17] found the degree of coronary stenosis to correlate inversely with HDL₂-cholesterol concentrations but not with HDL₃-cholesterol concentrations in plasma, and Levy et al [18] reported that the number of severely diseased coronary arteries correlated inversely with serum concentrations of HDL₃-mass but not with HDL₂-mass as measured by analytic ultracentrifugation. Ballantyne et al [19] reported that survivors of myocardial infarctions had less HDL-total mass, HDL₂-cholesterol, HDL₂-phospholipid, HDL₂-protein, and HDL₂-apo A-I than did apparently healthy case-control subjects. The only prospective study to measure both HDL₂ and HDL₃ that we are aware of found that low serum concentrations of both were significantly predictive of future CHD [16].

The association between CHD and LDL-cholesterol is well established [20], but less is known about the relationship of CHD to lipoprotein subfractions within the low to very-low-density regions. High serum concentrations of total LDL-mass (S_f 0-1 2) were reported to correlate with the number of severely diseased coronary arteries [18] and to predict myocardial infarctions prospectively in men [16], and VLDL-mass of both S_f 20-100 and S_f 100-400 predicted myocardial infarctions in the Framingham cohort [16]. Women have on the average less LDL-cholesterol, LDL-mass of S_f 0-7, less IDL-mass, and less VLDL-mass of S_f 20-400, and more HDL₂-mass and LDL-mass of S_f 7-12 than men [10]. Some or all of these lipoprotein differences may contribute to the lower CHD incidence in women vis-a-vis men.

To understand better the associations between lipoprotein concentrations and long-term exercise training, and the possible roles of hepatic and lipoprotein lipase activity levels in mediating these associations, this report compares the lipoprotein subfraction concentrations and lipase activity levels of 12 middle-aged long-distance male runners and 81 middle-aged sedentary men (designated as nonrunners in the text that follows) who volunteered to later participate in a one-year running program [5-7]. Results are also presented by individual flotation intervals to delineate the runner versus nonrunner lipoprotein differences to the finest resolution currently distinguished by analytic ultracentrifugation.

MATERIALS AND METHODS

Sample and Laboratory Procedures

The 12 long-distance runners were selected because of their participation in the sport for several years and their moderate or high activity level at the time of the survey (mean $\pm$ SD: 64 $\pm$ 35 km/wk). Nonrunners were neither currently on a program of regular exercise (defined as regular participation 3 or more times per week), nor employed in a strenuous job [5–7].

Blood from runners and nonrunners was collected after an overnight fast (12–14 hours) and plasma cholesterol, triglyceride, HDL-cholesterol, LDL-cholesterol, and VLDL-cholesterol were measured by the methods of the Lipid Research Clinics [21]. Their serum lipoproteins were measured by analytic ultracentrifugation [22], and concentrations of total lipoprotein mass were estimated using computer techniques [8,9,22] for 15 HDL flotation intervals (half-integer increments between $F_{1,20}$ 0–6.0 and integer increments thereafter), 11 LDL flotation intervals between S_f 0–12 (integer increments between S_f 0–10 and then S_f 10–12), 4 IDL flotation intervals between S_f 12–20 (two unit increments), and 14 VLDL flotation intervals between S_f 20–400 (increments of ten units below S_f 100 and increments of 50 units thereafter).

Postheparin lipoprotein lipase and hepatic lipase activities were determined for the 12 runners and 20 randomly selected nonrunners from blood samples collected in tubes containing 2 U heparin/mL whole blood, 10 minutes after rapid intravenous administration of 10 U/kg body weight of sodium heparin. Extrahepatic (ie, lipoprotein) lipase was distinguished from hepatic lipase by its selective inactivation by protamine sulfate [23].

Statistical Procedures

Serum lipoprotein concentrations and lipase activity levels of runners and nonrunners are summarized in terms of the means and standard deviations for the two groups. The modest sample size of the exercise group may increase the sensitivity of the analysis to extreme or outlier observations and suggests caution in the use of parametric techniques that postulate asymptotic normality. Therefore, the significance of the differences between the two groups are evaluated initially by the nonparametric Wilcoxon sign rank test (Similar results were obtained for the parametric t-test). Analysis of covariance is used to adjust the runner and nonrunner lipoprotein differences for effects due to adiposity.

