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Linda M. LeMura · Serge P. von Duvillard
Joseph Andreacci · Jodi M. Klebez
Sara A. Chelland · Joseph Russo

Lipid and lipoprotein profiles, cardiovascular fitness, body composition, and diet during and after resistance, aerobic and combination training in young women

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Abstract The purpose of this study was to evaluate the effects of various modes of training on the time-course of changes in lipoprotein-lipid profiles in the blood, cardiovascular fitness, and body composition after 16 weeks of training and 6 weeks of detraining in young women. A group of 48 sedentary but healthy women [mean age 20.4 (SD 1) years] were matched and randomly placed into a control group (CG, $n = 12$), an aerobic training group (ATG, $n = 12$), a resistance training group (RTG, $n = 12$), or a cross-training group that combined both aerobic and resistance training (XTG, $n = 12$). The ATG, RTG and XTG trained for 16 weeks and were monitored for changes in blood concentrations of lipoprotein-lipids, cardiovascular fitness, body composition, and dietary composition throughout a 16 week period of training and 6 weeks of detraining. The ATG significantly reduced blood concentrations of triglycerides (TRI) ($P < 0.05$) and significantly increased blood concentrations of high-density lipoprotein-cholesterol (HDL-C) after 16 weeks of training. The correlation between percentage fat and HDL-C was 0.63 ($P < 0.05$), which explained 40% of the variation in HDL-C, while the correlation between maximal oxygen uptake ($\dot{V}O_{2max}$) and HDL-C was 0.48 ($P < 0.05$), which explained 23% of the variation in HDL-C. The ATG increased $\dot{V}O_{2max}$ by 25% ($P < 0.001$) and decreased percentage body fat by 13% ($P < 0.05$) after 16 weeks. Each of the alterations in the ATG had disappeared after the 6 week detraining period. The concentration of total cholesterol (TC), TRI, HDL-C and low density lipoprotein-cholesterol in the blood did not change during the study in RTG, XTG and CG. The RTG increased upper and lower body strength by 29% ($P < 0.001$) and 38%, respectively. The 6 week detraining strength values obtained in RTG were significantly greater than those obtained at baseline. The XTG increased upper and lower body strength by 19% ($P < 0.01$) and 25% ($P < 0.001$), respectively. The 6 week detraining strength values obtained in XTG were significantly greater than those obtained at baseline. The RTG, XTG and CG did not demonstrate any significant changes in either $\dot{V}O_{2max}$, or body composition during the training and detraining periods. The results of this study suggest that aerobic-type exercise improves lipoprotein-lipid profiles, cardiorespiratory fitness and body composition in healthy, young women, while resistance training significantly improved upper and lower body strength only.

Key words Aerobic exercise · Blood lipid concentrations · Body composition · Detraining · Resistance training

S. P. von Duvillard (✉)
Human Performance Laboratory,
Department of Physical Education and Exercise Science,
University of North Dakota,
P.O. Box 8235, Hyslop Sports Center,
Grand Forks, ND, USA
e-mail: vonduvil@badlands.nodak.edu
Tel.: +1-701-777-4351

L. M. LeMura · J. Andreacci · J. M. Klebez
S. A. Chelland · J. Russo
Human Performance Laboratory,
Bloomsburg University of Pennsylvania,
Bloomsburg, PA, USA

Introduction

The salutary effects of exercise in the primary and secondary treatment of cardiovascular disease in men and women are well known. Cross-sectional studies of the relationship between high levels of fitness and selected cardiac risk factors have demonstrated a more favorable risk profile in fit women (Blair et al. 1989; Powell et al. 1987). However, strong evidence resulting from controlled studies on the beneficial effects of exercise on the reduction of risk factors is lacking. There remains a need for additional research on the usefulness of chronic exercise, particularly among women.

There are significant questions regarding the effects of type, duration, and frequency of training on various risk factors for cardiovascular disease. For example it is unclear how much and what type of exercise is required to induce lipoprotein-lipid changes in women and whether exercise must be aerobic or may be a form of resistance training also requires clarification since the results from training studies to date are far from conclusive. Although a plethora of studies exist which have suggested a relationship between physical training and a favorable lipoprotein-lipid profile in men and women (Hurley 1989), the results from resistance training studies have been conflicting. Some investigators have reported no substantive changes in lipoprotein-lipid profiles after resistance training (Manning et al. 1991; Kokkinos et al. 1988), while others have reported increases in HDL-C ranging from 10% to 15% (Goldberg et al. 1984) and decreases in LDL-C ranging from 5% to 39% (Hurley et al. 1988). These changes are comparable in magnitude to those reported in aerobic training studies; however, many of these studies have been criticized for methodological flaws or design limitations that make the results somewhat questionable. These flaws have included the lack of a separate, inactive control group, no dietary control, the use of only one blood sample to establish baseline values or investigators not eliminating the possible acute effects from the last training session, and anabolic steroid use (Hurley 1989). Each of these design and methodological issues preclude a clear understanding of the impact of training on lipoprotein-lipid profiles.

