Increases in High-Density Lipoprotein Cholesterol With Endurance Exercise Training Are Blunted in Obese Compared With Lean Men

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The effectiveness of endurance exercise training (without concomitant weight loss) for improving lipoprotein lipid levels in obese individuals remains controversial. The purpose of this study was to determine whether lipoprotein lipid responses to endurance exercise training are affected by obesity. Healthy middle-aged and older (57 ± 2 years) lean (n = 16; body mass index [BMI], 22 to 26 kg/m^2), moderately obese (n = 15; BMI, 27 to 30 kg/m^2), and obese (n = 15; BMI, 31 to 37 kg/m^2) men underwent a 9-month endurance exercise training program. The groups differed in the initial degree of obesity, waist circumference, and waist to hip ratio (WHR), but not in age or maximal aerobic capacity ($\dot{V}O_2$max). The obese group had lower baseline levels of high-density lipoprotein cholesterol (HDL-C) and HDL$_2$-C, and higher triglyceride (TG) levels than the lean group. Exercise training increased $\dot{V}O_2$max to a comparable degree in lean, moderately obese, and obese groups (18%, 24%, and 18%, respectively, $P < .01$). Exercise training significantly decreased TG levels in all groups, whereas total cholesterol and low-density lipoprotein cholesterol (LDL-C) decreased only in the obese group. Exercise training increased HDL-C and HDL$_2$-C levels in lean (14% and 81%, respectively, $P < .05$) and moderately obese (7% and 59%, respectively, $P < .05$) men, whereas neither HDL-C nor HDL$_2$-C changed in obese men. The change in HDL-C correlated negatively with initial BMI ($r = -.42$, $P < .01$) and waist circumference ($r = -.43$, $P < .01$). These results show that the effects of exercise training on HDL-C are blunted in obese middle-aged and older men, whereas improvements in TG occur independently of the degree of obesity.

DYSLIPIDEMIA is a major risk factor for the development of coronary artery disease (CAD) in older individuals.[1,2] Besides aging, obesity and physical inactivity are important determinants of lipoprotein lipid levels in the elderly.[3,4] As a result, endurance exercise training and weight loss are advocated as treatments to improve lipoprotein lipid profiles in older populations. In general, cross-sectional studies confirm that physically active middle-aged and older men have better lipid profiles than sedentary individuals.[3,5,6] However, this may be largely due to the lean body habitus associated with vigorous long-term exercise. In addition, a number of studies in subjects of all ages suggest that loss of body fat is necessary to elicit changes in lipid metabolism with endurance exercise training.[7-9] Thus, the effectiveness of endurance exercise training (without concomitant weight loss) in improving lipoprotein lipid levels in obese individuals remains controversial.

We previously showed that dietary-induced weight loss resulted in greater improvements in lipid lipoprotein profiles compared with endurance exercise training without weight loss in two groups of men matched for age, obesity, and maximal aerobic capacity ($\dot{V}O_2$max).[9] These results raised the question of whether the lipid responses to endurance exercise training were blunted because the men were obese. To investigate this issue, we examined the effects of 9 months of endurance exercise training without weight loss on lipid metabolic risk factors for coronary artery disease (CAD) in groups of lean, moderately obese, and obese middle-aged and older men.

SUBJECTS AND METHODS

Subjects

Healthy, nonsmoking, sedentary male volunteers aged 45 to 80 years with a wide range of obesity and physical conditioning status were recruited from the Baltimore-Washington metropolitan area for participation in cross-sectional and longitudinal intervention studies that examined the effects of obesity and physical conditioning on cardiovascular risk factors for CAD. All men underwent a screening physical examination and a fasting blood profile to exclude subjects with overt diabetes (fasting glucose level >7.8 mmol/L), hypertension (blood pressure >160/90 mm Hg), hyperlipidemia (triglyceride [TG] or low-density lipoprotein cholesterol [LDL-C] >90th percentile for age and sex),[10] cancer, liver, renal, or hematologic disease, or orthopedic limitations. A graded exercise test according to the protocol used by Bruce and Horstein[11] was also performed to exclude men with exercise-induced myocardial ischemia. None of the men were on medications that affect blood pressure or lipid or glucose metabolism. All men provided informed consent to participate in the study according to the guidelines of the University of Maryland and Johns Hopkins Bayview Medical Center Human Studies Institutional Review Boards.

