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Race differences in the response of postheparin plasma lipoprotein lipase and hepatic lipase activities to endurance exercise training in men
Results from the HERITAGE Family Study

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Abstract

Endurance exercise training is known to produce favorable changes in the metabolic profile including reduced plasma triglyceride (TG) and increased high-density lipoprotein (HDL) cholesterol concentrations. These metabolic improvements are likely to contribute to the reduced coronary heart disease (CHD) risk often observed in physically active individuals. However, the physiological mechanisms responsible for such improvements in TG and HDL cholesterol concentrations with endurance exercise are not fully understood. The effect of a 20-week endurance exercise training program on plasma lipoproteins as well as on post-heparin plasma lipoprotein lipase (PH-LPL) and hepatic lipase (PH-HL) activities were therefore examined in a sample of 200 White and 69 Black men who were part of the HERITAGE Family Study. As expected, there were decreases in adiposity and in abdominal fat accumulation following training in both White and Black men. We also found that exercise training was associated with decreases in plasma cholesterol, TG and apolipoprotein B levels, as well as with an increase in HDL cholesterol concentrations in White men. In contrast, Black men showed an increase only in HDL_2 cholesterol over the 20-week period. Higher PH-LPL and lower PH-HL activities were noted in both ethnic groups at follow-up. Whereas in White men improvement of the lipoprotein–lipid profile was related to increased PH-LPL activity, no association between PH-LPL (or PH-HL) and lipoprotein–lipid variables was observed in Black men. Results of the present study suggest that in Whites, the increase in PH-LPL activity in response to endurance exercise training is associated with a better lipoprotein–lipid profile, therefore reducing CHD risk. However, the generally better metabolic profile of Black individuals may minimize further improvement of lipoprotein–lipid concentrations by exercise training.

1. Introduction

Physical activity is known to have beneficial effects on the metabolic risk profile. For example, endurance exercise training has been shown to improve insulin

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sensitivity [1–4], decrease plasma triglyceride (TG) concentrations, as well as increase high-density lipoprotein (HDL) cholesterol levels [1,5–11], especially in the HDL_2 subfraction. These changes are believed to be responsible, at least in part, for the reduced risk of coronary heart disease (CHD) often reported in physically active or fit individuals [12–18]. Although the physiological mechanisms underlying these metabolic adaptations are not fully understood, changes in the activity of lipoprotein lipases may play a major role in the more favorable lipoprotein–lipid profile of physically active individuals as post-heparin plasma lipoprotein lipase (PH-LPL) activity has been reported to be higher in endurance-trained individuals compared to inactive controls [19,20].

Race differences in fasting plasma lipoprotein concentrations have already been reported, with Blacks being characterized compared to Whites by lower plasma TG and higher HDL cholesterol concentrations [21–23]. Concordant with these previous studies, Blacks of the HERITAGE Study are characterized by higher PH-LPL and lower hepatic lipase (PH-HL) activities compared to Whites [24]. However, the comparison of the effects of regular endurance exercise training on lipoprotein levels, as well as on PH-LPL and PH-HL activities in individuals of different ethnic backgrounds have not been thoroughly examined. Because the design of HERITAGE Family Study offered us a unique opportunity to study these issues, the present study examined the response of and associations between plasma lipoprotein–lipid levels and activities of PH-LPL and PH-HL following a 20-week endurance exercise training program in White and Black men.

2. Methods

2.1. Subjects

The HERITAGE Family Study cohort has been previously described [25]. This paper describes data from 200 White and 69 Black men who completed the training program. Subjects were healthy and sedentary and met a number of inclusion and exclusion criteria [25]. The study protocol had been previously approved by the Institutional Review Board at each of the four clinical centers (Arizona State University/Indiana University since January 1996, Laval University, University of Minnesota and The University of Texas at Austin/Texas A&M since September 1997). Informed consent was obtained from each subject. Subjects underwent the same battery of tests before and following exercise training.

2.2. Endurance exercise training program

Subjects completed a 20-week endurance exercise training program (3 days/week for a total of 60 exercise sessions) on cycle ergometers that were computer controlled to maintain the participants' heart rates at fixed levels associated with programmed percentages of their aerobic capacity (V_{O_2max}). The training program started at 55% V_{O_2max} for 30 min/session and gradually increased to 75% V_{O_2max} for 50 min/session, where it was maintained during the last 6 weeks of training.

