Race and Sex Similarities in Exercise-Induced Changes in Blood Lipids and Fatness

CHRIS I. ARDERN[1], PETER T. KATZMARZYK[1,2], IAN JANSSEN[1,2], ARTHUR S. LEON[3], JACK H. WILMORE[4], JAMES S. SKINNER[5], DABEERU C. RAO[6], JEAN-PIERRE DESPRÉS[7], TUOMO RANKINEN[8], and CLAUDE BOUCHARD[8]

[1]School of Physical and Health Education, [2]Department of Community Health and Epidemiology, Queen's University, Kingston, ON, CANADA; [3]Division of Kinesiology, University of Minnesota, Minneapolis, MN; [4]Department of Health and Kinesiology, Texas A&M University, College Station, TX; [5]Department of Kinesiology, Indiana University, Bloomington, IN; [6]Division of Biostatistics and Departments of Psychiatry and Genetics, Washington University School of Medicine, St. Louis, MO; [7]Department of Food Sciences and Nutrition, Québec Heart Institute, and Lipid Research Center, Laval University Medical Research Center, Sainte-Foy, QC, CANADA; and [8]Human Genomics Laboratory, Pennington Biomedical Research Center, Baton Rouge, LA

ABSTRACT

ARDERN, C. I., P. T. KATZMARZYK, I. JANSSEN, A. S. LEON, J. H. WILMORE, J. S. SKINNER, D. C. RAO, J.-P. DESPRÉS, T. RANKINEN, and C. BOUCHARD. Race and Sex Similarities in Exercise-Induced Changes in Blood Lipids and Fatness. *Med. Sci. Sports Exerc.*, Vol. 36, No. 9, pp. 1610–1615, 2004. **Purpose:** This study explores sex and race differences in the association between changes in fat mass (FM), abdominal visceral fat (AVF), and abdominal subcutaneous fat (ASF) on blood lipid changes consequent to aerobic exercise training. **Methods:** The sample included 613 participants (428 white and 185 black, 46% men) from the HERITAGE Family Study. Total FM was determined by densitometry, whereas AVF and ASF cross-sectional areas were determined by computed tomography at the L4–L5 level. Blood lipid measurements included total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), triglycerides (TG), and the TC/HDL-C ratio, which were obtained before and after 20 wk of supervised aerobic exercise. Canonical correlation was used to determine the multivariate associations between body fatness and blood lipids at baseline and the changes induced by exercise training. **Results:** Body fat accounted for 26–36% of the variance in baseline blood lipids, and changes in body fat accounted for 7–21% of the variance in changes in blood lipids with exercise training. The pattern of loadings indicated similar relationships between body fatness and blood lipids at baseline, and their respective changes with exercise training among the four sex-by-race groups. Greater fat loss, characterized by loss of FM, AVF, and ASF, was associated with a greater blood lipid response characterized by an increase in HDL-C and decreases in LDL-C, TG, TC, and TC/HDL-C. Although the pattern of loadings was similar in all groups, the strength of the association was stronger in blacks than in whites. **Conclusion:** The multivariate associations among fat loss and changes in blood lipids consequent to aerobic exercise training are similar in black and white men and women. **Key Words:** ETHNICITY, EXERCISE TRAINING, LIPOPROTEINS, ADIPOSE TISSUE, CANONICAL CORRELATION, AEROBIC FITNESS

A hallmark adaptation of chronic physical activity is cardiovascular disease risk reduction. Intervention studies in populations at mild-to-moderate coronary risk have demonstrated improvements in blood lipid profiles with standardized exercise training (14,17). To date, the mechanisms behind these improvements remain unresolved, and the independent contribution of physiological training effects on the cardiovascular system *per se* versus exercise-induced alterations in body composition remain a topic worthy of further exploration (27).

The results of a recent meta-analysis suggest that improvements in blood lipids consequent to exercise training may be dependent upon loss of body weight (16). For example, a recent analysis from the HERITAGE Family study found no association between changes in maximal oxygen consumption ($\dot{V}O_{2max}$) and changes in blood lipids consequent to exercise training; however, there was a significant relationship with changes in fat mass (FM) that was independent of sex and race (13). The metabolic effects of chronic exercise training may be due to loss of total fat (6,13) or abdominal visceral fat (AVF) (6).