Therefore, the purpose of this investigation was to evaluate the effects, of a variety of training methods on the time course of changes in lipoprotein-lipid profiles, cardiovascular fitness, and body composition in sedentary, and otherwise healthy young women. To achieve this objective, we studied 48 women over 16 weeks who were members of one of four groups: (1) aerobic training (ATG), (2) resistance training (RTG), (3) combination cross-training (XTG), or (4) control (CG). The results of this investigation will elucidate the optimal training modality that may be recommended to control selected risk factors for cardiovascular disease in young women.

Methods

Subjects, baseline tests, and experiment design

A group of 48 healthy, sedentary college-age women [mean age 20.4 (SD 1) years] volunteered to be subjects in this study. The subjects were students of Bloomsburg University of Pennsylvania recruited by placing an advertisement in the University newspaper. After receiving instructions regarding the purpose and procedures of the investigation, the subjects provided written informed consent. None of the subjects had been involved in regular physical activity for a period of 4 months before the study, and each subject was screened by using a health history questionnaire, and taking a resting blood pressure and an electrocardiogram. The subjects did not receive any remuneration for their participation. This study received the approval of the Institutional Review Board of Bloomsburg University.

Maximal oxygen consumption

To establish baseline fitness values, each subject completed a maximal exercise test on a motor-driven treadmill before the study to determine maximal oxygen consumption ($\dot{V}O_{2max}$). The maximal exercise test protocol consisted of stages of increasing intensity until exhaustion was reached. The test increased speed and gradient to elicit $\dot{V}O_{2max}$. in approximately 10 min. Expired gases were obtained using a Beckman Metabolic Cart (Beckman Instruments, Fullerton, Calif.) that was calibrated with gases of known concentrations before each test. A *true* $\dot{V}O_{2max}$ was identified as an increase in exercise intensity that was not accompanied by an increase in oxygen uptake of greater than 150 ml $\cdot$ min^{-1} or 2.1 ml $\cdot$ kg^{-1} $\cdot$ min^{-1} and a respiratory exchange ratio value at test termination in excess of 1.10 as has been suggested by Taylor et al. (1955). All subjects met these criteria and were included in the analyses. Heart rate (HR) was monitored continuously throughout the test (Polar Vantage XL, Stamford, Conn.). The maximal exercise test was repeated in all subjects at week 8, week 16, and 6 weeks after the last training session.

The experiment design of this study was a before matched, after four-group design. The $\dot{V}O_{2max}$ scores were placed in rank order from the highest $\dot{V}O_{2max}$ to the lowest. Each $\dot{V}O_{2max}$ score was then randomly assigned until all of the subjects were placed into one of four treatment groups. The treatment groups included: (1) RTG ($n = 12$), (2) ATG ($n = 12$), (3) XTG (training consisted of both aerobic and resistance training) ($n = 12$) and, (4) CG ($n = 12$). None of the subjects reported using any medications known to alter lipid metabolism, including oral contraceptives. All of the subjects were non-smokers. The physical characteristics of the subjects assigned to each group are displayed in Table 1.

Body composition

Body composition was estimated from determination of body density by weighing underwater. The underwater mass recorded was the mean of the three highest mass values obtained, reproducible within 30 g, from ten trials. Underwater mass was adjusted for residual lung volume using the oxygen dilution method as has been described by Wilmore et al. (1980) immediately after underwater weighing while the subject was still in the water. Percentage body fat was estimated from body density values using the equation of Brozek et al. (1963). To further describe the physical characteristics of our subjects, body mass was measured using a balance beam scale with the subjects in light clothing, height was measured using a medical grade stadiometer, and body mass index was calculated using mass (kg)/stature (m^2). The measurements of body composition were repeated at week 8, week 16, and 6 weeks after the last training session.