RESULTS

Although none of the runners smoked cigarettes, 17 of the 81 nonrunners were smokers who smoked an average of 14.9 cigarettes per day. These 17 sedentary smokers had significantly lower ($P \leq 0.05$) plasma HDL-cholesterol (mean $\pm$ SD: 44.9 $\pm$ 8.5 vs. 49.6 $\pm$ 8.7 mg/dL) and serum HDL-mass of both the predominately HDL₂ density region of $F_{1,20}$ 3.5–9.0 (29.2 $\pm$ 31.0 vs. 42.7 $\pm$ 31.0 mg/dL) and the predominately HDL₃ density region of $F_{1,20}$ 0–3.5 (212.5 $\pm$ 35.4 vs. 236.6 $\pm$ 35.7 mg/dL) than the 64 sedentary men who did not smoke (Fig 1). Therefore, to eliminate the effects of cigarette smoking on the lipoprotein differences of the runners and nonrunners, we excluded the 17 smokers from the sample of sedentary men and compared the lipoprotein profiles of the 12 runners with the lipoprotein profiles of the remaining 64 nonrunners who did not smoke cigarettes (16 of whom received post heparin plasma lipase measurements) in all subsequent analyses.

Low-Density Lipoproteins

Table 1 shows that the runners had significantly lower concentrations of plasma total cholesterol and LDL-cholesterol and serum lipoprotein mass of the smaller LDL particles of S_f 0–7 (including LDL-II, LDL-III, and LDL-IV) than the nonrunners, but that serum

lipoprotein mass of S_f 7-12 (predominantly LDL-I) was not significantly different between the two groups. LDL-mass concentrations of individual flotation intervals were significantly lower in runners than nonrunners for the five individual flotation intervals within S_f 2-7 (data not shown). Figure 2, which compares the average distribution of the LDL particles by flotation rate for the runners and nonrunners, shows that the lower concentrations of the slower floating, smaller LDL particles of S_f 0-7 in the runners occurred without a proportional increase in the faster floating particles of S_f 7-12. This finding suggests that running reduced the mass of the smaller LDL particles as opposed to shifting the lipoprotein mass distribution from the smaller to the larger LDL particles.

Intermediate and Very-Low-Density Lipoproteins

Table 1 reveals no significant group differences in serum IDL concentrations, but significantly lower triglyceride, VLDL-cholesterol, and VLDL-mass concentrations (total mass of S_f 20-400 and individually significant for the 10 flotation intervals occurring within the S_f 20-200 region) in runners than nonrunners.

High-Density Lipoproteins

Figure 3 shows that runners had less HDL-mass for the three individual flotation intervals between $F_{1.20}$ 0-1.5 (predominately HDL₃) and more HDL-mass for the individual flotation intervals between $F_{1.20}$ 2.0-9.0 (predominately HDL₂) than nonrunners. The mean total serum lipoprotein mass concentrations of these two HDL regions were also statistically significant between the groups (Table 1). The difference curves (Fig 3, lower panel), calculated as the HDL-mass distribution of male runners minus the HDL-mass distribution of male nonrunners, reveals two peaks in the $F_{1.20}$ 2.0-9.0 region. These peaks correspond to two HDL₂ subspecies: smaller, slower-floating particles of HDL_{2a} and larger, faster-floating particles of HDL_{2b}, that can be separated by ultracentrifugation and electrophoretic processes [11].

The traditional estimate of HDL₃ as the total lipoprotein mass between $F_{1.20}$ 0-3.5 was not significantly different ($P = 0.34$) between runners (245.4 ± 41.2 mg/dL) and nonrunners (236.6 ± 35.7 mg/dL). The absence of a significant difference appears to be due to a multiplicity of effects within this region; compared to the mean serum concentrations in nonrunners, the HDL-mass of runners was lower for $F_{1.20}$ 0-1.5 and higher for $F_{1.20}$ 2-3.5 (Fig 3). HDL₂, as estimated by the lipoprotein mass concentration within $F_{1.20}$ 3.5-9.0, was significantly higher ($P = 0.0002$) in runners (88.8 ± 34.8 mg/dL) than nonrunners (42.7 ± 31.0 mg/dL). Also, plasma HDL-cholesterol concentrations were 31% higher in runners than nonrunners.