Evidence from cross-sectional studies indicates that total FM, abdominal subcutaneous fat (ASF), and AVF demonstrate significant sex (12) and racial (black, white) (11) dimorphism. Further, at baseline, patterns of blood lipids differ between blacks and whites (17). To the same extent, sex differences in adipose tissue metabolism are well described (2), and are potentially explained by higher hepatic lipase activity in men (4). Given the increasing prevalence of obesity in the United States (7), and the relationship between physical activity and blood lipids (21), the inter-

Address for correspondence: Peter T. Katzmarzyk, School of Physical and Health Education, Queen's University, Kingston, ON K7L 3N6, Canada; E-mail: katzmarz@post.queensu.ca.
Submitted for publication January 2004.
Accepted for publication May 2004.

0195-9131/04/3609-1610
MEDICINE & SCIENCE IN SPORTS & EXERCISE®

DOI: 10.1249/01.MSS.0000139798.54405.AF

action between blood lipids, total and regional fat, and response to exercise training warrants further study.

Because cardiovascular disease risk is not easily captured by a single risk factor in isolation, the examination of multivariate associations between body fatness and blood lipids may lead to an improved understanding of race and sex differences in the cardiovascular risk reduction observed with exercise training. Therefore, the intent of this study was to determine the multivariate relationships between FM and specific fat depots (ASF and AVF) with plasma lipids at baseline and in response to 20 wk of aerobic exercise training, and to explore race and sex differences in the associations.

METHODS

Participants. The HERITAGE Family Study was designed to investigate the genetics of cardiovascular and metabolic risk factors, and subsequent responses to a 20-wk program of standardized aerobic exercise training. The present sample includes 613 participants (428 white and 185 black) who completed 20 wk of aerobic exercise training and had the required measurements before and after training. All participants were screened for baseline physical activity levels and were included if they were *sedentary*, defined as less than 30 min·wk^{-1} of high intensity physical activity (<50 yr: 7–8 METs; ≥50 yr: 5–6 METs) over the previous 3 months.

Participating institutions included four clinical centers [Arizona State University (now Indiana University), Laval University (now Pennington Biomedical Research Center), University of Minnesota, and The University of Texas at Austin (now Texas A&M University)] and a data coordinating center at Washington University in St. Louis. All protocols were approved by all participating institutions' ethics review boards for studies involving human subjects, and written informed consent was obtained from all participants.

Study personnel were centrally trained in all aspects of recruitment and measurement. The HERITAGE Family Study employed a rigorous program of data quality and assurance, including repeat assays on 5% of blood samples and analyses of split samples prepared at each clinical center (10). Reliability studies have found good consistency among centers and among individual technicians for all clinical measurements (5). Participants were excluded from the study if at screening they had a body mass index over 40 kg·m^{-2}, if they had elevated blood pressure (≥160/100 mm Hg), potential cardiovascular pathology or respiratory distress, metabolic disorders, or if they were currently using blood pressure, blood glucose, or blood lipid-lowering medication.

Blood lipid measurements. Blood samples were drawn from an antecubital vein in the morning after a 12-h fast and collected into Vacutainer tubes with EDTA. Samples in women were collected during the early follicular phase of the menstrual cycle. Plasma concentrations of total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), and triglycerides (TG) were assayed. Plasma was ultracentrifuged and the top fraction containing VLDL was quantitatively recovered. The LDL in the ultracentrifuged bottom fraction was precipitated with heparin and $MgCl_2$ (3,19) and the HDL was obtained in the supernatant. The concentrations of cholesterol (1) and total TG (8) were measured by autoanalyzer (Technicon RA-500).

For the purpose of this study, baseline blood lipid measurements (PRE) were reported as the average of measurements on two separate days, and posttraining (POST) as the average of 24- and 72-h measurements after the last exercise bout, given that there were minimal differences in the 24- and 72-h posttraining plasma lipid measurements (17). Corrections for potential training-induced plasma volume changes were made using the Biuret method (Roche Molecular Biochemicals, Dallas, TX) by adjusting the POST specimens for the difference between PRE and POST plasma total protein levels (17).