Lipoprotein/lipid analyses

After a 12–14 h overnight fast, a 50 ìl blood sample was taken, on 2 separate mornings less than 1 week apart, using a finger stick and assayed for concentrations of total cholesterol (TC), triglyceride (TRI), and high density lipoprotein cholesterol (HDL-C). If the values for either TC or HDL-C differed by more than 7%, a third sample was taken on another day. The baseline value reported is the average of these determinations. Low density lipoprotein cholesterol (LDL-C) was calculated using the equation: LDL-C = TC − (HDL-C + TRI/5), since plasma TRI concentrations were less than 400 mg $\cdot$ dl^{-1} in all subjects (Lipid Research Clinics Program 1974). The analyses of TC, TRI and HDL-C were conducted using a Kodak Ektachem DT60 (Rochester, N.Y.), which operates using the principles of thin-film reflectance spectrophotometry. Each of the reactions that occurred using these methods have been validated and described elsewhere (Shirey 1983; Spayd 1978). Determinations of lipid and lipoprotein concentrations were made before the study began and at weeks 8 and 16. At the conclusion of the 16 week training period, blood samples were taken 1 day [21 (SD 3) h] and 2 days [47 (SD 6) h] after the last exercise

Table 1 Physical characteristics of the training and control groups. *BMI* Body mass index, $\dot{V}O_{2max}$ maximal oxygen uptake

Variable	Baseline		Week 8		Week 16		6 weeks post		Baseline		Week 8		Week 16		6 weeks post	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Resistance training group (n = 11)									Aerobic training group (n = 10)							
Age (years)	20.0	1.0							21.0	2.0						
Height (cm)	162.5	4.0							165.1	3.8						
Body mass (kg)	63.6	4.5	64.0	4.3	64.5	4.5	63.4	4.3	62.7	3.9	61.9	3.9	59.0	4.0	61.9	3.7
Fat (%)	24.8	3.0	23.1	3.3	22.2	3.4	23.0	2.9	26.4[a]	2.9	24.1	3.1	22.9[a,b]	2.7	24.8[b]	2.9
Fat free mass (kg)	48.1	3.8	50.1	3.9	50.3	4.1	48.5	3.9	45.4	4.1	47.8	4.1	46.1	3.8	47.0	3.4
BMI ($kg \cdot m^{-2}$)	24.1	2.4	24.3	2.2	24.3	2.1	24.0	1.9	22.9	2.2	22.7	2.0	21.6	2.1	22.8	1.9
$\dot{V}O_{2max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	32.6	4.1	31.0	3.3	33.1	3.8	33.6	2.9	33.5[a]	4.4	38.0	3.8	42.2[a,b]	4.5	33.6[b]	2.8
Cross training group (n = 12)									Control group (n = 12)							
Age (years)	19.0	2.0							20.0	1.0						
Height (cm)	167.6	4.1							164.9	4.3						
Body mass (kg)	58.6	3.7	58.9	3.5	59.0	3.9	57.5	3.7	59.5	4.1	60.1	4.1	59.8	3.6	59.0	3.8
Fat (%)	28.0	2.9	26.5	3.1	25.1	3.1	26.5	3.3	27.9	2.8	27.0	2.9	28.1	2.7	26.8	2.7
Fat free mass (kg)	42.9	2.9	43.8	3.1	44.1	3.1	42.2	3.3	43.4	3.2	44.3	3.3	43.1	3.9	42.5	3.8
BMI ($kg \cdot m^{-2}$)	20.8	2.1	21.0	2.1	21.0	1.8	20.5	2.2	21.8	2.3	22.0	1.7	21.9	1.9	21.6	2.2
$\dot{V}O_{2max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	36.9	3.7	37.4	3.1	39.1	4.1	34.6	3.7	33.3	2.7	34.1	2.1	33.5	3.1	33.1	2.8

Means followed by a common letter are different ($P < 0.05$): [a] different from baseline to week 16, [b] different from week 16 to 6 weeks post

session in the training groups. A blood sample was also taken 6 weeks after the 16 week training period.

Dietary record

Diets were recorded for 7 days prior to pre-training blood sampling and repeated for a week prior to the 8 week and 16 week blood samples. Dietary records were also obtained for 1 week before the end of the 6 week detraining period. The subjects were periodically reminded not to change their dietary habits and were instructed by a registered dietitian on appropriate daily recording techniques to ensure accuracy. The subjects were asked to abstain from alcohol consumption for 5 days before blood sampling. Mean energy content (kilojoules), percentage of energy derived from carbohydrates, proteins, and fats were computed using the EVERYDIET computer program (Gargiulo 1983).

Assessment of physical activity during detraining

At the beginning of the 6 week detraining period, the subjects were reminded not to engage in regular physical exercise. During the 6 week detraining period, a physical activity profile was obtained from each subject using the 7 day Physical Activity Recall (PAR) that has been described by Sallis et al. (1985). The PAR contained a list of 23 popular activities with provision for adding activities that were more unusual. The subjects were instructed to record the duration and nature of activities in which they had participated during the previous week. Metabolic equivalents were then ascribed to each physical activity item on the list as described by Wilson et al. (1986). A trained interviewer reviewed the physical activity profile at the end of each week with every subject for the 6 week detraining period to determine the quantity of physical activity that had occurred after the 16 weeks of formal training. The interviewer asked questions to ascertain time spent in physical activity, strength, and flexibility activities during the 7 days before the interview. The CG was also requested to complete the questionnaire during the detraining period to ensure that none of the subjects in that group had begun exercising regularly.

