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Resistance exercise training reduces hypertriglyceridemia in HIV-infected men treated with antiviral therapy

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Yarasheski, Kevin E., Pablo Tebas, Barbara Stanerson, Sherry Claxton, Donna Marin, Kyongtae Bae, Michael Kennedy, Woraphot Tantisiriwat, and William G. Powderly. Resistance exercise training reduces hypertriglyceridemia in HIV-infected men treated with antiviral therapy. *J Appl Physiol* 90: 133–138, 2001.—Hypertriglyceridemia, peripheral insulin resistance, and trunk adiposity are metabolic complications recently recognized in people infected with human immunodeficiency virus (HIV) and treated with highly active antiretroviral therapy (HAART). These complications may respond favorably to exercise training. Using a paired design, we determined whether 16 wk of weight-lifting exercise increased muscle mass and strength and decreased fasting serum triglycerides and adipose tissue mass in 18 HIV-infected men. The resistance exercise regimen consisted of three upper and four lower body exercises done for 1–1.5 h/day, 4 days/wk for 64 sessions. Dual-energy X-ray absorptiometry indicated that exercise training increased whole body lean mass 1.4 kg ($P = 0.005$) but did not reduce adipose tissue mass (P = NS). Axial proton-magnetic resonance imaging indicated that thigh muscle cross-sectional area increased 5–7 cm^2 ($P < 0.005$). Muscle strength increased 23–38% ($P < 0.0001$) on all exercises. Fasting serum triglycerides were decreased at the end of training (281–204 mg/dl; $P = 0.02$). These findings imply that resistance exercise training-induced muscle hypertrophy may promote triglyceride clearance from the circulation of hypertriglyceridemic HIV-infected men treated with antiviral therapy.

AIDS; metabolic complications; progressive resistance exercise training; muscle protein mass; lipid metabolism; magnetic resonance imaging

THE METABOLIC COMPLICATIONS associated with infection and treatment of human immunodeficiency virus (HIV) include muscle wasting, peripheral insulin resistance, hypertriglyceridemia, hypercholesterolemia, central adiposity, and osteopenia (1, 17, 19, 23, 28). These metabolic complications may be associated with increased morbidity, mortality, and disability in people infected with HIV, despite the fact that highly active antiretroviral therapy (HAART) has been so effective in lowering plasma viremia and increasing CD4+ T lymphocyte counts in most patients. Resistance exercise training (weight lifting) and endurance exercise training are established and efficacious interventions for the management of these complications in non-HIV-infected people. Their efficacy as nonpharmacological treatments for HIV-related metabolic complications has received little attention.

To date, reports indicate that supervised weight lifting and aerobic exercise are not contraindicated in people infected with HIV (1, 4, 13, 16–19, 21, 22). An acute bout of stepping exercise did not increase circulating HIV RNA (viremia) detected with a sensitive RT-PCR assay (18). Eight weeks of progressive resistance exercise training (3 days/wk, 1 h/day, 80% maximum lifting capacity) increased maximum voluntary muscle strength and physical function in people infected with HIV (17, 21). The exercise program increased fat-free mass (FFM) in wasting subjects (2.8 kg) and in nonwasting subjects (1.4 kg FFM). These findings suggested that exercise effectively increased lean tissue mass in HIV-infected men and women and may be more effective at restoring lean mass in HIV-wasting patients. A recent preliminary report suggests that combined aerobic and resistance exercise training may reduce trunk adiposity in HIV-infected patients receiving HAART (19).

We examined whether an individualized and supervised progressive resistance exercise-training program improves the metabolic complications associated with HIV. We hypothesized that weight lifting exercises training (16 wk × 4 day/wk) would increase skeletal muscle cross-sectional area, increase maximum voluntary muscle strength, reduce central adipose tissue mass, and reduce serum cholesterol and total triglyceride concentrations in HIV-infected men.