The first 170 obese (body mass index [BMI] > 27 kg/m^2) men were enrolled over a 6-year period in a randomized clinical trial that compared the effects of weight loss alone versus aerobic exercise training alone on CAD risk factors. The results of this clinical trial have been previously reported.[9] During the final 2 years of the trial, an additional 28 lean men (BMI < 27 kg/m^2) were enrolled in an aerobic exercise training intervention. This permitted an examination of the relationship between baseline body composition and the effects of aerobic exercise training on CAD risk factors. Therefore, a total of 99 sedentary men (71 with BMI > 27 kg/m^2 and 28 with BMI < 27 kg/m^2) were enrolled in interventions with exercise training only. Of

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these 99 men, 65 completed the exercise training protocol; the others withdrew for personal reasons. In this study, we report data on 46 men who met the following criteria: (1) an increase in $\dot{V}O_2$max of greater than 5% and (2) less than 5% loss of body weight with training. The age range of these men was 46 to 72 years, with 10 of the men younger than 50 years and eight older than 65 years. Data on the 30 men with a BMI greater than 27 kg/m^2 have been previously reported,[9] whereas data on the men with a BMI less than 27 kg/m^2 have not been reported previously.

Study Protocol

Before baseline testing, the subjects were instructed by a registered dietitian for 3 months on the principles of an American Heart Association (AHA) phase I diet.[12] By the end of 3 months, all of the men were in compliance with the AHA phase I diet. Subjects were asked to remain weight-stable and to follow this diet throughout the entire exercise intervention. Compliance was monitored weekly by recording body weight and reviewing 7-day food-exchange records. The macronutrient composition of the diets was 52% ± 6% for carbohydrate, 30% ± 4% for fat, and 18% ± 2% for protein, both the week before the start of baseline testing and the week before posttesting. To further ensure dietary stability during metabolic testing, subjects were provided with a 6-day weight-maintaining AHA phase I diet to be consumed during the testing period. The macronutrient composition of this diet was consistent with what was reported on each subject's 7-day food record.

After completion of baseline testing, all subjects entered a 9-month endurance exercise intervention. The men exercised on treadmills, cycle ergometers, and ski machines 3 d/wk at our facility under the supervision of a trained exercise physiologist, a nurse, and the investigators. The exercise intensity was prescribed based on target heart rates (THRs) calculated from the Karvonen equation, [HRR · (0.50-0.80)] + HRrest, where heart rate reserve (HRR) is maximal HR minus resting HR obtained from each subject's $\dot{V}O_2$max.[13] The duration and intensity of the exercise progressed from 10 to 20 minutes at 50% to 60% HRR during the first week of exercise, to 45 to 60 minutes at 70% to 80% HRR by the fourth month. The subjects warmed-up and cooled-down by walking 3 to 5 minutes at a slow pace. To ensure compliance with the training intensity, at least three heart rate readings were taken during each exercise session and recorded in an exercise log book. Body weight was recorded once per week to monitor weight stability, and if any subject lost more than 0.5 kg from the previous week, calories were increased to maintain body weight. Compliance with the training program was maintained by telephoning subjects who missed two consecutive sessions. Attendance was greater than 80% of all possible exercise sessions for all three groups. Following the 9 months of exercise training, the men were again provided weight-maintaining AHA phase I diets for 6 days before and during metabolic testing. Post–exercise training evaluations of body composition, $\dot{V}O_2$max, and lipoprotein lipids were performed 24 to 36 hours after the last exercise session for each subject.

Testing Procedures

All measures of body composition were performed at the same time of the morning after a 12-hour fast. The minimal waist circumference and the circumference of the maximal gluteal protuberance were measured in duplicate, and the waist to hip ratio (WHR) was calculated. Body density was measured by hydrostatic weighing with correction for residual lung volume determined by helium dilution. Percent body fat was calculated using the Siri equation.[14] Fat-free mass was calculated as the difference between total body mass and fat mass.

$\dot{V}O_2$max was measured on a motor-driven treadmill during a progressive exercise test to voluntary exhaustion, as previously described.[15] $\dot{V}O_2$max was considered valid when at least two of three criteria were met: (1) maximal heart rate greater than 90% of age-predicted maximal heart rate (220 bpm − age), (2) respiratory exchange ratio of at least 1.10, and (3) plateau in $\dot{V}O_2$ (<0.2 L/min change) with increasing work rate. $\dot{V}O_2$max is expressed in liters per minute and, milliliters per kilogram per minute.