2.3. Anthropometry, body composition and fat distribution

Body weight, height, waist and hip circumferences were measured following standardized procedures [26], and the waist-to-hip ratio (WHR) was calculated. Body density was measured by the hydrostatic weighing technique [27]. The mean of the highest three (out of ten) measurements was used in the calculation of percent body fat from body density as previously described [28,29]. Fat mass was obtained by multiplying body weight by percent body fat. These measurements are highly reproducible, with no difference between clinical centers and no drift over time [28]. Visceral adipose tissue (AT) accumulation was assessed by computed tomography using previously described procedures [30,31]. Briefly, subjects were examined in the supine position with both arms stretched above the head. The scan was performed at the abdominal level (between L4 and L5 vertebrae) using an abdominal scout radiograph in order to standardize the position of the scan to the nearest millimeter. Total AT area was calculated by delineating the abdominal scan with a graph pen and then computing the AT surface with an attenuation ranging from −190 to −30 Hounsfield Units [30–32]. The abdominal visceral AT area was measured by drawing a line within the muscle wall surrounding the abdominal cavity. The abdominal subcutaneous AT area was calculated by subtracting the visceral AT area from the total abdominal AT area.

2.4. Plasma lipid, lipoprotein and apolipoprotein measurements

Blood sampling was achieved in subjects after a 12-h fast. Cholesterol and TG levels were determined in plasma and in lipoprotein fractions by enzymatic methods using the Technicon RA-500 analyzer (Bayer Corporation Inc, Tarrytown, NY), as previously described [33]. Plasma very low-density lipoprotein ($d < 1.006$ g/ml) were isolated by ultracentrifugation and the HDL fraction obtained after precipitation of low-density lipoproteins (LDL) in the infranatant ($d > 1.006$ g/ml) with heparin and $MnCl_2$ [34]. The cholesterol and TG contents of the infranatant fraction were measured before and after the precipitation step. Apolipoprotein (apo) B concentration was measured in plasma by the rocket immunoelectrophoretic method of Laurell [35],

as previously described [36]. The lyophilized serum standards for apolipoprotein measurements were prepared in our laboratory and calibrated with reference standards obtained from the Centers for Disease Control (Atlanta, GA). The cholesterol content of HDL_2 and HDL_3 subfractions was also determined after further precipitation of HDL_2 with dextran sulfate [37]. Reproducibility of all lipid–lipoprotein measurements has been examined and is excellent [38].

2.5. Post-heparin plasma lipase activities

Post-heparin LPL and HL activities were also measured on one occasion in subjects after a 12-h overnight fast, 10 min after an intravenous injection of heparin (60 IU/kg body mass). The post-heparin plasma lipase activities (PHLA) were measured using a modification of the method of Nilsson-Ehle and Ekman [39], as previously described [40]. The two lipolytic enzyme activities were expressed as nmoles of oleic acid released per ml of plasma per min. These measures are also highly reproducible [38].

2.6. Statistical analyses

Spearman correlation coefficients were used to quantify associations between variables. Data were analyzed using a 2×2 experimental model for repeated measures. This allowed for the assessment of within-race effects (repeated measure), between-race effects (Whites vs. Blacks) and a within x between-race interaction term. As reported in the original report on the aims, design, and measurement protocol of the HERITAGE Family Study [25], the sample size for Caucasian subjects had been determined by power studies for numerous types of data analyses (epidemiological models of response to exercise training, path analysis, segregation analysis, association and linkage studies). The smaller group of Black men raised the possibility of insufficient statistical power for differences noted in these individuals. We have therefore tested the following hypothesis in order to quantify the probability (power) that a study with 69 subjects will detect a relationship between an independent and a dependent variable at a two-sided 0.05 significance level, for a 10% change in a variable. Our analyses revealed that a study on a sample of 69 subjects will have enough power (usually around 80% or more) to detect a 10% change in variable up to a standard deviation that is three times greater than the change of the variable per se. These assumptions are pretty conservative and are, at least for our paper, concordant with what was observed for most of the variables. We therefore feel confident that the smaller sample size of Black men in our study did not compromise our results. However, we are aware that our observations need to be validated in larger samples of subjects. All analyses were conducted with the SAS statistical package (SAS Institute, Cary, NC).

3. Results

Baseline and follow-up physical characteristics of White and Black men are shown in Table 1. With the exception of abdominal visceral AT accumulation which was greater in Whites than in Blacks ($P < 0.0005$), there was no difference in baseline body fatness and AT distribution. The 20-week exercise training program was associated with an increase in cardiorespiratory fitness, maximal oxygen consumption (V_{O_2max})

Table 1
Physical characteristics of White and Black men before and following a 20-week endurance exercise training program[a]