Body composition measures. Height was measured using a stadiometer while barefoot and standing on a flat surface, and weight was measured with a leveled platform scale. Body mass index (BMI) was subsequently calculated as weight (kg) divided by squared height (m^2), and waist circumference (WC; cm) was measured with an inelastic tape at the narrowest waist. Computed tomography (CT) scans at the intervertebral disk level of the fourth and fifth lumbar vertebrae (L4–L5) were used to measure the cross-sectional area (cm^2) of ASF and AVF. All scans were analyzed according to the method of Sjöström and colleagues (23). Hydrostatic weighing was used to measure FM in a postabsorptive state using a standard protocol, with correction for residual lung volume by the oxygen dilution technique (29) in three of the centers, and by the helium dilution technique (20) at Laval University.

Aerobic exercise training program. Participants exercised for 20 wk on a cycle ergometer, 3× wk^{-1} for 30–50 min per session. Details on the training component of HERITAGE can be found elsewhere (24). Briefly, the power output of the cycle ergometer was automatically adjusted to the heart rate response of the participant during each of the training sessions. Participants started at 55% of their baseline $\dot{V}O_{2max}$ for 30 min per session and progressed in intensity or duration every 2 wk following a standardized protocol until they were working at 75% $\dot{V}O_{2max}$ for 50 min per session for the final 6 wk. Participants were counseled at baseline and midway (10 wk) through the exercise training program not to alter their usual health and lifestyle habits outside of the study, including diet and physical activity levels.

Statistical analyses. Participants were stratified into four sex-by-race groups for comparison, and all statistical analyses were performed using SAS procedures (SAS Institute; Cary, NC) (22). The distribution for TG was positively skewed and was normalized by a log transformation before further analysis. Associations between individual baseline blood lipid measurements and each of the three adiposity measures, as well as their changes, were assessed

with partial correlations, controlling for age. Canonical correlation was used to quantify the multivariate associations between baseline body fatness (ASF, AVF, and FM) and blood lipids (TC, HDL-C, LDL-C, TG, and TC/HDL-C), and among the change scores consequent to 20 wk of aerobic exercise training. Briefly, canonical correlation collapses clusters of related variables into composite indices, or canonical variates. The linear association between the two "sets" of variables is assessed in such a way as to maximize the correlation between the two canonical variates. The canonical correlation is the bivariate correlation between the two canonical variates, whereas the squared canonical correlation is an indication of the variance shared by the two canonical variates. Bivariate correlations between the original variables and their respective canonical variates are interpreted as loadings, which define the canonical variates. In other words, the loadings represent the contribution of each original variable to the canonical variate. Graphical presentations of loadings allow the visualization and interpretation of canonical variates. The blood lipid and body fatness measures used in the analysis were selected to provide the most clinically relevant blood lipid and body fatness profile with minimal overlap between component variables.

RESULTS

Table 1 presents the baseline characteristics of study participants, by sex and race. Black men had significantly lower AVF and higher TG levels than white men. Black women had significantly greater BMI, WC, FM, ASF, but lower TG and TC than white women.

Partial correlations, adjusted for age, indicate that the associations between baseline fatness and blood lipids were similar across the four race-by-sex groups (Table 2, top panel). However, the associations between changes in individual indicators of fatness and blood lipids, while similar in blacks and whites within the two sex groups, were generally stronger in women than in men within the two racial groups (Table 2, bottom panel).

TABLE 1. Baseline characteristics of HERITAGE participants.

	Men		Women	
	Black (N = 77)	White (N = 206)	Black (N = 108)	White (N = 222)
Age (yr)	33.6 ± 11.8	36.4 ± 15.0	32.2 ± 11.1	34.8 ± 13.9
BMI (kg·m^{-2})	27.1 ± 5.1	26.4 ± 4.6	27.9 ± 6.0	24.9 ± 13.9*
WC (cm)	91.9 ± 15.1	93.9 ± 12.9	90.4 ± 15.5	85.6 ± 14.7*
FM (kg)	20.4 ± 10.7	19.9 ± 10.5	27.5 ± 12.2	21.2 ± 11.2*
ASF (cm^2)	225 ± 163	223 ± 124	338 ± 172	283 ± 148*
AVF (cm^2)	76.4 ± 56	106 ± 92*	68.4 ± 43.0	74.6 ± 53.8
TC (mmol·L^{-1})	4.46 ± 0.9	4.57 ± 1.0	4.17 ± 0.8	4.45 ± 0.90*
HDL-C (mmol·L^{-1})	1.02 ± 0.4	0.94 ± 0.2	1.13 ± 0.3	1.16 ± 0.3
LDL-C (mmol·L^{-1})	2.99 ± 0.8	3.08 ± 0.9	2.79 ± 0.7	2.93 ± 0.79
TG (mmol·L^{-1})	1.27 ± 0.8	1.56 ± 0.9*	0.90 ± 0.5	1.19 ± 0.6*
TC/HDL-C	4.72 ± 1.7	5.10 ± 1.6	3.87 ± 1.1	4.01 ± 1.14