Venous blood samples for measurement of lipoprotein lipid levels were drawn after a 12-hour fast on days 4 and 6 of the controlled metabolic diet. The reported values are the mean of these two samples. The samples were transferred to chilled tubes containing 1 mg EDTA/mL blood, and the plasma was separated by centrifugation at 4°C. Total cholesterol, high-density lipoprotein cholesterol (HDL-C), HDL_2-C, and TG levels were measured, and LDL-C was calculated as previously described.[9]

Statistics

The 46 men were classified into three groups according to a tertile split for the initial level of obesity as measured by BMI. The lean (BMI, 22 to 26 kg/m^2), moderately obese (BMI, 27 to 30), and obese (BMI, 31 to 37) groups consisted of 16, 15, and 15 men, respectively. Data were analyzed using the Macintosh Statview program (Abacus Concepts, Calabasas, CA). Within-group differences between preintervention and postintervention measures of all variables were determined using a paired *t* test. Differences between groups at baseline and as a result of the intervention were tested for significance using ANOVA and Scheffe's test. Stepwise multiple regression analyses were performed to determine independent predictors of the changes in lipid variables. The level of significance was set at *P* less than .05 for all analyses. Data are reported as the mean ± SD.

RESULTS

Baseline Characteristics

All three groups differed significantly in the initial measures of obesity, including waist circumference and WHR (Table 1). There were no significant differences between groups in initial age or $\dot{V}O_2$max. The obese group had lower initial levels of HDL-C and HDL_2-C than the lean group and higher TG levels than the lean and moderately obese groups (Table 2).

Effect of Exercise Training on Lipoprotein Lipids

Exercise training increased $\dot{V}O_2$max ($P < .0001$) in all three groups (18%, 24%, and 18% for lean, moderately obese, and obese, respectively). There were small but significant ($P < .05$) decreases in body weight (<2 kg) and percent body fat (<1.5%) in all groups as a result of the 9 months of endurance

Table 1. Baseline Physical Characteristics of the Lean, Moderately Obese, and Obese Groups

Characteristic	Lean (n = 16)	Moderately Obese (n = 15)	Obese (n = 15)
Age (yr)	57 ± 6	59 ± 6	55 ± 7
Weight (kg)	81 ± 9[a]	89 ± 9[b]	97 ± 12[c]
BMI (kg/m^2)	25 ± 1[a]	29 ± 1[b]	33 ± 2[c]
Body fat (%)	23 ± 6[a]	27 ± 5[b]	31 ± 6[b]
Fat-free mass (kg)	63 ± 6	65 ± 8	67 ± 10
Waist (cm)	91 ± 6[a]	100 ± 5[b]	111 ± 6[c]
WHR	0.91 ± 0.05[a]	0.95 ± 0.04[b]	1.00 ± 0.04[c]
$\dot{V}O_2$max			
L/min	2.6 ± 0.5	2.6 ± 0.6	2.7 ± 0.5
mL/kg/min	32 ± 5	30 ± 5	28 ± 4

NOTE. Data are the mean ± SD. Different superscripts distinguish differences at $P < .05$ among groups by Scheffe's test.

Table 2. Fasting Lipid Values Before and After 9 Months of Endurance Exercise Training in Lean, Moderately Obese, and Obese Men

Group	TG	Total Cholesterol	HDL-C	HDL_2-C	LDL-C
Lean (n = 16)					
Pretraining	1.17 ± 0.49[a]	4.63 ± 0.59	0.93 ± 0.10[a]	0.09 ± 0.05[a]	3.15 ± 0.52
Posttraining	0.96 ± 0.27*	4.47 ± 0.72	1.06 ± 0.18†	0.16 ± 0.12*	2.97 ± 0.57
Change	−0.20 ± 0.35	−0.19 ± 0.48	0.10 ± 0.09[a]	0.04 ± 0.07	−0.21 ± 0.51
Moderately obese (n = 15)					
Pretraining	1.24 ± 0.40[a]	4.65 ± 0.98	0.85 ± 0.16	0.05 ± 0.05[b]	3.23 ± 0.93
Posttraining	1.10 ± 0.38*	4.50 ± 0.80	0.91 ± 0.18*	0.10 ± 0.11*	3.08 ± 0.75
Change	−0.15 ± 0.29	−0.16 ± 0.53	0.06 ± 0.08[a]	0.05 ± 0.07	−0.15 ± 0.46
Obese (n = 15)					
Pretraining	1.73 ± 0.63[b]	4.68 ± 0.52	0.78 ± 0.13[b]	0.03 ± 0.03[b]	3.08 ± 0.59
Posttraining	1.43 ± 0.51†	4.29 ± 0.39*	0.80 ± 0.13	0.04 ± 0.04	2.84 ± 0.39*
Change	−0.29 ± 0.37	−0.37 ± 0.41	0.01 ± 0.07[b]	0.01 ± 0.03	−0.25 ± 0.39