Variables	Whites		Blacks	
	Baseline	Follow-up	Baseline	Follow-up
Number of subjects	200	–	69	–
Age (yr)	36 ± 15	–	33 ± 11	–
Weight (kg)	83.6 ± 15.1	83.2 ± 14.9*	83.4 ± 16.5	82.8 ± 16.0*
Body-mass index (kg/m^2)	26.5 ± 4.5	26.3 ± 4.5*	26.9 ± 4.8	26.7 ± 4.7*
%Body fat	22.8 ± 8.8	21.9 ± 8.8*	22.9 ± 7.1	22.0 ± 7.1*
Fat mass (kg)	20.0 ± 10.5	19.2 ± 10.4*	19.9 ± 9.5	19.0 ± 9.2*
Fat-free mass (kg)	63.5 ± 7.7	64.0 ± 7.7*	63.5 ± 8.6	63.7 ± 8.4
Waist circumference (cm)	94.0 ± 13.0	93.1 ± 13.0*	91.2 ± 13.8	89.9 ± 13.4*
Waist/hip ratio	0.912 ± 0.07	0.908 ± 0.07*	0.892 ± 0.08	0.885 ± 0.08*
Abdominal adipose tissue areas (cm^2)				
Visceral	106 ± 60	99 ± 57*	75 ± 56	68 ± 49*
Subcutaneous	225 ± 127	215 ± 125*	219 ± 148	210 ± 142*
V_{O_2max} (l/min)	3.04 ± 0.59	3.50 ± 0.64*	2.75 ± 0.46	3.19 ± 0.49*

[a] Values are mean ± S.D.
* Significantly different from baseline, P at least <0.05.

Table 2
Plasma lipoprotein profile and post-heparin plasma lipase activities of White and Black men before and following a 20-week endurance exercise training program[a]

Variables	Whites		Blacks	
	Baseline	Follow-up	Bascline	Follow-up
Number of subjects	200		69	
Cholesterol (mmol/l)	4.58 ± 1.03	4.48 ± 1.05*	4.47 ± 0.97	4.46 ± 1.00
Triglycerides (mmol/l)	1.56 ± 0.93	1.39 ± 0.90*	1.26 ± 0.83	1.19 ± 0.69
LDL cholesterol (mmol/l)	3.08 ± 0.88	3.04 ± 0.92	3.00 ± 0.83	3.00 ± 0.85
HDL cholesterol (mmol/l)	0.94 ± 0.20	0.96 ± 0.22*	1.02 ± 0.38	1.03 ± 0.36
HDL_2 cholesterol (mmol/l)	0.27 ± 0.11	0.27 ± 0.13	0.30 ± 0.26	0.34 ± 0.25*
HDL_3 cholesterol (mmol/l)	0.67 ± 0.12	0.69 ± 0.14*	0.72 ± 0.15	0.70 ± 0.14
Apolipoprotein B (g/l)	0.90 ± 0.25	0.88 ± 0.26*	0.84 ± 0.24	0.86 ± 0.25
Total/HDL cholesterol	5.12 ± 1.61	4.86 ± 1.49*	4.73 ± 1.67	4.57 ± 1.43**
Post-heparin plasma lipase activity (nmol/ml/min)				
Lipoprotein Lipase	47.8 ± 26.1	59.6 ± 25.0*	67.9 ± 31.4	73.7 ± 25.9
Hepatic lipase	245.2 ± 61.4	230.6 ± 59.5*	182.3 ± 65.8	166.4 ± 60.6*
HL/LPL	9.16 ± 14.15	5.34 ± 5.86*	4.00 ± 4.81	2.74 ± 1.78*

[a] Values are mean ± S.D.
* Significantly different from baseline, P at least <0.05.
** $P<0.0544$ vs. baseline.

being significantly higher at follow-up in both groups. The training program was also associated with significant changes in body composition and AT distribution indices as body fat mass, waist circumference, plus visceral and subcutaneous AT accumulations were all significantly reduced after the training period in both White and Black men.

The fasting plasma lipoprotein–lipid profile was improved by exercise training in both groups of men (Table 2). However, most of the changes that reached statistical significance were noted in White subjects, e.g. decreases in plasma cholesterol, TG, apo B levels and in the total/HDL cholesterol ratio. White men also had a significant increase in HDL cholesterol concentrations, which was largely due to the elevation of HDL_3 cholesterol levels. On the other hand, Black men had a significant increase only in HDL_2 cholesterol level over the training period.