METHODS

Eighteen asymptomatic HIV-infected male volunteers were enrolled and completed the 16-wk exercise training

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program (Table 1). All were stable on antiviral therapy for at least 1 mo before enrollment. None had a history of cardiovascular disease, and resting electrocardiograms were normal in all participants. Volunteers were excluded if they reported use of testosterone derivatives or recombinant human growth hormone within 60 days before enrollment. Weight history was available for 17 of the 18 subjects. In the year before enrollment, seven subjects had >5% weight loss, seven had <5% weight loss, and three had gained weight (+1–9%). Volunteers whose physical activity patterns exceeded the minimum guidelines for exercise training established by the American College of Sports Medicine were excluded from the study (15). Written, informed consent was obtained after the purpose, procedures, and potential risks were explained to each volunteer. This study was approved by the Institutional Review Board at Washington University Medical School.

The weight-lifting program consisted of three upper and four lower body exercises. The program required 1–1.5 h/day, 4 days/wk for 64 sessions. The initial sessions were low intensity (50–65% of maximum strength) and high repetition (10+ lifts/exercise), and each exercise was done two or three times per session. A personal trainer monitored, prescribed, and progressively augmented the exercise prescription, so that by *week 16* the exercise intensity was 75–85% of final maximum voluntary muscle strength at low repetition (5–8 lifts/exercise), and each exercise was done three to four times per session.

During the first three to four exercise sessions, one-repetition maximum (1 RM) was determined on each of six exercise machines (Lido). 1 RM was determined as the maximum amount of weight a subject could lift just once (and attempting twice) using proper form and only primary and secondary muscle groups required for the lifting motion. On *day 1*, 1 RM was determined by trial and error. On *days 2–4*, the initial 1 RM plus additional weight was tested. The highest 1 RM achieved was considered the baseline maximum voluntary muscle strength. Maximum voluntary torque production (0 and 60°/s) of the knee extensor and flexor muscles was determined on an isokinetic dynamometer (Cybex).

At baseline, blood CD4+ T cell count and plasma HIV RNA (Roche Amplicor Assay) were measured, and current antiviral medications were documented. Fasting serum triglycerides and total and high-density lipoprotein (HDL) cholesterol were measured in the Lipid Research Center Core Laboratory at Washington University Medical Center. Serum total cholesterol and glycerol-blanked triglyceride concentrations were measured by using enzymatic kits from Bayer and Diagnostics on a Hitachi 917 analyzer. HDL cholesterol concentration was measured as above after precipitation of apoB-containing lipoproteins from the serum using dextran sulfate (26). Low-density lipoprotein (LDL) cholesterol concentration was estimated by using the Friedewald equation (6). The accuracy of these methods is verified and standardized by participation in the Centers for Disease Control lipid standardization program and cholesterol reference method laboratory network and the College of American Pathologists external proficiency program (14). Insulin, C-peptide, glucagon, and proinsulin concentrations were measured in a commercial radioimmunoassay laboratory (Linco Research Labs, St. Charles, MO). Baseline and postexercise blood samples were obtained after a 10- to 12-h overnight fast (at 0730–0930).

Table 1. *Baseline characteristics*

No. of subjects	18
Age, yr	42 ± 2
Gender, M/F	18/0
Anti-HIV medications	16 HAART 2 PI-naive
CD4, counts/μl (range)	152–840
Plasma viral load	
copies/ml	<400 in 11 subjects
range	$1.7–93 \times 10^3$
Height, cm	177 ± 2
Weight, kg	71 ± 3
BMI, kg/m^2	23 ± 1
Whole body adiposity, %	19 ± 1
Trunk/appendicular adiposity	1.53 ± 0.16

Values are means ± SE. HIV, human immunodeficiency virus; HAART, highly active antiretroviral therapy; PI, protease inhibitor; BMI, body mass index.

Whole body and regional lean and adipose tissue masses were measured using a Hologic QDR-2000 dual-energy X-ray absorptiometer. Images were processed using Hologic Enhanced Array Whole body software (v5.71A). A ratio of trunk-to-appendicular adipose tissue ≥1.1 was used to identify subjects with trunk adiposity at baseline (23).