Values are means ± SD.
* Independent t-tests $P < 0.05$ for race differences, within sex.
BMI, body mass index; FM, total fat mass; ASF, abdominal subcutaneous fat; AVF, abdominal visceral fat; TC, total cholesterol; HDL-C, high-density lipoprotein-cholesterol; LDL-C, low-density lipoprotein-cholesterol; TG, triglycerides.

TABLE 2. Partial correlations (adjusted for age) between baseline and exercise-induced changes in blood lipids and adipose tissue.

	Black			White		
	Baseline					
	FM	ASF	AVF	FM	ASF	AVF
Men						
HDL-C	−0.31	−0.32	−0.35	−0.28	−0.28	−0.29
LDL-C	0.35	0.37	0.22	0.29	0.30	0.14
TC	0.36	0.41	0.24	0.30	0.32	0.18
TC/HDL-C	0.47	0.51	0.47	0.42	0.43	0.36
TG	0.45	0.49	0.45	0.39	0.40	0.33
Women						
HDL-C	−0.34	−0.34	−0.45	−0.31	−0.28	−0.37
LDL-C	0.28	0.26	0.30	0.24	0.24	0.32
TC	0.24	0.21	0.25	0.15	0.16	0.25
TC/HDL-C	0.43	0.40	0.53	0.37	0.36	0.49
TG	0.43	0.40	0.51	0.16	0.22	0.32
	Black			**White**		
	Changes					
	ΔFM	ΔASF	ΔAVF	ΔFM	ΔASF	ΔAVF
Men						
ΔHDL-C	−0.17	−0.18	−0.12	**−0.21**	**−0.26**	**−0.17**
ΔLDL-C	0.02	−0.02	0.13	0.07	0.01	−0.01
ΔTC	0.11	0.18	0.12	0.06	0.04	0.04
ΔTC/HDL-C	−0.06	−0.18	−0.12	−0.06	−0.04	−0.04
ΔTG	**0.24**	**0.38**	0.10	0.09	**0.17**	0.09
Women						
ΔHDL-C	−0.04	−0.08	−0.13	0.01	−0.03	0.05
ΔLDL-C	**0.32**	**0.40**	0.17	**0.18**	**0.15**	**0.21**
ΔTC	**0.33**	**0.38**	0.16	**0.18**	**0.15**	**0.19**
ΔTC/HDL-C	**−0.31**	**−0.33**	0.16	**−0.18**	**−0.16**	**−0.19**
ΔTG	**0.20**	0.17	0.15	0.03	0.10	−0.03

Bold indicates $P < 0.05$ for *Change* scores; all *Baseline* correlations are significant at the $P < 0.05$ level; HDL-C, high-density lipoprotein-cholesterol; LDL-C, low-density lipoprotein-cholesterol; TC, total cholesterol; TG, triglycerides.

Table 3 summarizes the results of the canonical correlation analyses for baseline values and for change scores consequent to 20 wk of exercise training. The strength of the multivariate associations between both baseline and changes in blood lipids and body fatness were similar in men and women within the two racial groups. However, there was a stronger relationship between both baseline and changes in blood lipids and body fatness in blacks compared with white men and women. Overall, between 26% and 36% of the variance in blood lipids at baseline was accounted for by body fatness, whereas only 7–21% of the variance in changes in blood lipids with training was accounted for by changes in body fatness.