Attrition

Of the original 48 subjects 44 have been included in the data analysis, 4 subjects being excluded due to noncompliance with the arrangements for the study.

Treatment groups

Resistance training group

The subjects assigned to RTG trained on Nautilus machines three times a week for 16 weeks on nonconsecutive days. Each subject was shown the proper technique to use by a qualified instructor and underwent preliminary tests to establish a one-repetition maximum (1 RM). The first 2 weeks of tests involved two sets of 8–10 repetitions at 60%–70% of 1 RM, and increased to three sets per training session during the next 14 weeks. During each session, the subjects performed the following exercises: leg extension, leg press, leg curl, triceps extension, biceps curl, chest press, decline press, deltoid lateral raise, behind the neck pullover, pectoral adduction and abdominal crunches. The concentric phase of the contraction was performed for 2 s and the eccentric phase for 4 s. The rest interval between sets was between 50 and 60 s. The performance: rest ratio was monitored by experienced instructors. The 1 RM were obtained at 4 week intervals so that the weights could be adjusted throughout the 16 week training period. Post-training 1 RM values were obtained 1–3 days after the last training session and at the conclusion of the 6 week detraining period.

Aerobic training group

The subjects assigned to ATG initially trained three times a week for 16 weeks on nonconsecutive days. Each training session included a 10 min warm-up, 30 min of continual exercise, and a 10 min cool-down period. The exercise intensity during the endurance component of the training session corresponded to a HR between 70% to 75% of the maximal heart rate (HR_{max}) obtained during the maximal exercise test. The subjects selected their preferred endurance exercise from one of the following activities: cycling ergometry, rowing ergometry, and treadmill walking/jogging. The subjects wore a HR monitor (Polar Vantage XL, Stamford, Conn.) for the duration of the endurance component to ensure that the appropriate intensity was maintained. The maximal exercise test conducted at week 8 was used to document any changes in the cardiovascular parameters that could result in adjusting the subjects' exercise prescription. The adjustments that were made included an increased training duration (up to approximately 45 min), an increased training intensity (up to approximately 85% of HR_{max}), and an increased training frequency up to four times a week.

Cross-training group

The subjects assigned to XTG performed a combination of aerobic training and resistance training each twice a week for 16 weeks on alternating days. For the aerobic component, the subjects were assigned an exercise prescription based upon the training principles applied to the ATG, with the only difference being that aerobic training was conducted 2 days a week. For the resistance training component, the subjects were assigned a training prescription in a manner similar to RTG, but again, trained 1 day less a week. The 1 RM tests were conducted at 4 week intervals.

Control group

The subjects assigned to CG were asked not to engage in any regular training during the 16 week training period and during the detraining period. To ensure that the CG subjects did not begin exercising regularly during the course of the study they were requested to complete an activity log and to submit it for review weekly. Each of the subjects in CG complied with this request. They experienced the same measurement schedule as the subjects assigned to the treatment groups for the dependent variables lipoprotein-lipids, $\dot{V}O_{2max}$, body composition, and strength.

Statistical procedures

Determination of the effects of the training methods on $\dot{V}O_{2max}$, body composition, lipids and lipoproteins was conducted by analyzing the differences between the baseline, midtraining, posttraining, and 6 week post training group mean values. This analysis was accomplished with a four (treatments) × four(groups) mixed, one between, one within, analysis of variance (ANOVA) for repeated measures. After an analysis of simple main effects, Tukey's post-hoc test was employed to locate specific differences. Statistical power calculations demonstrated power ranges in this investigation from 0.80 to 0.84. The relationship between changes in selected variables was made by using simple regression. All analyses were performed using the Sigma Stat 4.0 statistical package. An alpha of $P < 0.05$ was established a priori for all significant main effects.

Results

Statistical analysis of daily nutrient intake for each of the four groups revealed no significant differences among the groups in total kilojoules per day, or in the total percentage contribution of protein, fat, and carbohydrate to daily energy (kilojoules) consumed.

The 16 week training program resulted in a significant increase (25%) in relative $\dot{V}O_{2max}$ (millilitres per kilogram per minute) in the ATG, only. The mean $\dot{V}O_{2max}$ in the ATG was significantly reduced from week 16 to the 6 week detraining period to a level that was nearly identical to the mean baseline value. Additional analyses of the changes in $\dot{V}O_{2max}$ in RTG and XTG revealed that the training stimulus in these groups was insufficient to result in any significant alterations.