NOTE. Data are the mean ± SD in mmol/L. Different superscript letters distinguish differences at $P < .05$ among groups by Scheffe's test.
*$P < .05$, †$P < .01$: within-group pre- v post-exercise training.

exercise training. Fat-free mass did not change in any group. Neither absolute nor percent changes in $\dot{V}O_2$max and body weight differed among groups (Fig 1).

Exercise training significantly decreased TG levels in all groups, whereas total cholesterol and LDL-C decreased only in the obese group (Table 2). However, absolute and relative changes in total cholesterol, LDL-C, and TG following exercise did not differ among groups (Fig 2). Exercise training increased HDL-C and HDL_2-C levels in lean (14% and 81%, respectively, $P < .05$) and moderately obese (7% and 59%, respectively, $P < .05$) men, whereas neither HDL-C nor HDL_2-C changed in obese men (Table 2). Absolute and relative changes in both HDL-C and HDL_2-C were significantly greater in lean compared with obese men (Fig 3).

Relationship of Body Composition to Changes in Lipoprotein Lipids

Univariate regression analyses were used to determine the contribution of the change in $\dot{V}O_2$max and initial measures of obesity to changes in lipoprotein lipids using data from all 46 men. Absolute and relative changes in $\dot{V}O_2$max did not correlate with changes in any of the lipid variables. Changes in total cholesterol, LDL-C, and TG were not related to initial BMI, percent body fat, or waist circumference. However, the increase in HDL-C with exercise training correlated negatively with initial BMI ($r = -.42$, $P < .01$) and waist circumference ($r = -.43$, $P < .01$), but not with percent body fat ($r = -.23$, $P = .13$). The increase in HDL_2-C did not correlate with initial BMI ($r = -.26$, $P = .08$) or waist circumference ($r = -.21$, $P = .16$), but did correlate negatively with percent body fat ($r = -.36$, $P < .05$) (Fig 4).

Stepwise multiple regression analyses were used to determine the independent predictors of the change in HDL-C. When initial BMI, waist circumference, and percent body fat were included in the model, the analysis indicated that waist circumference was the only independent predictor of the change in HDL-C ($r = -.43$, $P < .01$).