Figure 1 presents the correlations between individual blood lipids and body fat measures and their respective canonical variates. The pattern of loadings was similar among races and sexes at baseline. A profile of low FM, ASF, and AVF was associated with lower LDL-C, TG, TC, TC/HDL-C, and higher HDL-C. In general, the variance in baseline blood lipids accounted for by baseline adipose stores was similar in men and women, but was higher in blacks than whites. In men, body fatness accounted for 34% and 26% of the variance in blood lipid profile in blacks and whites, respectively. In women, the associated variances were 36% and 26%, respectively.

Despite the baseline similarities, the multivariate relationship between changes in blood lipids and body fatness consequent to exercise training revealed a slightly different pattern (Fig. 2). The patterns of loadings were similar be-

TABLE 3. Results of canonical correlation analyses, controlling for age, between blood lipids and body fatness at baseline and changes induced by exercise training.

	r_c	r_c^2	EV	*F*	*P*
			Baseline		
Black men	0.59	0.34	0.53	2.90	<0.001
Black women	0.60	0.36	0.56	3.54	<0.001
White men	0.51	0.26	0.35	4.68	<0.001
White women	0.51	0.26	0.35	5.80	<0.001
			Changes		
Black men	0.46	0.21	0.26	1.63	0.07
Black women	0.46	0.21	0.26	2.77	<0.001
White men	0.32	0.10	0.12	1.95	0.02
White women	0.26	0.07	0.07	1.23	0.24

r_c, canonical correlation; r_c^2, squared canonical correlation; EV, eigenvalue.

tween black and white men consequent to exercise training; however, the strength of the relationship between individual factor contributions differed for individual components. In women, changes in HDL-C and TG contributed very little to the blood lipid variates compared with men, particularly in white women. The change in AVF was not as strongly associated with changes in lipids in black, by comparison with white (Fig. 2) men and women.

DISCUSSION

The purpose of this study was to extend earlier results from this cohort demonstrating that exercise-induced changes in blood lipids are associated with changes in fat mass more so than with changes in aerobic fitness *per se*

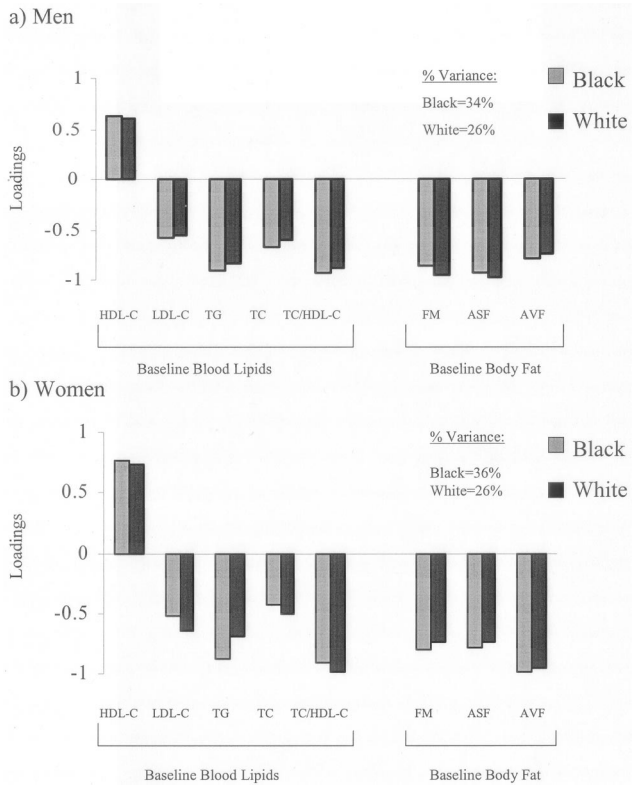

FIGURE 1—Loadings on first canonical variates for baseline blood lipid parameters and measures of body fatness in blacks and whites. The blood lipid variate is represented by HDL-C, TC, LDL-C, TG, and TC/HDL-C, and the body fatness variate by FM, ASF, and AVF.