Fig. 1 illustrates the absolute changes in physical and metabolic characteristics following the exercise training program in both White and Black men. Significant differences between the two groups were noted in the cholesterol contents of the HDL_2 ($P < 0.05$) and HDL_3 ($P < 0.05$) subfractions as well as in apo B levels ($P < 0.005$). Indeed, Black men were characterized by a greater increase in HDL_2 and decrease in HDL_3 cholesterol concentrations over the 20-week period compared to Whites. Changes in post-heparin plasma lipase activities following training were also noted (Table 2); in both ethnic groups, PH-HL activity was significantly lower after 20 weeks of endurance exercise training. However, the increase in PH-LPL activity reached statistical significance only in White men. These changes observed over the 20-week exercise training program resulted in significant decreases in the PH-HL/PH-LPL ratio in both subgroups. When the changes in PH-LPL and PH-HL activities of White and Black men after training were compared, there was no significant

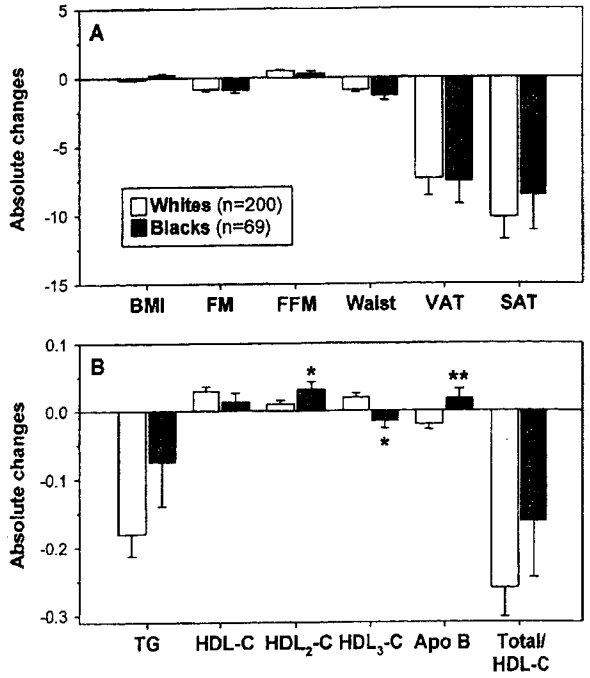

Fig. 1. Absolute changes in body fatness and AT distribution (panel A) as well as lipoprotein–lipid concentrations (panel B) in White (white bars) and Black (black bars) men following the 20-week endurance exercise training program. (*, **) change significantly different from White men (within x between-race interaction), $P < 0.05$ and 0.005, respectively.

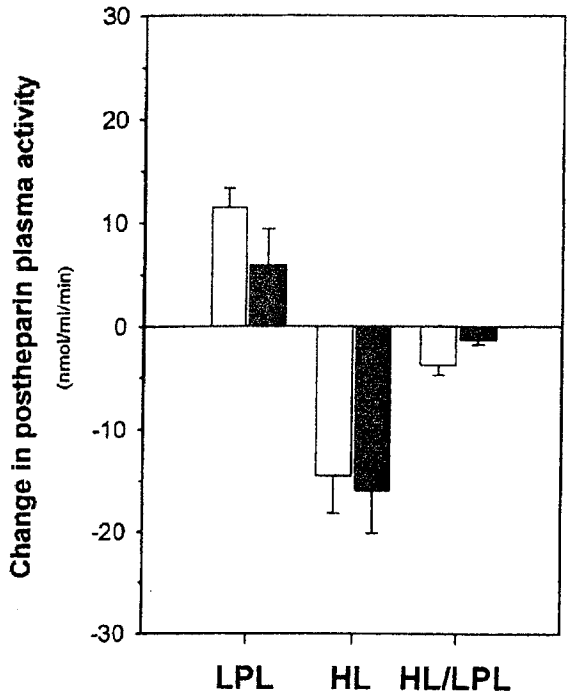

Fig. 2. Absolute changes in post-heparin LPL and HL activities in White (white bars) and Black (black bars) men following the 20-week endurance exercise training program.

difference between the two groups (Fig. 2). However, White subjects showed a greater reduction of the PH-HL/PH-LPL ratio compared to Black individuals. Statistical significance of relative changes in physical and metabolic variables was also tested and essentially similar results were obtained (data not shown).

Table 3 shows the relationships between the changes in post-heparin plasma lipase activities, body fatness, AT distribution and the lipoprotein–lipid profile in both groups of men. We found that the change in PH-LPL activity following exercise training was more closely related to the changes in adiposity and metabolic variables than was the decrease in PH-HL activity which showed no association with the same set of variables. For instance, the decrease in abdominal AT, either expressed as the change in waist circumference, subcutaneous AT (in Whites) or visceral AT (in Whites and Blacks), was associated with the elevation of PH-LPL activity. Increased PH-LPL activity after training was also related to the reduction of TG and increase in HDL cholesterol concentrations (Fig. 3) in both White and Black men. Overall, the correlations between the changes in PH-LPL, body fatness, AT distribution and lipoprotein–lipid levels in both groups of men were relatively low ($r \leqslant 0.30$).

4. Discussion

The extensive data collection of the HERITAGE Family Study offered unique opportunities to examine metabolic adaptations to training. In the present study, we have compared the effects of a 20-week endurance exercise training program on lipoprotein levels, as well as on PH-LPL and PH-HL activities in White and Black men.