Lipases

The 12 long-distance runners had significantly higher mean lipoprotein lipase activity and significantly lower mean hepatic lipase activity than the 16 nonrunners with lipase measurements. These findings are consistent with studies that have shown that adipose and muscle tissue of endurance-trained runners have higher lipoprotein lipase activities than those of nonrunners [24].

Adjustment for Adiposity

The long-distance runners were significantly leaner than the nonrunners (Table 1). However, an analysis of covariances (not displayed) suggests that the differences in lipoprotein concentrations are probably not entirely attributable to their differences in leanness. Adjustment for body mass index (kg/m^2) reduces slightly the significance of the plasma cholesterol difference ($P=0.06$) and the serum HDL-mass difference of $F_{1.20}$ 0-1.5 ($P=0.07$), but all of the other lipoprotein differences identified as significant in Table 1 when unadjusted, remain significant at $P \leq 0.05$ after adjustment for body mass index.

Adjustment for body mass index reduced the significance of the difference between the runners and nonrunners for both post heparin lipoprotein lipase ($P=0.09$) and hepatic lipase activities ($P=0.29$). The coefficient relating body-mass index with lipase activities in the analysis of covariance was statistically significant for hepatic lipase ($P=0.003$) but not for lipoprotein lipase activity ($P=0.09$).

These findings for lipoproteins and lipases must be interpreted cautiously in light of the indirectness of the obesity measurement (for example, as compared to hydrostatically determined body density) and the potentially complex interrelationships that may exist between exercise-induced weight loss and changes in lipoprotein concentrations [7].

DISCUSSION

Our analyses suggest that low levels of LDL-cholesterol in men who have regularly engaged in long distance running for several years primarily reflects a reduction in the serum concentrations of the smaller, less-buoyant LDL particles. Runners were also more likely to have higher serum mass concentrations of the larger, more-buoyant HDL particles (mostly HDL₂) and lower serum mass concentrations of triglyceride, total cholesterol, and VLDL cholesterol, and the smaller and less-buoyant HDL particles (predominantly the most dense particles of the HDL₃ region) than sedentary men. Previous studies have reported that HDL₂-cholesterol concentrations are elevated in association with exercise, whereas reports for HDL₃-cholesterol are mixed, with studies reporting decreased, increased, or no change in HDL₃-cholesterol in association with exercise [4,5,7,25–29]. These inconsistencies may relate in part to the existence of particle heterogeneity within these categories [30,31] and to the variable definitions of the HDL₂ and HDL₃ particle regions for different assays.

Analytic ultracentrifugation provides a means to discern lipoprotein subpopulation differences between runners and nonrunners with a high degree of resolution using lipoprotein mass concentrations within individual flotation intervals. Our previous one-year training study of initially sedentary men revealed a significant increase in HDL mass in association with exercise (i.e., miles run per week) for individual flotation intervals within the F_{1,20}3.5-9 and positive (albeit nonsignificant) correlations between exercise level and HDL-mass concentrations for intervals within F_{1,20} 2.0-3.5 [32]. Among men who have run long distances for at least several years, statistically significant increases in HDL were observed over the entire F_{1,20} 2.0-9.0 region. The lower serum concentrations of denser, smaller LDL particles in male long-distance runners compared to nonrunners is consistent with their elevated serum concentrations of less-dense HDL-mass of F_{1,20} 2.0-9.0 and the strong inverse correlation which exists between these two lipoprotein subgroups [10].

Low Risk Lipoprotein Profiles of Long-Distance Runners

The lipoprotein profiles of the 12 male long-distance runners diverge from the lipoprotein profiles of sedentary men in the direction characteristic of the lipoprotein mass distribution in women [10]. Male long-distance runners and sedentary women in middle age both have significantly higher serum concentrations of HDL-mass between F_{1,20} 2.0-9.0 (Fig 4) and significantly lower serum concentrations of LDL-mass between S_f 0-7 (Fig 5) and VLDL-mass between S_f 20-400 (not displayed) than sedentary, middle-aged men. In men, running does not appear to affect the serum LDL concentrations of S_f 7-12, which are generally higher in sedentary women than in sedentary men (Fig 5).