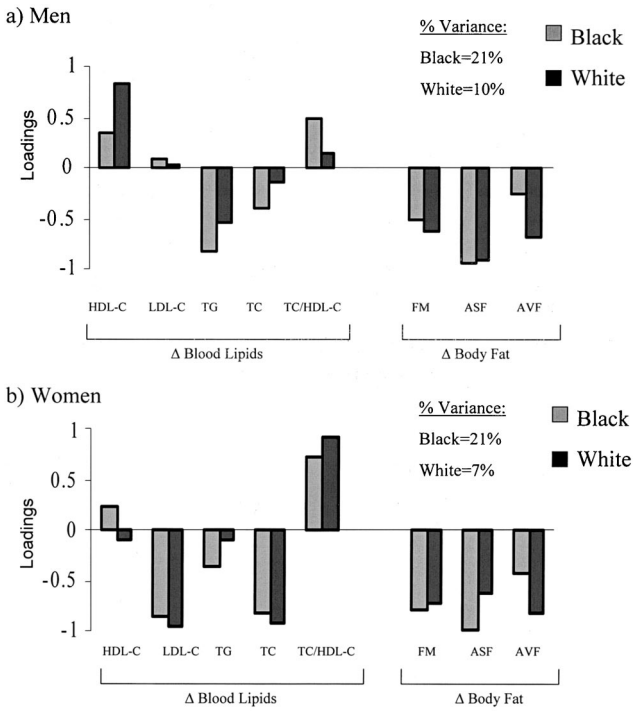

FIGURE 2—Loadings on first canonical variates for changes in blood lipids and changes in body fatness consequent to 20 wk of exercise training in blacks and whites. The blood lipid variate is represented by HDL-C, TC, LDL-C, TG, and TC/HDL-C, and the body fatness variate by FM, ASF, and AVF.

(13). The results from the HERITAGE Family Study are consistent with those of two randomized controlled trials comparing the blood lipid responses of dieters and exercisers (28,30). In the first study, there were significant correlations between changes in both body weight and fat mass and changes in HDL-C, HDL_2-C, and HDL_3-C in exercisers and dieters during a 1-yr intervention (30). In the second study, changes in BMI were significantly correlated with changes in HDL_2-C, small LDL, and LDL peak flotation rate in both dieters and exercisers (28).

To date, few studies have examined the independence of exercise training with and without weight loss on changes in blood lipids in a randomized controlled setting. One such study compared exercise-induced weight loss, exercise without weight loss, and weight loss without exercise over 12 wk (25). There were no independent effects of weight loss or exercise on TC, LDL-C, or TG. Exercise and weight loss separately and independently increased HDL-C, and their effects were additive. Only weight loss was found to have a significant effect on the TC/HDL-C ratio. Two earlier meta-analyses have also explored the independence of exercise and weight loss on cardiovascular risk factors (18,26). In the first, analysis of studies with participants that did not lose body weight in response to training demonstrated smaller reductions in LDL-C and TC in comparison with those in studies reporting exercise-induced weight loss (26). In the second meta-analysis, exercise weight stable studies demonstrated little change in TC, by comparison with an average reduction of 0.34 $mmol \cdot L^{-1}$ in those with exercise-induced weight loss in women (18). This study also

reported positive correlations between changes in body weight and changes in TC and TG (18).

The results of the current study indicate that total fat loss as well as fat loss in the ASF and AVF depots are all associated with improvements in the blood lipid profile as there appear to be no substantive differences in the relation of FM, ASF, and AVF and blood lipids by partial correlation (Table 2). However, a previous HERITAGE study that demonstrated a positive association between percent change in HDL-C and changes in body mass, FM, and AVF was marked by great heterogeneity in individual HDL-C response (15). It is also of interest that change in AVF was less of a contributor to the relationship between changes in blood lipids and body fatness in blacks compared with whites once FM and ASF were accounted for (Fig. 2); this highlights the importance of race in the current debate on the role of central obesity in the development of metabolic abnormalities (9), including the potential influence of race on plasma lipids and lipoproteins.

The tighter coupling between blood lipids and body fatness in blacks, both at baseline and in response to training, suggests that exercise programs that are designed to induce weight loss may be particularly effective at improving the average blood lipid profile in blacks. The exercise-training regimen of the HERITAGE Family Study was designed to increase cardiorespiratory fitness rather than to decrease body fat. The intervention was successful in accomplishing this, as the average increase in $\dot{V}O_{2max}$ was 17.5%, whereas the average decrease in FM was 3.3% (13). Although the mean change in FM was not large, there was great heterogeneity in response. The intent of this trial was not to induce weight loss; however, the improvement in the risk factor profile was associated with changes in body fat. By design, the HERITAGE Family Study did not include a control group, rather the subjects acted as their own controls. Given that changes in aerobic fitness with exercise training have been well documented, and that the intent of this analysis was to examine individual differences in the response to training, the lack of a control group should not be viewed as a weakness of this study. However, these associations should be examined further using controlled trials in which participants are randomized into exercise-weight stable and exercise-weight loss groups to determine the mechanisms whereby exercise improves the risk factor profile.