Body composition changes were noted in both White and Black men training, as body fatness decreased. Abdominal AT accumulation was also reduced in both groups of men. Despite these changes, White subjects remained characterized by higher abdominal AT accu-

Table 3
Associations between changes in post-heparin plasma lipase activities, body composition, fat distribution and lipoprotein–lipid levels to a 20-week endurance exercise training program in White and Black men[a]

Variables	Whites			Blacks		
	PH-LPL	PH-HL	PH-HL/PH-LPL	PH-LPL	PH-HL	PH-HL/PH-LPL
Body composition and fat distribution						
Fat mass	−0.10	0.00	0.08	−0.20	0.05	0.16
Waist circumference	−0.18*	−0.04	0.16*	−0.14	0.08	0.12
Visceral AT	−0.17*	0.00	0.12	−0.26*	−0.09	0.19
Subcutaneous AT	−0.23***	−0.12	0.15*	−0.13	0.17	0.19
Lipoprotein–lipids						
Triglycerides	−0.20***	−0.14	0.12	−0.30*	−0.04	0.23****
HDL cholesterol	0.21***	−0.03	0.15*	0.26*	0.04	−0.10
HDL_2 cholesterol	−0.02	−0.02	0.03	0.12	0.02	−0.05
HDL_3 cholesterol	0.23***	−0.01	−0.17*	0.18	0.03	−0.03
Apolipoprotein B	0.07	−0.08	−0.12	−0.09	0.15	0.17
Total/HDL cholesterol	−0.06	−0.07	0.00	−0.23****	0.13	−0.19
V_{O_2max}	−0.02	−0.02	−0.05	0.03	−0.11	−0.28*

[a] Triglyceride values are log transformed.
* $P<0.05$; ** $P<0.01$; *** $P<0.005$; **** $P=0.06$.

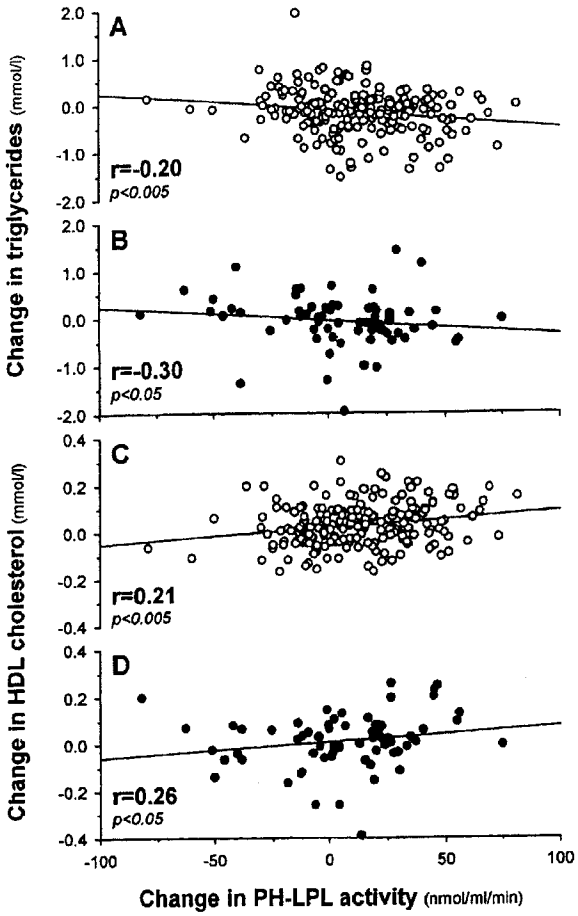

Fig. 3. Associations between the changes in PH-LPL activity and plasma TG (panels A and B) as well as HDL cholesterol concentrations (panels C and D) in White (white circles) and Black (black circles) men following the 20-week endurance exercise training program.

mulation compared to Blacks. However, the response of body fatness and of AT distribution indices were not different between White and Black men, suggesting that weight loss and AT distribution changes in response to exercise training did not seem to be influenced by ethnicity. Although data used for the present study came from a subsample of the HERITAGE subjects, similar body composition and AT distribution changes in response to training were found in the complete cohort [29].

The exercise training program was associated with changes in the lipoprotein–lipid profile but most of these changes were noted in Whites. At follow-up, plasma TG were decreased and HDL cholesterol concentrations increased in White men. Our observations are concordant with those of previous studies [5–11]. On the other hand, plasma lipid concentrations of Black individuals did not appear to be affected by the training program. In fact, the only significant change noted in Blacks was a slight increase in HDL_2 cholesterol levels. As already shown [24], Black men generally have a more favorable fasting lipoprotein–lipid profile than Whites. Therefore, in the present study, the better lipoprotein–lipid profile of Blacks at baseline (e.g. lower TG and apo B levels, plus higher HDL cholesterol concentrations) may have limited the beneficial impact of exercise training on lipoprotein–lipid levels in these individuals.