Thigh muscle and adipose tissue cross-sectional areas were measured by using proton-magnetic resonance imaging. Eight T_1-weighted axial scans were obtained 12–24 cm above the lateral condyle on the head of the tibia at baseline and after 16 wk of exercise training. Right and left thigh muscle, adipose tissue, and intramuscular adipose tissue cross-sectional areas were determined in all eight axial scans by using a macro that operates under NIH Image software (v1.61b7). The macro removes the bone area from the cross section, determines the range of pixel intensities for muscle and adipose tissue, and sets threshold intensity ranges for these tissues. The macro reduces some of the subjectivity associated with identification of adipose tissue and lean tissue compartments within the cross section. The average muscle and adipose cross-sectional areas for all eight scans were calculated in baseline and postexercise images.

Before and at the end of training, macronutrient and energy intakes were estimated from 3-day diet records that were analyzed by a research dietician. Nutritionist IV software was used to calculate energy, protein, carbohydrate, fat, and alcohol intake.

Statistics. Means ± SE are reported. Two-tailed, paired *t*-tests were used to compare baseline to *week 16* measurements. Linear regression analysis was used to examine the relationship between exercise-induced changes in body composition and fasting serum triglyceride concentrations. A P value <0.05 was accepted as statistically significant.

RESULTS

On the basis of the highest weight documented in the past year, weight loss was small in these subjects. On enrollment, average weight was −4 ± 2% of the highest weight in the previous year. At baseline, trunk adiposity was present in 14 of 18 subjects. Their baseline body fat was 19 ± 1% of their weight, but they had 50% more adipose tissue on their trunk than on their arms and legs (Table 1). Healthy, lean seronegative subjects have 15–28% less adipose tissue on their trunk than on their arms and legs (23).

The trunk-to-appendicular adipose ratio was elevated at baseline, and it was not reduced after exercise training (Table 3). Whole body, trunk, arm, and leg adiposity were not reduced after exercise (Tables 3 and

Table 2. *Maximum voluntary muscle strength improvements after exercise*

Measurement	HIV-Positive	HIV-Negative
Chest press	23 ± 3	43 ± 6
Shoulder press	28 ± 6	53 ± 6
Leg press	34 ± 6	26 ± 4
Knee extension		
Isotonic	38 ± 7	63 ± 10
Isokinetic	11 ± 2	17 ± 3
Isometric	15 ± 3	14 ± 4
Knee flexion		
Isotonic	34 ± 6	47 ± 8
Isokinetic	17 ± 2	15 ± 2
Isometric	18 ± 2	20 ± 2

Values are means ± SE and represent % increase from baseline. One-repetition maximum (1 RM) data from HIV-negative subjects were reported previously (27) but were not collected on the identical exercise devices used in the present study. For this reason, statistical comparisons were not considered appropriate. Isotonic = 1 RM.

4). The exercise program increased body weight and whole body, trunk, and arm lean masses in these HIV-infected men (Table 3). After exercise, average thigh muscle cross-sectional area increased 5.0–5.2 cm^2 ($P < 0.005$). Thigh adipose tissue cross-sectional area and intramuscular adipose tissue area were not changed after exercise training (Tables 3 and 4).

The weight lifting program increased maximum voluntary muscle strength on all six exercises (range = 23–38%; $P < 0.001$). Knee extensor and flexor isokinetic (60°/s) and isometric (0°/s) muscle strength were also increased after training (range = 11–17%; $P < 0.0001$; Table 2). In general, the lower body improvements in muscle strength were similar, but the upper body improvements were less than those reported previously in HIV-negative men (27). The HIV-negative men were trained using identical progressive resistance exercise training principles, identical procedures for measuring percent improvement in 1 RM, and the identical isokinetic and isometric dynamometer for measuring percent improvement in knee flexor and extensor strength, but trained using guided-motion exercise equipment made by a different manufacturer.