Taken together, the results of this study indicate that FM, ASF, and AVF all contribute to *baseline* levels of blood lipids and there are minimal sex or race differences in the associations. This reinforces the importance of considering both total and regional adiposity when assessing the health risks associated with obesity. Although there is considerable variability in the response to exercise, it is apparent that black and white men and women can all improve their blood lipid profile with exercise training. It is equally apparent that these improvements are somewhat dependent on the loss of body fat, particularly in the black participants. Thus, exercise training programs that encourage fat loss may be more beneficial for the improvement in blood lipids and the consequent reduction in risk of cardiovascular diseases.

The HERITAGE Family Study is supported by the National Heart, Lung, and Blood Institute through the following grants: HL45670 (C. Bouchard), HL47323 (A. S. Leon), HL47317 (D. C. Rao), HL47327 (J. S. Skinner), and HL47321 (J. H. Wilmore). It is also supported by a National Institutes of Health grant to the University of Minnesota Clinical Research Center. A. S. Leon is supported in part by the Henry L. Taylor Professorship in Exercise Science and Health Enhancement. C. Bouchard is partially supported by the George A. Bray Chair in Nutrition. J.-P. Després is Chair Professor of Nutrition and Lipidology supported by Pfizer, Provigo, and the foundation of the Québec Heart Institute. I. Janssen is supported by a Canadian Institutes of Health Research Postdoctoral Fellowship, and C. Ardern is supported by a Doctoral Research Award from the Heart and Stroke Foundation of Canada and the CIHR Institute of Gender and Health.

REFERENCES

1. Allain, C. C., L. S. Poon, C. S. Chan, W. Richmond, and P. C. Fu. Enzymatic determination of total serum cholesterol. *Clin. Chem.* 20:470–475, 1974.
2. Blaak, E. Gender differences in fat metabolism. *Curr. Opin. Clin. Nutr. Metab. Care* 4:499–502, 2001.
3. Burstein, M., H. R. Scholnick, and R. Morfin. Rapid method for the isolation of lipoproteins from human serum by precipitation with polyanions. *J. Lipid Res.* 11:583–595, 1970.
4. Després, J.-P., C. Couillard, J. Gagnon, et al. Race, visceral adipose tissue, plasma lipids, and lipoprotein lipase activity in men and women: the Health, Risk Factors, Exercise Training, and Genetics (HERITAGE) family study. *Arterioscler. Thromb. Vasc. Biol.* 20:1932–1938, 2000.
5. Després, J.-P., J. Gagnon, J. Bergeron, et al. Plasma post-heparin lipase activities in the HERITAGE Family Study: the reproducibility, gender differences, and associations with lipoprotein levels. HEalth, RIsk factors, exercise Training and GEnetics. *Clin. Biochem.* 32:157–165, 1999.
6. Després, J.-P., M.-C. Pouliot, S. Moorjani, et al. Loss of abdominal fat and metabolic response to exercise training in obese women. *Am. J. Physiol. Endocrinol. Metab.* 261:E159–E167, 1991.
7. Flegal, K. M., M. D. Carroll, C. L. Ogden, and C. L. Johnson. Prevalence and trends in obesity among US adults, 1999–2000. *JAMA* 288:1723–1727, 2002.
8. Fossati, P., and L. Prencipe. Serum triglycerides determined calorimetrically with an enzyme that produces hydrogen peroxide. *Clin. Chem.* 28:2077–2080, 1982.
9. Frayn, K. N. Visceral fat and insulin resistance: causative or relative? *Br. J. Nutr.* 83:S71–S77, 2000.
10. Gagnon, J., M. A. Province, C. Bouchard, et al. The HERITAGE Family Study: quality assurance and quality control. *Ann. Epidemiol.* 6:520–529, 1996.
11. Hill, J. O., S. Sidney, C. E. Lewis, K. Tolan, A. L. Scherzinger, and E. R. Stamm. Racial differences in amounts of visceral adipose tissue in young adults: the CARDIA (Coronary Artery Risk Development in Young Adults) Study. *Am. J. Clin. Nutr.* 69:381–387, 1999.
12. Janssen, I., and R. Ross. Effects of sex on the changes in visceral, subcutaneous adipose tissue and skeletal muscle in response to weight loss. *Int. J. Obes. Relat. Metab. Disord.* 23:1035–146, 1999.
13. Katzmarzyk, P. T., L. Pérusse, T. Rice, et al. Changes in blood lipids consequent to aerobic exercise training related to changes in body fatness and aerobic fitness. *Metabolism* 50:841–848, 2001.
14. Kraus, W. E., J. A. Houmard, B. D. Duscha, et al. Effects of the amount and intensity of exercise on plasma lipoproteins. *N. Engl. J. Med.* 347:1483–1492, 2002.
15. Leon, A. S., S. L. Gaskill, T. Rice, et al. Variability in the response of HDL-C to exercise training in the HERITAGE Family Study. *Int. J. Sports Med.* 29:1–9, 2002.