Within each group of men, PH-LPL activity was significantly increased after 20 weeks of exercise. Our observations are in accordance with those from previous reports showing that PH-LPL activity is higher in endurance-trained individuals [19,20]. However, we found no difference in the changes in PH-LPL or PH-HL activity between White and Black men.

In both groups of men, but mostly in Whites, the changes in PH-LPL activity were significantly correlated with changes in TG and HDL cholesterol levels. However, no significant correlations were noted between changes in PH-HL and lipid concentrations in either Whites or Blacks. As LPL is more closely related to TG degradation for either energy (muscle) or storage (AT), HL plays a more important role in lipoprotein remodeling, especially of HDL particles. Therefore, it seems most likely that the changes in energy needs that are related to exercise may have had a greater impact on LPL activity than on HL activity. In our study, the increase in PH-LPL activity was associated with a decrease in plasma TG and an increase in HDL cholesterol concentrations in both groups of men, the latter observation presumably the result of increased TG-rich lipoprotein lipolysis, as reported in previous studies [41–44]. Furthermore, our study does not suggest that PH-HL activity is strongly affected by physical activity.

We found that the change in PH-LPL activity was associated with the reduction of abdominal visceral AT accumulation. As visceral obesity is also associated with alterations in lipoprotein–lipid concentrations [45–49], the reduction in the visceral fat depot may have been a confounding factor in the relationships between changes in PH-LPL activity and the metabolic profile over the 20-week period. In the present study, statistical adjustment for the change in visceral AT did not affect the associations between the changes in PH-LPL activity and TG or HDL cholesterol concentrations in White and Black men (data not shown). Thus, it appears that the processes by which LPL influences plasma lipoprotein lipid levels may be independent of the decrease in visceral AT. In support of this observation, it has been suggested that PH-LPL activity measured shortly after heparin infusion reflects LPL derived from skeletal muscle rather than from AT [50]. In addition, LPL activity has been shown to be increased in skeletal muscle [51–54] and reduced in AT

following endurance exercise training [54]. Therefore, the increase in LPL activity noted in the present study is likely to be independent of the decrease in adiposity during the training program. On the other hand, improvement of insulin sensitivity associated with the reduction of visceral AT accumulation needs to be considered as a possible explanation for the higher LPL activity following training. Unfortunately, we were not able to verify this hypothesis in the present study.

In summary, our results show that regular endurance exercise training is associated with decreases in body fat and abdominal AT accumulation, as well as in changes in plasma lipoprotein–lipid concentrations, i.e. a reduction in plasma TG and an increase in HDL cholesterol levels. Higher PH-LPL activity following exercise training accounts for some of the improvements in the metabolic profile, but the rather weak associations between the changes in PH-LPL activity and lipoprotein–lipid concentrations suggest that other mechanisms are responsible for the better metabolic profile observed among physically trained individuals. Further studies are clearly warranted to elucidate these physiological processes.

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References

[1] Lamarche B, Després JP, Pouliot MC, et al. Is body fat loss a determinant factor in the improvement of carbohydrate and lipid metabolism following aerobic exercise training in obese women? Metabolism 1992;41:1249–56.

[2] Björntorp P, De Jounge K, Sjöstrom L, Sullivan L. The effect of physical training on insulin production in obesity. Metabolism 1970;19:631–8.

[3] Krotkiewski M, Björntorp P. Muscle tissue in obesity with different distribution of adipose tissue. Effects of physical training. Int J Obes 1986;10:331–41.

[4] Oshida Y, Yamanouchi K, Hayamizu S, Sato Y. Long-term mild jogging increases insulin action despite no influence on body mass index or V_{O_2max}. J Appl Physiol 1989;66:2206–10.

[5] Wood PD, Haskell W, Klein H, Lewis S, Stern MP, Farquhar JW. The distribution of plasma lipoproteins in middle-aged male runners. Metabolism 1976;25:1249–57.

[6] Krauss RM. Exercise, lipoproteins, and coronary artery disease. Circulation 1989;79:1143–5.

[7] Raz I, Rosenblit H, Kark JD. Effect of moderate exercise on serum lipids in young men with low high density lipoprotein cholesterol. Arteriosclerosis 1988;8:245–51.

[8] Després JP, Pouliot MC, Moorjani S, et al. Loss of abdominal fat and metabolic response to exercise training in obese women. Am J Physiol 1991;261:E159–67.

[9] Durstine JL, Haskell WL. Effects of exercise training on plasma lipids and lipoproteins. Exerc Sport Sci Rev 1994;22:477–521.