Fasting serum triglyceride concentrations were reduced after the exercise training program, but fasting total, HDL, and LDL cholesterol, insulin, C-peptide, proinsulin, and glucagon concentrations were not affected (Table 5 and Fig. 1). Serum triglycerides were reduced after exercise in 11 of 18 subjects. Nine of these 11 were hypertriglyceridemic (>200 mg/dl) at baseline. The decrease in serum triglycerides was greatest in the subjects with the highest baseline triglyceride concentrations. There was no relationship between the reduction in triglycerides and baseline trunk adiposity ($r^2 = 0.05$) or the initial-to-final change in insulin concentration ($r^2 = 0.02$). Trends toward greater reductions in fasting serum triglyceride concentrations with greater increases in whole body lean mass ($P < 0.07$, $r^2 = 0.19$) and greater reductions in trunk fat ($P < 0.07$, $r^2 = 0.20$) were noted. The power (1-β) of these relationships was 0.44–0.46. Finally, weight loss in the year before enrollment was not correlated with increase in thigh muscle cross-sectional area, muscle strength improvement, or reduction in serum triglycerides.

Table 4. *Muscle, adipose tissue, and intramuscular adipose tissue cross-sectional areas measured in both thighs before and at the end of the resistance exercise training program*

	Before Exercise	After Exercise	*P* Value
Right thigh			
Muscle area, cm^2	71.3 ± 3.9	76.5 ± 4.4	0.005
Adipose area, cm^2	28.3 ± 3.1	27.1 ± 3.0	NS
Intramuscular adipose area, cm^2	5.6 ± 0.4	5.7 ± 4.5	NS
Left thigh			
Muscle area, cm^2	70.5 ± 4.2	75.4 ± 4.5	0.001
Adipose area, cm^2	28.8 ± 2.9	28.9 ± 3.1	NS
Intramuscular adipose area, cm^2	5.8 ± 0.5	6.0 ± 0.5	NS

Values are means ± SE. NS, not significant.

Macronutrient and energy intakes were similar at baseline and at the end of exercise. Baseline vs. end-of-exercise daily caloric intake (2,311 ± 182 vs. 2,646 ± 219 kcal/day), and percent of calories from carbohydrate (53 ± 2 vs. 51 ± 3%), fat (32 ± 2 vs. 33 ± 2%), protein (16 ± 1 vs. 17 ± 1%), and alcohol (0 vs. 4 ± 4 gr) were not significantly different.

DISCUSSION

In men living with HIV, 4 mo of supervised weight-lifting exercise increased thigh muscle cross-sectional area, whole body lean mass, and maximum voluntary

Table 3. *Regional lean and adipose tissue masses before and after exercise*

	Before Exercise	After Exercise	Change	*P* Value
Body weight	70.8 ± 2.5	72.2 ± 2.6	1.4 ± 0.3	0.001
Whole body lean mass	56.6 ± 1.5	58.0 ± 1.6	1.4 ± 0.4	0.005
Trunk lean mass	27.7 ± 0.7	28.3 ± 0.8	0.6 ± 0.2	0.020
Arms lean mass	6.6 ± 0.2	6.9 ± 0.2	0.3 ± 0.1	0.001
Legs lean mass	16.7 ± 0.6	17.0 ± 0.6	0.3 ± 0.2	0.097
Whole body adipose mass	14.2 ± 1.3	14.2 ± 1.3	0.0 ± 0.4	0.95
Trunk adipose mass	7.8 ± 0.9	8.0 ± 0.9	0.2 ± 0.2	0.53
Arms adipose mass	1.7 ± 0.2	1.5 ± 0.1	−0.2 ± 0.1	0.098
Legs adipose mass	3.7 ± 0.3	3.7 ± 0.4	0.0 ± 0.1	0.74
Trunk-to-appendicular adipose ratio	1.53 ± 0.16	1.61 ± 0.16	0.08 ± 0.06	0.23

Values are means ± SE in kg.