The time course changes in body composition showed a significant decrease in the percentage of body fat (13.2%; $P < 0.05$) after 16 week in ATG, only. These changes diminished during the detraining period. The RTG reduced body fat (11%) and increased fat free mass (4%); however, these changes returned to baseline values during the detraining period. Participation in XTG resulted in a decrease in mean body fat (10%), and an increase in fat free mass (3%). The body composition measures in XTG also returned to baseline values during the detraining period. The summary of these changes is shown in Table 1.

The results of the 1 RM strength tests in RTG resulted in a 29% increase in strength ($P < 0.001$) in the upper body, and a 38% increase in strength in the lower body ($P < 0.0001$) after 16 week of training. Significant increases of 17% and 20% for upper and lower body strength, respectively, were also observed at week 8 ($P < 0.001$). Strength tests at the conclusion of the detraining period revealed 19% and 24% increases in upper and lower body strength over baseline values, respectively. These increases remained significantly different from those obtained at baseline.

The XTG had experienced a 19% increase in strength after 1 RM ($P < 0.01$) upper body tests and a 25% increase in lower body strength ($P < 0.001$) after 16 week of training. Significant increases were experienced in XTG after 8 weeks of training in the upper body (12%) and lower body (18%) ($P < 0.01$). These changes were reduced to 13% and 18% increases in upper and lower body strength, respectively, at the conclusion of the detraining period. These detraining strength values remained significantly different from those obtained during the baseline tests ($P < 0.01$). No strength changes were observed in either ATG or CG.

The concentrations of TC, TRI, LDL-C, HDL-C and the TC:HDL-C ratio showed no significant changes over the duration of the study for RTG, XTG or CG. A significant decrease in TRI was found after 16 weeks of endurance training in ATG, but not after 8 weeks ($P < 0.05$). Although the HDL-C values had increased in the ATG after 8 weeks of training, significant increases were observed after 16 weeks ($P < 0.01$). The HDL-C value at week 16 represented a 28% increase over baseline values. At the conclusion of the 6 week detraining period, the mean HDL-C had returned to baseline values. Significant correlations were observed between changes in percentage body fat and $\dot{V}O_{2max}$ and HDL-C when combined among groups ($n = 44$). The correlation between percentage fat and HDL-C was 0.63 ($P < 0.05$) and accounted for 40% of the variation in HDL-C, while the correlation between $\dot{V}O_{2max}$ and HDL-C was 0.48 ($P < 0.05$) and accounted for 23% of the variation in HDL-C after 16 weeks. The TC was reduced by 10% ($P < 0.07$) and LDL-C by 6% in ATG. There were no differences in the magnitudes of changes in any of the lipoprotein-lipid profiles among groups. The values for the 1–2 day post 16 week sampling were no different from the 16 week values; thus, we concluded that there were no acute exercise effects from the last training session in any of the groups. The changes in the mean values for each of lipoprotein-lipid variables are found in Table 2, and the percentage change for each variable is shown in Fig. 1.

The activity logs completed by the subjects in CG for the duration of the 16 week training portion of this study revealed that none of the subjects had begun to exercise

Table 2 Changes in lipid and lipoprotein concentrations during the four protocols measured during the study. *RTG*, *ATG*, *XTG* Resistance ($n = 11$), aerobic ($n = 10$), and combined aerobic/resistance ($n = 11$) training groups, respectively, *CG* control group ($n = 12$), *TC* total cholesterol, *HDL-C*, *LDL-C* high and low density lipoprotein-cholesterol concentrations, respectively, *TRI* triglyceride concentrations