16. LEON, A. S., and O. A. SANCHEZ. Response of blood lipids to exercise training alone or combined with dietary intervention. *Med. Sci. Sports Exerc.* 33:S502–S515, 2001.
17. LEON, A. S., T. RICE, S. MANDEL, et al. Blood lipid response to 20 weeks of supervised exercise in a large biracial population: the HERITAGE Family Study. *Metabolism* 49:513–520, 2000.
18. LOKEY, E. A., and Z. V. TRAN. Effects of exercise training on serum lipid and lipoprotein concentrations in women: a meta-analysis. *Int. J. Sports Med.* 10:424–429, 1989.
19. LOPES-VIRELLA, M. F., P. STONE, S. ELLIS, and J. A. COLWELL. Cholesterol determination in high-density lipoproteins separated by three different methods. *Clin. Chem.* 23:882–884, 1977.
20. MENEELY, G. R., and N. L. KALTREIDER. The volume of the lung determined by helium dilution: description of the methods and comparison with other procedures. *J. Clin. Invest.* 28:129–139, 1949.
21. PANAGIOTAKOS, D. B., C. PITSAVOS, C. CHRYSOHOOU, et al. Effect of leisure time physical activity on blood lipid levels: the ATTICA study. *Coron. Artery Dis.* 14:533–539, 2003.
22. SAS, INC. *SAS/STAT User's Guide.* Cary, NC, 1989, 1686 pp.
23. SJÖSTROM L., H. KVIST, A. CEDERBLAD, and U. TYLEN. Determination of total adipose tissue and body fat in women by computed tomography, 40K, and tritium. *Am. J. Physiol. Endocrinol. Metab.* 250:E736–E745, 1986.
24. SKINNER, J. S., K. M. WILMORE, J. B. KRASNOFF, et al. Adaptation to a standardized training program and changes in fitness in a large, heterogeneous population: the HERITAGE Family Study. *Med. Sci. Sports Exerc.* 32:157–161, 2000.
25. SOPKO, G., A. S. LEON, D. R. Jr., JACOBS, et al. The effects of exercise and weight loss on plasma lipids in young obese men. *Metabolism* 34:227–236, 1985.
26. TRAN, Z. V., and A. WELTMAN. Differential effects of exercise on serum lipid and lipoprotein levels seen with changes in body weight: a meta-analysis. *JAMA* 254:919–924, 1985.
27. WILLIAMS, P. T. Health effects resulting from exercise versus those from body fat loss. *Med. Sci. Sports Exerc.* 33:S611–S621, 2001.
28. WILLIAMS, P. T., R. M. KRAUSS, K. M. VRANIZAN, and P. D. S. WOOD. Changes in lipoprotein subfractions during diet-induced and exercise-induced weight loss in moderately overweight men. *Circulation* 81:1293–1304, 1990.
29. WILMORE, J. H. A simplified method for determination of residual lung volumes. *J. Appl. Physiol.* 27:96–100, 1969.
30. WOOD, P. T., M. L. STEFANICK, D. M. DREON, et al. Changes in plasma lipids and lipoproteins in overweight men during weight loss through dieting as compared with exercise. *JAMA* 319:1173–1179, 1988.