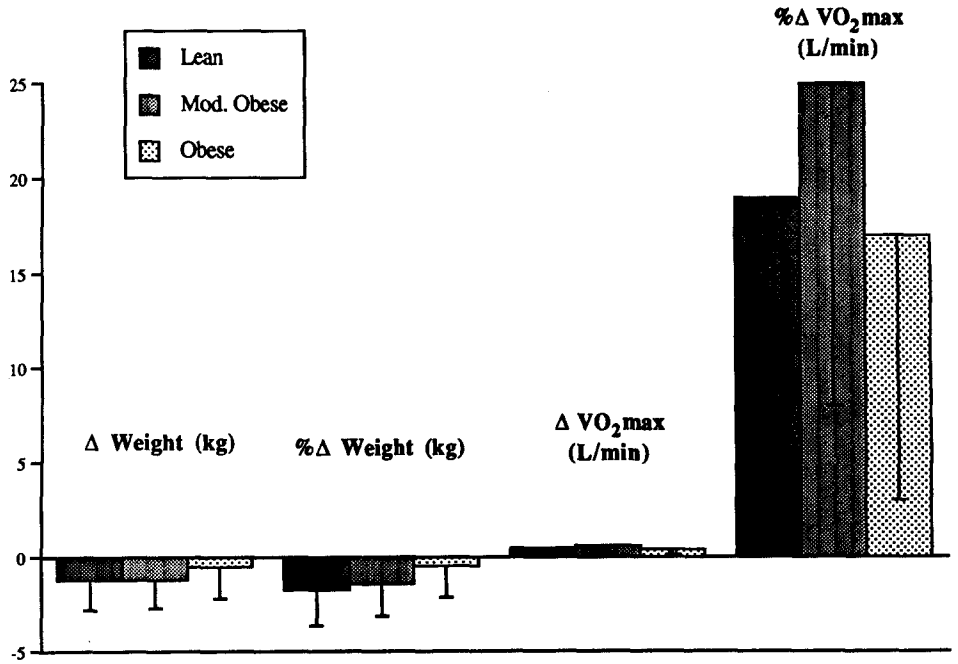

Fig 1. Absolute and relative changes in weight and $\dot{V}O_2$max following 9 months of endurance exercise training in lean, moderately obese, and obese men. There were no significant differences among groups.

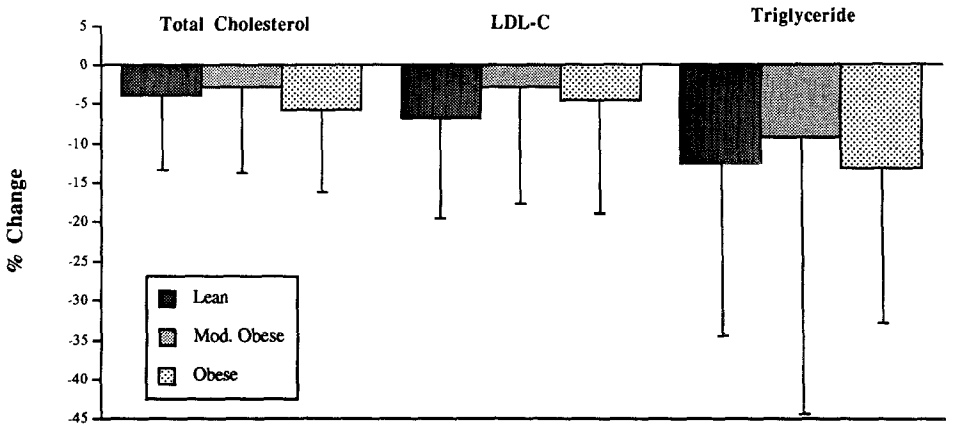

Fig 2. Relative changes in total cholesterol, LDL-C, and TG following 9 months of endurance exercise training in lean, moderately obese, and obese men. There were no significant differences among groups.

DISCUSSION

Endurance exercise training is advocated as an effective treatment for low HDL-C levels in older patients.[16] The results of this study show that 9 months of endurance exercise training decreased total cholesterol, TG, and LDL-C levels to a comparable degree in lean, moderately obese, and obese middle-aged and older men. However, exercise training increased HDL-C and HDL_2-C levels in lean and moderately obese men, but not in obese men. Furthermore, increases in HDL-C and HDL_2-C with training were inversely related to BMI and waist circumference at baseline. These results indicate that improvements in HDL-C levels with endurance exercise training are blunted in obese compared with lean men, and suggest that weight loss may need to be incorporated with exercise training to increase plasma HDL-C in obese middle-aged and older men.

We previously showed that dietary-induced weight loss increased HDL-C and HDL_2-C to a greater extent than endurance exercise training without weight loss in obese men of this age,[9] suggesting that weight loss is the preferred treatment for improving the lipid profile of healthy, obese middle-aged and older men. In that study, HDL-C did not significantly increase in 49 obese men who underwent 9 months of endurance exercise training that increased $\dot{V}O_2$max by 17% and who remained weight-stable. However, HDL-C levels increased 13% in 44 men who followed a hypocaloric diet for 9 months and lost a mean of 9.5 kg with no change in $\dot{V}O_2$max.

