THE EFFECT OF AN ENDURANCE AND WEIGHT TRAINING PROGRAM ON PLASMA TOTAL CHOLESTEROL AND HIGH-DENSITY LIPOPROTEIN-CHOLESTEROL

by

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(ABSTRACT)

Research has reported that increased levels of plasma TC are directly related, while low levels of plasma HDL-C are inversely related, to coronary heart disease. Regular physical exercise has been suggested as a method for reducing plasma TC and increasing plasma HDL-C. Thirty-one healthy, sedentary women (ages 18-30) were studied to determine the effects of a jogging, weight training, or a combined jogging and weight training program on plasma total cholesterol, high-density lipoproteins, and body composition. Experimental subjects were randomly assigned to the treatment conditions. The subjects trained three days a week for nine weeks. The R group ran for 30 minutes a session at 75% predicted maximum HR. The W group trained with weights utilizing exercises to strengthen all major muscle groups for one hour at 60% one repetition maximum the first 3 weeks and 75% one repetition maximum weeks 4 - 9. The RW group ran for 25 minutes a session at 75% predicted maximum HR, then lifted weights using the leg-strengthening exercises for 30 minutes, similar to the W group. Preceding and following the treatment period, plasma TC, HDL-C, body weight, and percent body fat was assessed for all four groups. Plasma TC was not significantly altered, although a downward trend was observed for all three treatment groups. Plasma HDL-C did not change over the treatment period for any group. The plasma TC/HDL-C ratio changed significantly among groups over the treatment period, with the R group decreasing their ratio from 3.5 to 2.9 ($p < .05$). No changes were noted in percent body fat, fat-free mass,

or body weight for any of the groups. The Pearson product-moment correlations performed between the changes in blood lipids and the changes in body composition found no significant relationships. The results of this study indicate that an exercise program consisting of endurance training for 30 minutes, 3 times per week, or weight training for one hour, 3 times per week, or a combination aerobic/weight training program 3 times per week is not adequate to significantly improve plasma TC or HDL-C in young females over a nine week period. However, significant improvements may be made in the plasma TC/HDL-C ratio which may decrease the risk for CHD.

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CHAPTER I

Introduction

Cardiovascular disease (CVD) has been the leading cause of death in the United States during the past 3 decades. Annually, it accounts for approximately one million deaths (Hutchinson, 1985). Coronary heart disease (CHD), a major form of CVD, is responsible for over 50% of these cardiovascular deaths and 30% of all deaths (Pollock, Willmore, & Fox, 1984). Approximately 5.3 million people have CHD, while 1.5 million coronary events occur annually (McCunney, 1987). 314,000 individuals under 65 have their first coronary attack every year, with 45% of these attacks ending in death (Passamani, Frommer, & Levy, 1984). Although there has been a recent decline in mortality rates, deaths due to CHD continue to remain significantly above those due to cancer, the second leading cause of death.

In the past decade there has been a 25% decrease in cardiovascular deaths. One reason contributing to the decline in CHD is the improved medical care (Brandenburg, Fuster, Giuliani, & McGoon, 1987; Feinlab, 1984). An even greater factor may be the lifestyle changes of the American people with reductions in cigarette smoking, control of high blood pressure, and reduced plasma total cholesterol (TC) ((Kannel, 1983). It appears that people are beginning to take more of an interest in preventing or at least delaying the onset of disease.

Three primary risk factors have been identified as causing CHD: Cigarette smoking, hypertension, and hypercholesterolemia. Any one of these risk factors has the chance of doubling one's CHD risk (Criqui,1986; Hutchinson, 1985). Hypercholesterolemia, however, will be the issue addressed in this study.

Statement of the Problem

The effects of endurance and weight training on plasma cholesterol have been researched separately by a number of investigators. The general body of literature suggests a strong positive correlation between low levels of plasma TC and a low risk of CHD (Gordon, Castelli, Hjortland, Kannel, & Dawber, 1977; Brown & Goldstein, 1984). As the level of plasma TC decreases, the risk of CHD also decreases. An inverse relationship between plasma high density lipoprotein cholesterol (HDL-C) and CHD has also been noted. As plasma HDL-C levels increase, the risk of CHD decreases.

A great deal of research has focused on the effects of endurance exercise (ie., jogging) on plasma TC and HDL-C. Many studies have found that this type of exercise results in unchanged or slightly lower plasma TC values and increased plasma HDL-C levels.

On the other hand, the few studies looking at the effects of weight training on plasma TC and HDL-C have been conflicting. Some note significant decreases in plasma TC and increases in plasma HDL-C with weight training while others noted no changes in either. Reasons for this disparity may be the differences in degree of resistance, number of repetitions, and length of rest intervals as well as the total workload used in training sessions (Hurley & Kokkinos, 1987). No studies have compared the effects of a combined program of endurance and weight training on plasma TC and high density lipoprotein-cholesterol.