[10] Stefanick ML, Wood PD. Physical activity, lipid and lipoprotein metabolism, and lipid transport. In: Bouchard C, Shephard RJ, Stephens R, editors. Physical activity, fitness, and health. International proceedings consensus statement. Champaign, IL: Human Kinetics, 1994.

[11] Hardman AE. Physical activity, obesity and blood lipids. Int J Obes 1999;23(Suppl 3):S64–71.

[12] Paffenbarger RS, Hale WE. Work activity and coronary heart mortality. N Engl J Med 1975;292:545–50.

[13] Paffenbarger RS Jr., Hyde RT, Wing AL, Steinmetz CH. A natural history of athleticism and cardiovascular health. J Am Med Assoc 1984;252:491–5.

[14] Leon AS, Connett J, Jacobs DR Jr., Rauramaa R. Leisure-time physical activity levels and risk of coronary heart disease and death. The multiple risk factor intervention trial. J Am Med Assoc 1987;258:2388–95.

[15] Blair SN, Kohl HWD, Paffenbarger RS Jr., Clark DG, Cooper KH, Gibbons LW. Physical fitness and all-cause mortality. A prospective study of healthy men and women. J Am Med Assoc 1989;262:2395–401.

[16] Wood PD, Stefanick ML. Fitness and atherosclerosis. In: Bouchard C, Shephard RJ, Stephens T, Sutton JR, McPherson BD, editors. Exercise, fitness and health. A consensus of current knowledge. Champaign. IL: Human Kinetics, 1988:409–20.

[17] Powell KE, Thompson PD, Caspersen CJ, Kendrick JS. Physical activity and the incidence of coronary heart disease. Ann Rev Publ Health 1987;8:253–87.

[18] Folsom AR, Arnett DK, Hutchinson RG, Liao F, Clegg LX, Cooper LS. Physical activity and incidence of coronary heart disease in middle-aged women and men. Med Sci Sports Exerc 1997;29:901–9.

[19] Podl TR, Zmuda JM, Yurgalevitch SM, et al. Lipoprotein lipase activity and plasma triglyceride clearance are elevated in endurance-trained women. Metabolism 1994;43:808–13.

[20] Kantor MA, Cullinane EM, Sady SP, Herbert PN, Thompson PD. Exercise acutely increases high density lipoprotein-cholesterol and lipoprotein lipase activity in trained and untrained men. Metabolism 1987;36:188–92.

[21] Lovejoy JC, de la Bretonne JA, Klemperer M, Tulley R. Abdominal fat distribution and metabolic risk factors: effects of race. Metabolism 1996;45:1119–24.

[22] Albu JB, Murphy L, Frager DH, Johnson JA, Pi-Sunyer FX. Visceral fat and race-dependent health risks in obese nondiabetic premenopausal women. Diabetes 1997;46:456–62.

[23] Schmidt MI, Duncan BB, Watson RL, Sharrett AR, Brancati FL, Heiss G. A metabolic syndrome in Whites and African-Americans. The atherosclerosis risk in communities baseline study. Diabetes Care 1996;19:414–8.

[24] Després JP, Couillard C, Gagnon J, et al. Race, visceral adipose tissue, plasma lipids and lipoprotein lipase activity in men and women – the HERITAGE Family Study. Arterioscler Thromb Vasc Biol 2000;20:1932–8.

[25] Bouchard C, Leon AS, Rao DC, Skinner JS, Wilmore JH, Gagnon J. The HERITAGE Family Study. Aims, design, and measurement protocol. Med Sci Sports Exerc 1995;27:721–9.