Table 5. *Serum cholesterol and counterregulatory hormone concentrations*

	Before Exercise	After Exercise
Total cholesterol, mg/dl	202 ± 10	200 ± 10
HDL cholesterol, mg/dl	37 ± 3	36 ± 2
LDL cholesterol, mg/dl	119 ± 9	126 ± 9
Insulin, μU/mL	11 ± 1	13 ± 2
C-peptide, ng/ml	1.8 ± 0.2	1.7 ± 0.2
Proinsulin, pM	12 ± 2	12 ± 1
Glucagon, pg/ml	83 ± 11	74 ± 7

Values are means ± SE; no differences after exercise.

muscle strength and reduced fasting serum triglyceride concentrations without reducing whole body or regional adiposity. We propose that an exercise-induced increase in muscle mass may enhance triglyceride clearance from the circulation in hypertriglyceridemic HIV-infected men with trunk adiposity.

We have confirmed that progressive resistance exercise training increases lean tissue mass in men infected with HIV. Using magnetic resonance imaging (MRI), we directly demonstrated that progressive resistance exercise training increased thigh muscle cross-sectional area (hypertrophy) in people living with HIV. Maximum voluntary muscle strength also improved, so it appears likely that the increased muscle cross-sectional area contributed to the increased muscle strength. The exercise-induced increases in muscle area, lean mass, and muscle strength indicate that resistance exercise training is an effective intervention for increasing lean mass and muscle strength in men living with HIV. Roubenoff et al. (17) have demonstrated that HIV-infected individuals with the most muscle wasting respond better to weight-lifting exercise training than do asymptomatic HIV-infected people. This was not observed in the present study, but recent weight loss was uncommon and quantitatively small. Roubenoff et al. (19) have also demonstrated that whole body adiposity is reduced when an aerobic exercise component is added to the resistance exercise program. Aerobic exercise was not part of the present regimen, which likely explains why adiposity was not reduced.

A portion of the increase in muscle strength was also attributable to improved neural recruitment patterns ("learning effect"). This was confirmed by the greater increase in thigh muscle strength determined by using 1 RM testing (range 23–38%) on the exercise equipment used daily, compared with that determined by using isokinetic and isometric strength testing (11–20%). In general, we noted a similar increase in exercise-induced lower body isotonic muscle strength in these HIV-infected men (34–38%) and a group of slightly younger noninfected men (26–63%) studied after a similar progressive resistance exercise training program (27). Conversely, upper body muscle strength (chest and shoulder press) improvements in the HIV-infected men (23–28%) were not as large as in noninfected men (43–53%) (27). One possible explanation for these findings is that the two studies used weight-lifting exercise equipment available from different manufacturers. Another possible explanation for the smaller increase in upper body muscle strength in the HIV-infected men is their exposure to HIV-nucleoside analog reverse transcriptase inhibitors (17 of 18 subjects), which have been associated with peripheral neuropathy (5). It is possible that peripheral neuropathy limited the ability of the motor nerves in the upper body musculature to adapt to the increased contractile demand of progressive resistance exercise training.

A novel observation is the reduction in fasting serum triglyceride concentrations at the end of the 16-wk exercise training program. This was especially evident in the subjects with baseline hypertriglyceridemia, and it tended to correlate with the exercise-induced increase in lean mass and the small change in trunk adipose mass. In this study, we cannot clearly attribute the reduction in serum triglycerides to either of these alterations in body composition. In addition, several studies found that fasting serum triglyceride levels are not reduced after progressive resistance exercise training in patients at risk for coronary heart disease (9), in sedentary men (7, 12, 24), in weight-trained athletes (8), and in elderly men and women (10). A single randomized crossover study found that resistance exercise training does reduce triglyceride levels in type 1 diabetic men (2). Combined endurance and resistance exercise training reduced serum triglycerides in sedentary hyperinsulinemic men (25).