The men in each group of the present study were of comparable age and initial cardiovascular fitness level (either expressed in liters per minute or normalized for body weight) but differed with respect to baseline body fat composition and distribution as indicated by significant differences in BMI, percent body fat, waist circumference, and WHR among the groups. The men also differed with respect to initial HDL-C and TG concentrations. This raised the question as to whether differences in these baseline parameters could account for the differential response to training in lean and obese men. Multiple regression analyses performed to test this question showed that BMI and waist circumference were the strongest predictors of the change in HDL-C, with no independent contribution from the change in $\dot{V}O_2$max, WHR, or baseline HDL-C or TG concentrations. It is possible that our results were obtained because the obese men were not able to exercise at the same level of intensity as the moderately obese and leaner men. However, this is unlikely, because attendance was the same for

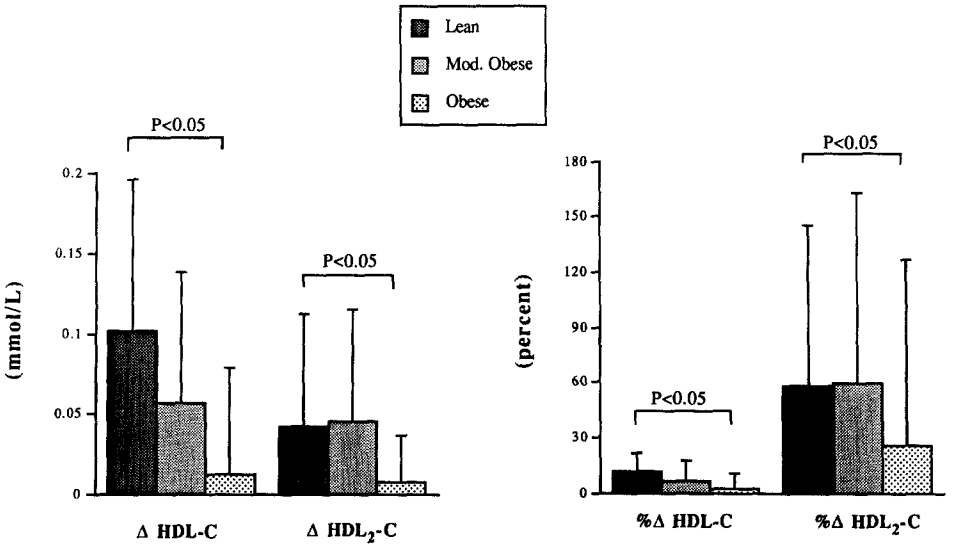

Fig 3. Absolute and relative changes in HDL-C and HDL_2-C following 9 months of endurance exercise training in lean, moderately obese, and obese men.

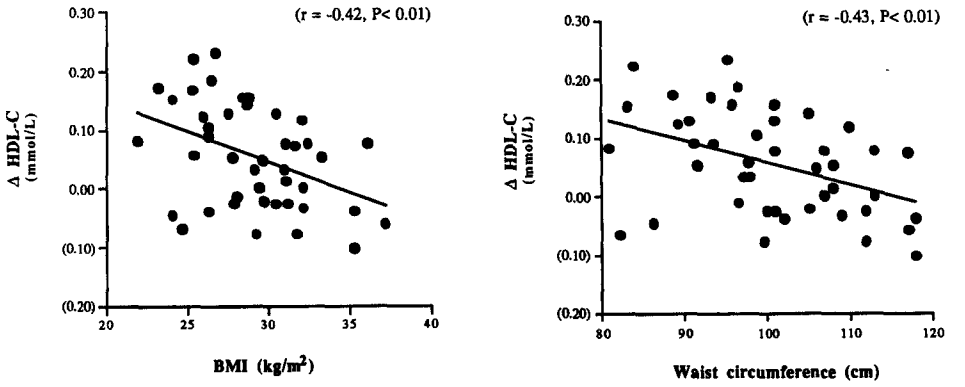

Fig 4. Relationship between changes in HDL-C levels and initial BMI and waist circumference.

all groups, and by the fourth month of training, all groups were exercising at the same relative intensity (70% to 80% heart rate reserve) for the same duration (45 to 60 minutes). Thus, the blunted HDL-C response seen in the obese men is most likely due to the excess amount of adipose tissue.

The results of endurance exercise training studies on HDL-C levels in middle-aged and older individuals are disparate. Contrasting results may be due to differences in baseline body composition or to a failure to control changes in body weight with exercise training. In a meta-analysis that included 95 studies, Tran and Weltman[17] showed that there is a significant relationship between changes in body weight and lipoprotein lipid responses to exercise training in subjects of all ages. Studies in middle-aged and older individuals show that subjects who have an increase in HDL-C with exercise training either are not obese (BMI < 27 kg/m^2)[18-20] or lost body weight during the exercise training period.[7,8,21-23] Furthermore, few of these studies controlled for differences in dietary composition between subjects, which could influence the response of lipoprotein lipids to endurance exercise training. Subjects in the present study were stabilized on the AHA phase I diet for the duration of the study. Also, to eliminate differences in macronutrient composition before obtaining the duplicate blood samples for determination of lipoprotein lipid levels, subjects were provided with an identical-composition weight-maintaining AHA phase I diet for 4 to 6 days both before and after exercise training.