While the similarities in lipoprotein profiles of long-distances runners and sedentary women do not necessarily require a common antecedent, both male long-distance runners [33–35] and sedentary women [23] have increased lipoprotein lipase activity and reduced hepatic lipase activity in post heparin plasma compared to sedentary men. Nikkila et al estimated that total

body extrahepatic lipase activity of long-distance male runners was similar to the total body activity level of sedentary women [24]. In the case of runners, increased lipoprotein lipase activity may be a response to their high caloric flux over an extended period, and this may produce greater quantities of VLDL and chylomicron surface components to be incorporated into HDL particles [36]. Hepatic lipase is postulated to promote the removal of HDL lipid components in the liver [36]. Conceivably, reduced hepatic lipase activity could result in increased serum HDL₂ concentrations in runners through slower clearance.

Reduced hepatic and increased lipoprotein lipase activity may also contribute to similarities in the LDL-lipoprotein mass concentrations in male runners and sedentary women. Adipose tissue lipoprotein lipase activity is reported to correlate negatively with serum LDL-mass concentrations of individual flotation intervals within S_f3-5 and positively with the mass contained within the S_f7-10 region in sedentary, moderately obese middle-aged men examined cross-sectionally [37]. In cynomolgus monkeys, the administration of antisera to inhibit hepatic lipase activity has been reported to decrease serum concentrations of smaller LDL particles (S_f0-9) and increase concentrations of larger LDL and IDL (S_f9-20) [38].

Selection Bias

Cross-sectional comparisons of elite exercisers and sedentary men usually cannot distinguish the physiologic consequences of training from differences arising from self-selection. We have reported elsewhere a tendency for men who choose to take up running as a sport to have significantly higher plasma HDL-cholesterol and lower plasma triglyceride concentrations prior to participation in the sport [6]. In the present report we have attempted to minimize biases accruing from the self-selection of the 12 long-distance runners by comparing their lipoprotein values with the lipoprotein concentrations of men who were willing to participate later in a one-year exercise program. However, many of these sedentary men who were randomized into the exercise group failed to achieve the level of exercise apparently required to promote favorable changes in lipoprotein concentrations [5,6]. A more conservative statistical analysis of our data that takes into account both the willingness and ability of the men to run, compares the lipoprotein concentrations of the 12 long-distance runners with the baseline lipoprotein values of 21 sedentary men whose level of participation exceeded the median number of miles run by their group (ie, greater than 8.6 miles per week). With the exceptions of total cholesterol and HDL-mass of F_{1,20} 0-1.5 (P=0.07), all of the significant lipoprotein differences of Table 1 remained significant at P≤0.05 for this more conservative comparison involving the much reduced sample of 21 sedentary men. Although we can not match precisely the current running levels of the long-distance runners with the future running levels of the sedentary men, we find little evidence from these analyses to suggest that self-selection produced the lipoprotein differences of Table 1.

Limitations and Caveat

Although dietary information was not collected for the 12 long-distance runners, other studies suggest that the composition of the runners' and nonrunners' diets are unlikely to have produced the observed differences in lipoprotein concentrations. For example, Blair et al found that long-distance runners consumed more calories than sedentary men, but that with the possible exception of higher intakes of alcohol and miscellaneous carbohydrates, the composition of the diets of the runners (as percent of total calories) was not different from the dietary composition for sedentary men [39]. (See, however, caveats expressed by Thompson, et al [40] concerning the self-reported diet records of runners.) Longitudinal [41] and cross-sectional [32] studies suggest that moderately higher alcohol intake primarily elevates HDL₃, and has little effect on their HDL₂ or LDL subspecies. This suggests that a higher alcohol intake for the runners than for the nonrunners may contribute in part to the elevation of some

of the less-dense particles within the $F_{1,20}$ 0-3.5 region in the runners vis-a-vis nonrunners, but probably does not account for their other lipoprotein differences.

In conclusion, the higher serum concentrations of HDL-mass between $F_{1,20}$ 2.0-9.0 and the lower serum concentrations of HDL-mass of $F_{1,20}$ 0-1.5, LDL-mass of S_f 0-7, and VLDL-mass of S_f 20-400 reported here in runners vis-a-vis sedentary men could not be attributed to greater leanness or to self-selection in runners. The causal role of running is not necessarily proved by the lipoprotein differences between the exercising and the sedentary men, and caution should be used in extrapolating these results obtained for a modest, select sample of men to more general populations. Nevertheless, the similarities between the lipoprotein profiles of male long-distance runners and sedentary women suggest that long-distance running may reduce the risk of coronary heart disease through metabolic changes similar to those operating in women.