Variable	Group	Baseline		Week 8		Week 16		1–2 day post		6 weeks post	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
TC (mmol · l^{-1})	RTG	5.2	0.3	5.1	0.3	5.2	0.3	5.2	0.4	5.1	0.4
	ATG	5.1	0.4	4.9	0.3	4.8	0.4	4.7	0.4	5.1	0.4
	XTG	4.9	0.3	4.9	0.4	4.7	0.3	4.6	0.1	4.9	0.4
	CG	5.0	0.4	5.0	0.4	5.1	0.3	5.0	0.4	5.2	0.3
HDL-C (mmol · l^{-1})	RTG	1.5	0.1	1.5	0.1	1.5	0.1	1.4	0.1	1.5	0.1
	ATG	1.4[a]	0.1	1.5	0.1	1.8[a,b]	0.1	1.7	0.1	1.4[b]	0.1
	XTG	1.5	0.1	1.5	0.1	1.4	0.0	1.5	0.1	1.5	0.1
	CG	1.4	0.1	1.5	0.1	1.4	0.1	1.5	0.1	1.5	0.1
LDL-C (mmol · l^{-1})	RTG	3.1	0.2	3.1	0.2	3.0	0.1	3.1	0.2	3.0	0.2
	ATG	2.6	0.1	2.4	0.0	2.4	0.1	2.4	0.0	2.5	0.0
	XTG	3.1	0.2	3.1	0.1	2.9	0.2	2.9	0.0	3.1	0.2
	CG	3.2	0.2	3.1	0.2	3.1	0.1	3.1	0.2	3.3	0.0
TRI (mmol · l^{-1})	RTG	1.4	0.1	1.4	0.0	1.4	0.0	1.3	0.1	1.4	0.0
	ATG	1.4[a]	0.1	1.3	0.1	1.2[a,b]	0.1	1.2	0.1	1.3[b]	0.1
	XTG	1.4	0.0	1.3	0.1	1.3	0.0	1.3	0.1	1.3	0.1
	CG	1.2	0.0	1.2	0.0	1.2	0.0	1.2	0.1	1.3	0.1
TC/HDL-C	RTG	3.5	9.0	3.4	6.0	3.6	1.0	3.6	8.0	3.7	4.0
	ATG	3.7	7.0	3.3	1.0	2.7	5.0	2.7	7.0	3.7	2.0
	XTG	3.3	4.0	3.2	7.0	3.2	3.0	3.1	9.0	3.2	7.0
	CG	3.6	3.0	3.4	6.0	3.5	8.0	3.4	1.0	3.5	6.0

Means followed by a common letter are different ($P < 0.05$): [a] different from baseline to week 16, [b] different from week 16 to 6 weeks post

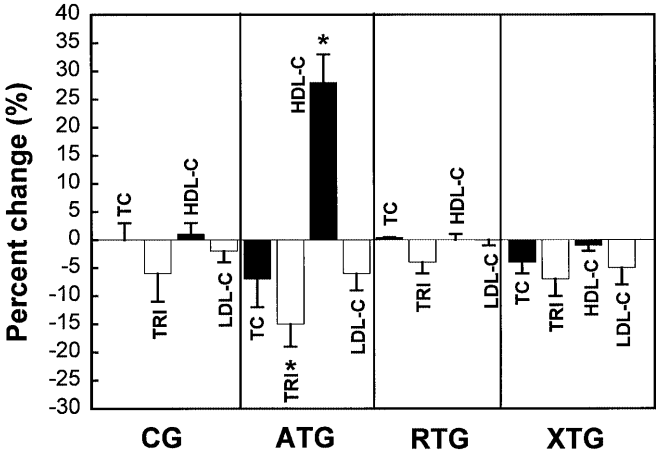

Fig. 1 Percentage change in lipid and lipoprotein concentrations for all groups after 16 weeks of training. *CG* control group, *ATG* aerobic training group, *RTG* resistance training group, *XTG* cross-training group, *TC* total cholesterol, *TRI* triglycerides, *HDL-C* high density lipoprotein-cholesterol, *LDL-C* low density lipoprotein-cholesterol. Values are mean and SD. *$P < 0.05$, **$P < 0.01$ compared to baseline

regularly. The PAR completed by subjects in the training groups indicated that all of the subjects ceased regular physical training and that CG remained inactive for the duration of the 6 week detraining portion of the study.

Discussion

The design of this investigation has allowed us to offer unique observations regarding the time course changes in cardiovascular fitness, body composition, blood lipids and lipoproteins during 16 weeks of training and through a period of detraining in apparently healthy and previously sedentary young female participants. The absence of a carefully monitored control group, or unknown dietary changes have been identified as serious criticisms of previous studies (Kokkinos and Hurley 1990).

The ATG demonstrated a significant increase in relative $\dot{V}O_{2max}$ (millilitres per kilogram per minute) at week 16. Perhaps equally important, the 25% increase in $\dot{V}O_{2max}$ in ATG was completely eliminated by the end of the 6 week detraining period. Although we did not attempt to measure directly the mechanisms responsible for the rapid decline in the observed gains in $\dot{V}O_{2max}$, the explanations are most likely similar to those often cited in the literature. They have included a reduction in plasma and stroke volume (Coyle et al. 1986), oxidative enzymes (Coyle et al. 1984), and muscle glycogen content (Costill et al. 1985a). We observed a 6% increase in $\dot{V}O_{2max}$ in XTG after 16 weeks. Thus, it is clear that the training stimulus from the aerobic component of the XTG training regimen was insufficient insofar as being able to produce significant changes in $\dot{V}O_{2max}$. The RTG did not produce changes in $\dot{V}O_{2max}$ during any point of the training or detraining periods of the investigation.