In the present study, we cannot exclude all possible confounders (3, 9) for the reduction in triglyceride levels. The fasting blood sample for serum triglyceride determination was obtained 15–17 h after the last bout of exercise. It may reflect an acute effect of the last exercise session, but this is not a universal finding (3, 11). Several potential confounders can be excluded. A change in eating habits or nutrient composition during the 16-wk period might explain a reduction in serum triglycerides. Food intake surveys compiled at baseline and at the end of exercise training suggested that subjects did not substantially alter total energy, carbohydrate, protein, and fat intake during the training period. However, nutrient consumption on the day before blood collections was not controlled. A reduction in weight or in whole body or trunk adiposity could also

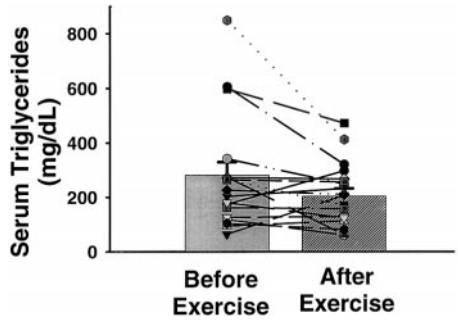

Fig. 1. Fasting serum total triglycerides were reduced at the end of the 16-wk resistance exercise training program ($P = 0.022$) (mg triglycerides/dl × 0.0113 = mM). Symbols represent individual subjects.

explain a reduction in serum triglyceride concentrations. But weight increased, whole body and regional adiposity were not significantly reduced, and these changes were not highly correlated with reductions in serum triglycerides after the exercise program. An exercise-induced increase in muscle insulin sensitivity could mediate reduced serum triglycerides, but indirect indicators of improved insulin sensitivity, i.e., fasting insulin, C-peptide, and proinsulin concentrations, were not reduced after exercise training. The reduction in triglycerides may reflect a change in disease status or treatment rather than a response to exercise training. However, this is unlikely because none of the subjects changed their medications during the course of the study. When we randomly selected from our database 28 nonexercising HIV-infected men who were stable on antiviral medications, their fasting total triglycerides were 208 ± 59 mg/dl. Twenty-four weeks later the same men had triglyceride levels of 220 ± 48 mg/dl ($P = 0.88$). Therefore, it appears that the resistance exercise-induced reduction in serum triglycerides observed in the present study is statistically, physiologically, and clinically important. At best, resistance exercise training reduced triglycerides in these hypertriglyceridemic men. At worst, resistance exercise training prevented an upward drift in triglycerides that might normally occur in nonexercising HIV-infected men.

Short-term aerobic exercise has been shown to stimulate muscle lipoprotein lipase (LPL) activity (20). LPL hydrolyzes circulating triglyceride-rich particles (LDL, VLDL) and facilitates entry of free fatty acids into muscle cells. We propose that resistance exercise and the increase in muscle contractile activity may have reduced serum triglycerides by increasing muscle LPL activity and free fatty acid availability to the muscle. This would not necessarily increase intramuscular adipose tissue cross-sectional area, and thigh muscle adipose content was not increased when measured using proton-MRI. However, the amount of intramuscular adipose tissue is very small, and MRI imaging may not be sufficiently sensitive to detect an increase in muscle triglyceride storage. Instead, we propose that muscle free fatty acid utilization, uptake, or storage as triglyceride droplets or sarcolemma membrane constituents might have been enhanced after exercise.

We conclude that a progressive resistance exercise training program increases lean mass, muscle cross-sectional area, and maximum voluntary muscle strength and reduces serum total triglycerides in HIV-infected men, especially in those with baseline hypertriglyceridemia and trunk adiposity. The trend toward a correlation between the exercise-induced reduction in serum triglyceride concentration and the significant increase in whole body lean mass supports the notion that the increase in lean tissue mass may initiate an intrinsic change in muscle that is responsible for increasing the clearance of triglycerides from the circulation. Because the metabolic complications associated with HIV infection include muscle wasting and hypertriglyceridemia, these findings suggest that progressive resistance exercise training, along with nutrition counseling, weight management, and compliance to medication regimens, be recommended to maintain effective viral suppression and manage the metabolic complications associated with HIV infection.

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