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Fig 1.

Upper panel displays the average distributions of serum high-density (HDL) lipoprotein mass concentration by flotation rate in sedentary male nonsmokers (N = 64) and sedentary male smokers (N = 17). The average HDL mass distributions for the nonsmokers and the smokers are computed by averaging the heights of the individual curves of the 64 nonsmokers and the 17 smokers, respectively, at each flotation rate. The HDL difference curve of the lower panel is computed by subtracting the average HDL-mass concentration of the nonsmokers from the average for the smokers.

Fig 2.

Upper panel displays the average distributions of serum low-density lipoprotein mass concentration by flotation rate for male runners (N=12) and male nonrunners (N=64). The lower panel displays the LDL difference curve for runners minus nonrunners.

Fig 3.

Upper panel displays the average distribution of serum high-density lipoprotein (HDL) mass concentration by flotation rate for male runners (N=12) and male nonrunners (N = 64). The lower panel displays the HDL difference curve for runners minus nonrunners.

Fig 4. HDL difference curves for 36 sedentary women minus 55 sedentary men, and 12 male runners minus 64 male nonrunners, all of middle age. The sedentary men and women are a subset of a previously published cross-sectional study [10], who were selected to be between 30–55 years and not on oral contraceptives or estrogen supplements.

Fig 5.

LDL difference curves for 36 sedentary women minus 55 sedentary men, and 12 male runners minus 64 male nonrunners, all of middle age. The sedentary men and women are a subset of a previously published cross-sectional study [10], who were selected to be between 30–56 years and not on oral contraceptives or estrogen supplements.

Table 1

Comparison of age, body mass index, lipids, lipoproteins, and hepatic and lipoprotein lipase measurements in cross-sectional samples of long-distance runners and sedentary men.

	Runners (mean±SD)	Nonrunners (mean±SD)	Difference (mean±SE)	Significance † (P)
Age (y)	46.9±7.5	45.7±6.1	1.3±2.3	0.81
Body mass index (kg/m ²)	22.6±2.0	25.1±3.3	-2.5±0.7	0.006
Lipids and lipoproteins				
Plasma total cholesterol (mg/dL)	190.9±36.6	217.0±31.1	-26.1±11.3	0.02
Plasma total triglycerides (mg/dL)	70.8±35.0	123.0±59.3	-52.2±12.5	0.001
Plasma HDL-cholesterol (mg/dL)	64.9±12.5	49.6±8.7	15.3±3.8	0.0001
Serum HDL-mass of F _{1,20} 0-1.5 (mg/dL)	70.0±13.7	82.3±17.5	-12.3±4.5	0.02
Serum HDL-mass of F _{1,20} 1.5-2.0 (mg/dL)	51.2±8.4	52.3±7.8	-1.1±2.6	0.98
Serum HDL-mass of F _{1,20} 2.0-9.0 (mg/dL)	213.0±45.6	144.8±47.9	68.2±14.5	0.0002
Plasma LDL-cholesterol (mg/dL)	116.1±30.7	147.0±27.5	-30.9±9.5	0.004
Serum LDL-mass of S _f 0-7 (mg/dL)	138.4±45.3	227.6±67.9	-89.2±15.6	0.0001
Serum LDL-mass of S _f 7-12 (mg/dL)	136.7±39.8	134.2±43.8	2.5±12.7	0.85
Serum IDL-mass of S _f 12-20 (mg/dL)	34.3±18.2	43.8±20.7	-9.5±5.9	0.16
Plasma VLDL-cholesterol (mg/dL)	9.1±8.3	20.4±11.7	-11.3±2.8	0.001
Serum VLDL-mass of S _f 20-400 (mg/dL)	36.8±41.6	106.1±72.0	-69.3±15.0	0.001
Postheparin lipase activity				
Lipoprotein lipase (mEq fatty acid/ml/h)	5.0±1.8	3.6±1.2	1.4±0.6	0.04
Hepatic lipase (mEq fatty acid/ml/h)	4.1±2.1	6.5±2.6	-2.4±0.9	0.02

* Sample sizes are 12 runners and 64 nonrunners for all lipid and lipoprotein variables, age and body mass index; and 12 runners and 16 nonrunners for lipoprotein and hepatic lipase measurements.

† All significance levels are obtained from two sample Wilcoxon sign rank tests.