The women assigned to RTG significantly improved strength over the course of the 16 week training period. They had retained 35% of upper body strength and 31% of lower body strength 6 weeks after the completion of the formal training period. The XTG which strength-trained only twice a week also showed significant upper and lower body improvements at week 8 and week 16,

and had retained 32% of upper body strength and 28% of lower body strength at the conclusion of the detraining period. However, it has been established that when training ceases, the frequency of neurological stimulation is reduced and normal fiber recruitment is disrupted (Costill et al. 1985a). Hence, part of the strength loss associated with detraining is likely to have been related to the inability to activate some muscle fibers. Research has also indicated that the strength losses after detraining are relatively small during the first few weeks in comparison to the losses observed in cardiorespiratory endurance after aerobic training (Costill et al. 1985b). After detraining, it has been reported that an individual can retain significant percentages of gained muscle strength for periods up to 6 weeks. Thus, our observations in strength changes during and after training are in agreement with those reported in the literature.

Several cross-sectional investigations have demonstrated that aerobic-type exercise training improves lipoprotein-lipid profiles for both normolipidemic (Dufaux et al. 1982) and hyperlipidemic subjects (Superko and Haskell 1987). Many of these improved profiles have been reflected by lower plasma concentrations of TRI and increased HDL-C concentrations. The results of longitudinal studies, however, have been far less convincing. Although some aerobic training programs have resulted in significant reductions in the concentration of plasma TRI and elevations in HDL-C in healthy men and women (Seals et al. 1984) and in patients with coronary artery disease (Heath et al. 1983), no consistent lipid lowering effects have been reported for either TC or LDL-C in women in comparison to untrained controls. Our data indicate that aerobic exercise, which allows for modifications in intensity and duration over the course of 16 weeks of training significantly reduces TRI and significantly increases HDL-C in young women. These observations were made in the absence of dietary changes during the course of training, and with the presence of a carefully monitored CG. In an attempt to understand more fully the stimulus responsible for the positive alterations in the lipoprotein-lipid profiles in the ATG, we also carefully assessed changes in body composition of our subjects. Conflicting evidence exists regarding the relationship among exercise training, body fat, TC and LDL-C. Kraemer et al. (1997) reported that loss of body mass after exercise resulted in lower concentrations of TC, HDL-C and LDL-C. In contrast, Hurley et al. (1988) reported reductions in concentrations of TC, LDL-C and an increase in HDL-C that were independent of changes in body composition. Our data indicated that a significant loss of body fat was not associated with any concomitant reductions in concentrations of TC and LDL-C. The results of a meta-analysis on the effects of exercise on lipoprotein concentrations seen with changes in body mass (Tran and Weltman 1985) has revealed that reductions in lipoprotein-lipid concentrations occurred more frequently when exercise was combined with body fat loss but could occur when there was no change in body mass. It remains unclear if a minimum threshold of energy expenditure is required to stimulate significant reductions in concentrations of TC and LDL-C. With regard to the reduction in TRI concentration, regular exercise is known to increase amounts of lipoprotein lipase (LPL) in adipose and muscle tissue. This is an enzyme that facilitates clearance of circulating TRI. Since we did not measure LPL activity, we can only speculate that this mechanism was responsible for the reduction of TRI in ATG.

In further support of this relationship, there were no changes in HDL-C concentration or in percentage body fat after only 8 weeks of training. The significant correlation between the change in body fat and HDL-C concentration would indicate that the greatest increases in HDL-C concentration were achieved in those who decreased body fat to the greatest extent. We did not measure the activity of lecithin cholesterol acyl transferase (LCAT), the enzyme that catalyzes intravascular esterification of cholesterol in HDL-C. Because LCAT has been shown to increase in regular exercisers, it may have, in part, explained the increase in HDL-C in ATG. In addition, results of studies of the effect of exercise in women can also be influenced by the timing of the lipid tests since there have been found to be variations in HDL-C concentrations related to phases of the menstrual cycle (Krummel et al. 1993). Finally, although we demonstrated a relationship between percentage body fat and HDL-C concentrations in the subjects in our ATG, we cannot eliminate the possibility that the cardio-protective effects of estrogen were operative in maintaining these variables within a desirable range as Toth and Poehlman (1995) have found.