The duration and intensity of the endurance training intervention may also affect the HDL-C response to exercise. In one study, 8 months of exercise training had no significant effect on HDL-C levels, whereas 14 months of training increased HDL-C levels by 6%.[24] In another study, 1 year of exercise had little effect on HDL-C levels in older adults, whereas individuals who continued to exercise for 2 years had a 5-mg/dL increase.[25] Similarly, 6 months of low-intensity exercise had no effect on HDL-C concentrations in 11 non-obese, older men and women, but six additional months of high-intensity training increased HDL-C levels by 14%.[19] Other studies suggest that there is a training intensity threshold at which beneficial changes in HDL-C levels occur.[22,26] This suggests that the duration and intensity of exercise training required for improvements in HDL-C concentrations are important and may be greater than previously appreciated. Thus, the effect of an exercise program on HDL-C levels seems dependent on the baseline weight and regional body fat distribution, the duration and intensity of exercise training, and the presence or absence of concomitant weight loss. It is possible that the obese men in our study would have had greater improvements in HDL-C concentrations if they had either exercised more vigorously for a longer period or lost a significant amount of weight.

A number of physiological mechanisms might be responsible for the blunted HDL-C response to exercise training in obese men. Obesity, particularly visceral obesity, is associated with increased free fatty acid (FFA) concentrations, which are associated with adverse effects on glucose and lipoprotein metabolism, including the development of insulin resistance, increased production of TG-rich very–low-density lipoproteins, and a reduction in HDL particles.[27] Although FFA concentrations were not measured in these subjects, previous studies in our laboratory demonstrated an inverse association between the suppression of plasma FFA levels during a euglycemic-hyperinsulinemic clamp and measures of total obesity and WHR.[28] Other investigators also report adverse effects of obesity and abdominal body fat distribution on FFA kinetic responses to exercise.[29] However, no study examined these relationships in lean and obese subjects. Whether abnormalities in FFA metabolism associated with abdominal obesity and insulin resistance can account for the blunted HDL-C response to exercise training in obese subjects in the present study remains to be determined.

In conclusion, these results suggest that endurance exercise training without concomitant weight loss increases HDL-C levels in lean and moderately obese middle-aged and older men, but not in obese men. Although exercise training did not significantly increase HDL-C concentrations in obese men, it did decrease TG, LDL-C, and total cholesterol levels. Thus, obese men achieve other lipid benefits from exercise training, despite a lack of improvement in HDL-C levels. Therefore, public health policy to reduce the risk for CAD in obese middle-aged and older individuals should more strongly emphasize the importance of endurance exercise training in combination with weight loss.

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REFERENCES

1. Anderson KM, Castelli WP, Levy D: Cholesterol and mortality: 30 years of follow-up from the Framingham Study. JAMA 257:2176-2180, 1987

2. Gordon DJ, Probstfield JL, Garrison RI, et al: High-density lipoprotein cholesterol and cardiovascular disease: Four prospective American studies. Circulation 79:8-15, 1989

3. Coon PJ, Bleecker ER, Drinkwater DT, et al: Effects of body composition and exercise capacity on glucose tolerance, insulin, and lipoprotein lipids in healthy older men: A cross-sectional and longitudinal intervention study. Metabolism 38:1201-1209, 1989

4. National Cholesterol Education Program Expert Panel, National Heart, Lung, and Blood Institute: Report of the National Cholesterol Education Program Expert Panel on detection, evaluation, and treatment of high blood cholesterol in adults. Arch Intern Med 148:36-69, 1988

5. Seals DR, Allen WK, Hurley BF, et al: Elevated high-density lipoprotein cholesterol levels in older endurance athletes. Am J Cardiol 54:390-393, 1984

6. Giada F, Vigna GB, Vitale E, et al: Effect of age on the response of blood lipids, body composition, and aerobic power to physical conditioning and deconditioning. Metabolism 44:161-165, 1995