[26] The Airlie (VA) consensus conference-Standardization of anthropometric measurements. In: Lohman T, Roche A, Martorel R, editors. Champaign, IL: Human Kinetics; 1988:p. 20–37.
[27] Behnke AR, Wilmore JH. Evaluation and regulation of body build and composition. Englewood Cliffs, CA: Prentice-Hall, 1974:20–37.
[28] Wilmore JH, Stanforth PR, Domenick MA, et al. Reproducibility of anthropometric and body composition measurements: the HERITAGE Family Study. Int J Obes 1997;21:297–303.
[29] Wilmore JH, Després JP, Stanforth PR, et al. Alterations in body weight and composition consequent to 20 week of endurance training: the HERITAGE Family Study. Am J Clin Nutr 1999;70:346–52.
[30] Després JP, Prud'homme D, Pouliot MC, Tremblay A, Bouchard C. Estimation of deep abdominal adipose-tissue accumulation from simple anthropometric measurements in men. Am J Clin Nutr 1991;54:471–7.
[31] Ferland M, Després JP, Tremblay A, et al. Assessment of adipose tissue distribution by computed axial tomography in obese women: association with body density and anthropometric measurements. Br J Nutr 1989;61:139–48.
[32] Kvist H, Sjöstrom L, Tylen U. Adipose tissue volume determinations in women by computed tomography: technical considerations. Int J Obes 1986;10:53–67.
[33] Moorjani S, Dupont A, Labrie F, et al. Increase in plasma high-density lipoprotein concentration following complete androgen blockage in men with prostatic carcinoma. Metabolism 1987;36:244–50.
[34] Burstein M, Samaille J. Sur un dosage rapide du cholesterol lié aux α- et β-lipoprotéines du sérum. Clin Chim Acta 1960;5:609.
[35] Laurell CB. Quantitative estimation of proteins by electrophoresis in agarose gel containing antibodies. Anal Biochem 1966;15:45–52.
[36] Avogaro P, Bon GB, Cazzolato G, Quinci GB. Are apolipoproteins better discriminators than lipids for atherosclerosis? Lancet 1979;1:901–3.
[37] Gidez LI, Miller GJ, Burstein M, Slagle S, Eder HA. Separation and quantitation of subclasses of human plasma high density lipoproteins by a simple precipitation procedure. J Lipid Res 1982;23:1206–23.
[38] Després JP, Gagnon J, Bergeron J, et al. Plasma post-heparin lipase activities in the HERITAGE Family Study – the reproducibility, gender differences and associations with lipoprotein levels. Clin Biochem 1999;32:157–65.
[39] Nilsson-Ehle P, Ekman R. Specific assays for lipoprotein lipase and hepatic lipase activities of post-heparin plasma. In: Peeters H, editor. Protides of biological fluids. Oxford: Permagon Press, 1978:243–6.
[40] St-Amand J, Moorjani S, Lupien PJ, Prud'homme D, Després JP. The relation of plasma triglyceride, apolipoprotein B, and high-density lipoprotein cholesterol to post-heparin lipoprotein lipase activity is dependent on apolipoprotien E polymorphism. Metabolism 1996;45:261–7.
[41] Aldred HE, Perry IC, Hardman AE. The effect of a single bout of brisk walking on postprandial lipemia in normolipidemic young adults. Metabolism 1994;43:836–41.
[42] Aldred HE, Hardman AE, Taylor S. Influence of 12 weeks of training by brisk walking on postprandial lipemia and insulinemia in sedentary middle-aged women. Metabolism 1995;44:390–7.
[43] Ziogas GG, Thomas TR, Harris WS. Exercise training, postprandial hypertriglyceridemia and LDL subfraction distribution. Med Sci Sports Exerc 1997;29:986–91.
[44] Weintraub MS, Rosen Y, Otto R, Eisenberg S, Breslow JL. Physical exercise conditioning in the absence of weight loss reduces fasting and postprandial triglyceride-rich lipoprotein levels. Circulation 1989;79:1007–14.
[45] Reaven GM. Role of insulin resistance in human disease. Diabetes 1988;37:1595–607.
[46] Després JP, Nadeau A, Tremblay A, et al. Role of deep abdominal fat in the association between regional adipose tissue distribution and glucose tolerance in women. Diabetes 1989;38:304–9.
[47] Després JP, Moorjani S, Ferland M, et al. Adipose tissue distribution and plasma lipoprotein levels in obese women. Importance of intra-abdominal fat. Arteriosclerosis 1989;9:203–10.
[48] Kissebah AH, Krakower GR. Regional adiposity and morbidity. Physiol Rev 1994;74:761–811.
[49] Lemieux S, Després JP. Metabolic complications of visceral obesity: contribution to the etiology of type II diabetes and implications for prevention and treatment. Diabete Metab 1994;20:375–93.
[50] Brunzell JD, Chait A, Nikkila EA, Ehnholm C, Huttunen JK, Steiner G. Heterogeneity of primary lipoprotein lipase deficiency. Metabolism 1980;29:624–9.
[51] Nikkilä EA, Taskinen MR, Rehunen S, Härkönen M. Lipoprotein lipase activity in adipose tissue and skeletal muscle of runners: relation to serum lipoproteins. Metabolism 1978;27:1661–7.
[52] Svedenhag J, Lithell H, Juhlin-Dannfelt A, Henriksson J. Increase in skeletal muscle lipoprotein lipase following endurance training in man. Atherosclerosis 1983;49:203–7.
[53] Kiens B, Lithell H. Lipoprotein metabolism influenced by training-induced changes in human skeletal muscle. J Clin Invest 1989;83:558–64.
[54] Lamarche B, Després JP, Moorjani S, et al. Evidence for a role of insulin in the regulation of abdominal adipose tissue lipoprotein lipase response to exercise training in obese women. Int J Obes 1993;17:255–61.