An additional important finding in this study was that the lipoprotein-lipid variables in ATG had returned to baseline values at the conclusion of the detraining period. A limitation of our data is that we cannot describe any changes that may have occurred in any of the HDL-C subfractions. Notwithstanding, these data provide strong evidence of the effects of aerobic training and indicate that the positive results may disappear in as few as 6 weeks of detraining.

The research on the effects of resistance training on lipoprotein-lipid profiles has been far less extensive than comparable studies using aerobic-type training. The number of reports on the effects of resistance exercise began to proliferate in the 1990s; however, the results from these studies preclude conclusive findings because of a variety of design limitations (Kokkinos and Hurley 1990). Kokkinos et al. (1991) have concluded that 20 weeks of strength training in middle-aged men with elevated baseline lipoprotein-lipid concentrations was not associated with significant changes in TC, TRI, HDL-C, LDL-C, LPL activity, hepatic lipase activity, $\dot{V}O_{2max}$ (millilitres per kilogram per minute), or body composition. In an earlier report, Kokkinos et al. (1988) have studied the effects of low and high repetition resistance training on lipoprotein-lipid variables in healthy, untrained males [mean age 21 ($\pm$1) years].

They have reported that 10 weeks of low-repetition resistance training resulted in no significant changes in plasma concentrations of TC, TRI, HDL-C, the subfraction HDL-C_2, or LDL-C. A similar profile was observed for the high-repetition group. They speculated that the absence of significant findings could have had several explanations, including:

1. A lack of increase in $\dot{V}O_{2max}$
2. Potential dietary changes
3. A low total cost of energy expenditure of both the low and high-resistance training groups
4. The age and low initial body fat of the subjects.

In contrast, Johnson et al. (1982) have found significant decreases in serum concentrations of TC and LDL-C, and a significant increase in HDL-C, but no change in TRI after 12 weeks of resistance training in middle-aged men. Major criticisms of their work were failing to take account of changes in body fat and for using only one blood sample to establish baseline values. In one of the few investigations that has analyzed sex differences, Goldberg et al. (1984) strength-trained 8 women (mean age 27 years) and 6 men (mean age 33 years) for 16 weeks and reported significant reductions in TC, TRI, and LDL-C, and a significant increase in HDL-C concentrations. However, a control group was not included, and the investigators measured neither body composition nor dietary changes. Thus, it is difficult to make conclusions about the independent effect of the training program on the lipid-lipoprotein variables in their study. More recently, Boyden et al. (1993) studied 88 premenopausal women with normal baseline serum lipid concentrations who strength-trained for 5 months. They reported significant reductions in TC and LDL-C concentrations in the exercise group, but no differences in either HDL-C or TRI concentrations. The changes in TC and LDL-C were not correlated with changes in body composition.

The 16 week training program in our RTG and XTG did not result in any significant changes in any of the lipoprotein-lipid variables. These results were not explained by changes in diet, and there were no significant alterations in percentage body fat, fat free mass or $\dot{V}O_{2max}$ during the training and detraining periods. The lack of changes in the lipoprotein-lipid variables was most likely attributable to the type and intensity of training completed by the subjects assigned to these groups. As much as 50% of the fat that is oxidized with exercise comes from the free fatty acids liberated from adipocytes by the hydrolysis of TRI by hormone-sensitive LPL. With sustained exercise of low-to moderate intensity, lipids may account for as much as 90% of oxidative metabolism. With higher intensity exercise such as the type performed in XTG and RTG, (more than 70% $\dot{V}O_{2max}$), glycogen is the primary fuel source. Thus, the exercise completed by RTG and XTG may not have resulted in the adaptations that lead to improved fat utilization during exercise. These adaptations have been described to include:

1. Increased epinephrine-stimulated hydrolysis from subcutaneous fat
2. An increase in the capacity of the trained muscle to oxidize lipids
3. Increased hydrolysis of TRI within the trained muscle
4. Increased hydrolysis of circulating TRI through LPL activity
5. Decreased insulin concentration, an inhibiting factor to lipid mobilization (DiPietro 1995).

The results of this study indicate that aerobic-type exercise provides the necessary stimulus to alter lipoprotein-lipid profiles of healthy and initially sedentary young women. In further support of the influence of aerobic exercise training on lipid-lipoprotein variables, all of the positive alterations in lipid-lipoprotein concentrations, body composition, and $\dot{V}O_{2max}$ experienced in ATG were eliminated after 6 weeks of detraining. In contrast, resistance training did not appear to change lipids in any appreciable way, and combination training that included a 2 days a week aerobic training component was inadequate to alter either lipoprotein concentrations or $\dot{V}O_{2max}$ in young women. We demonstrated significant increases in upper and lower body strength in RTG and XTG, respectively, and that strength remained significantly higher than baseline values even after a 6 week detraining period.

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