7. Wood PD, Stefanick ML, Dreon DM, et al: Changes in plasma lipids and lipoproteins in overweight men during weight loss through dieting as compared with exercise. N Engl J Med 319:1173-1179, 1988

8. Williams PT, Krauss RM, Vranizan KM, et al: Changes in lipoprotein subfractions during diet-induced and exercise-induced weight loss in moderately overweight men. Circulation 81:1293-1304, 1990

9. Katzel LI, Bleecker ER, Colman EG, et al: Effects of weight loss vs aerobic exercise training on risk factors for coronary disease in healthy, obese, middle-aged and older men. JAMA 274:1915-1921, 1995

10. Rifkind BM, Segel P: Lipid Research Center Program reference values for hyperlipidemia and hypolipidemia. JAMA 250:1869-1872, 1983

11. Bruce RA, Horstein TR: Exercise stress testing in evaluation of patients with ischemic heart disease. Prog Cardiovasc Dis 11:371-390, 1969

12. American Heart Association Steering Committee for Medical and Community Programs: Phase I diet. Circulation 62:A449-A455, 1985

13. Karvonen M, Kentala K, Musta O: The effects of training heart rate: A longitudinal study. Ann Med Exp Biol Fenn 35:307-315, 1957

14. Siri WE: The gross composition of the body. Adv Biol Med Phys 4:239-280, 1956

15. Meyers D, Goldberg AP, Coon PJ, et al: Relationship of obesity and physical fitness to cardiopulmonary and metabolic function in healthy older men. J Gerontol 46:M57-M65, 1991

16. Durstine JL, Haskell WL: Effects of exercise training on plasma lipids and lipoproteins. Exerc Sport Sci Rev 22:477-521, 1994

17. Tran ZV, Weltman A: Differential effects of exercise on serum lipid and lipoprotein levels seen with changes in body weight. A meta-analysis. JAMA 254:919-924, 1985

18. Motoyama M, Sunami Y, Kinoshita F, et al: The effects of long-term intensity aerobic training and detraining on serum lipid and lipoprotein concentrations in elderly men and women. Eur J Appl Physiol 70:126-131, 1995

19. Seals DR, Hagberg J, Hurley BF, et al: Effects of endurance training on glucose tolerance and plasma lipid levels in older men and women. JAMA 252:645-649, 1984

20. Schwartz RS, Shuman WP, Larson V, et al: The effect of intensive endurance exercise training on body fat distribution in young and older men. Metabolism 40:545-551, 1991

21. Williams PT, Wood PD, Krauss RM, et al: Does weight loss cause the exercise-induced increase in plasma high density lipoproteins? Atherosclerosis 47:173-185, 1983

22. Wood PD, Haskell WL, Blair SN, et al: Increased exercise level and plasma lipoprotein concentrations: A one-year, randomized, controlled study in sedentary, middle-aged men. Metabolism 32:31-39, 1983

23. Ohta T, Kawamura T, Hatano K, et al: Effects of exercise on coronary risk factors in obese, middle-aged subjects. Jpn Circ J 54:1459-1464, 1990

24. Blumenthal JA, Emery CF, Madden DJ, et al: Effects of exercise training on cardiorespiratory function in men and women >60 years of age. Am J Cardiol 67:633-639, 1991

25. King AC, Haskell WL, Young DR, et al: Long-term effects of varying intensities and formats of physical activity on participation rates, fitness, and lipoproteins in men and women aged 50 to 65 years. Circulation 91:2596-2604, 1995

26. Stein RA, Michielli DW, Glantz MD, et al: Effects of different exercise training intensities on lipoprotein cholesterol fractions in healthy middle-aged men. Am Heart J 119:277-283, 1990

27. Bjorntorp P: Fatty acids, hyperinsulinemia, and insulin resistance: Which comes first? Curr Opin Lipidol 5:166-174, 1994

28. Coon PJ, Rogus EM, Goldberg AP: Time course of plasma free fatty acid concentration in response to insulin: Effect of obesity and physical fitness. Metabolism 41:711-716, 1992

29. Kanaley JA, Cryer PE, Jensen MD: Fatty acid kinetic responses to exercise. Effects of obesity, body fat distribution and energy-restricted diet. J Clin Invest 92:255-261, 1993