Various cholesterol studies reported changes in body composition for those subjects involved in experimental treatments. Possible correlations between body composition changes and plasma HDL-C changes have been suggested (Moore, Hartung, Mitchell, Kappus, & Hinderlitter, 1983; Rotkis, Boyden, Stanforth, Pamenter, & Wilmore, 1984; Schwartz, 1987). As the amount of lean body tissue increases, plasma HDL-C levels

increase. Many studies have found that with no change in body weight, decreases in fat weight, and increases in plasma HDL-C levels were observed after exercise training (Goldberg, Elliot, Schutz, & Kloster, 1984).

Research Hypothesis

Ho: There was no difference between individuals who did not engage in physical exercise, those who trained with weights, those who trained aerobically, and those who combined weight training and an aerobic activity in the following variables:

1. Plasma TC and HDL-C
2. Percent body fat, fat-free mass, and body weight

Ho: There was no relationship between the changes in body composition and the changes in plasma TC or HDL-C in individuals who did not engage in physical exercise, those who trained with weights, those who trained aerobically, and those who combined weight training and an aerobic activity.

Significance of the Study

Numerous studies have addressed the issue of aerobic activity and its effect on plasma TC and HDL-C. Fewer studies have explored the effects of weight training on the two variables. The results of these studies are not in agreement. If the results of this study demonstrate a decrease in plasma TC and/or an increase in plasma HDL-C with weight training, this would suggest that weight training decreases risk of heart disease.

The purpose of this study was to investigate not only the effects of an aerobic activity and the effects of a weight training program, but also the effects of an exercise regimen which combined the two activities. Since this combined exercise program has not been addressed in the literature, the results of this study will seek to answer some of the existing research questions, as well as identify additional areas for further research.

It is also important to understand the relationship, if any, between body composition changes, plasma TC changes, and plasma HDL-C changes.

Limitations

The following limitations of the study were recognized:

1. Sampling was non-random since the subjects volunteered. Thus the results of the study are intuitively generalizable only to similar samples.
2. The subjects may have increased or decreased caloric intake during the treatment period which could have affected their cholesterol and/or plasma HDL-C levels.
3. Some of the subjects participating in the study were on birth control pills which may have had a confounding effect on plasma TC or HDL-C levels.
4. Diets were not to be monitored or changed during the course of this study. Subjects were encouraged to maintain normal dietary patterns.
5. Those subjects that jogged had to start a jog/walk exercise program at 60% of their age-predicted maximum heart rate in order to work up to a continuous jogging program at 75 - 80% of their age-predicted maximum heart rate.

Definitions and Symbols

The following definitions and symbols will be utilized throughout this paper:

1. Atherosclerosis - a form of arteriosclerosis in which deposits composed of fatty materials, cholesterol and dead cells form in the large and medium-sized arteries.
2. Cholesterol - an essential lipid found in all living cells, especially in the brain, liver, kidneys, adrenals and myelin sheaths surrounding nerves. It is transported to the tissues in the blood via lipoproteins.
3. Fat-free weight - total body weight minus fat weight.

4. High-density lipoprotein-cholesterol - a plasma lipoprotein containing high levels of protein, small amounts of triglycerides, moderate levels of phospholipids and relatively little cholesterol.
5. Kilocalorie - a measure of heat energy. A calorie represents the amount of heat needed to raise one gram of water one degree Celsius.
6. Lean body mass - body mass composed of muscle, bone, and other tissues. Includes an essential amount of lipid as necessary for membrane and nerve as well as other physiological functions.
7. Low-density lipoprotein-cholesterol - a plasma lipoprotein containing a low percentage of triglycerides, high levels of cholesterol and moderate levels of phospholipids and protein.
8. One repetition maximum (1 RM) - the greatest amount of weight that a subject is able to lift only once on a weight-training apparatus.
9. Percent body fat - the portion of the body which consists of fat, as opposed to lean muscle tissue, expressed in percentage form.
10. Serum total cholesterol - cholesterol in the blood serum, also called blood cholesterol.

The following symbols will be used throughout the text:

1. BW: body weight
2. C: control group
3. FFM: fat-free mass
4. g: gram
5. HDL-C: high-density lipoprotein-cholesterol
6. HR: heart rate
7. Kcal: kilocalorie
8. kg: kilogram

9. LBM: lean body mass
10. LDL-C: low-density lipoprotein-cholesterol
11. %BF: percent body fat
12. R: running group
13. RM: repetition maximum
14. RW: running/weight training group
15. TC: total cholesterol
16. VLDL-C: very low-density lipoprotein-cholesterol
17. W: weight training group

Basic Assumptions

The following assumptions have been met:

1. The subjects exercised no more or less than their prescribed workouts throughout the course of this study.
2. The subjects consumed a weight maintaining diet throughout the course of the study.
3. During hydrostatic weighing, the subject expelled all air and layed still enough for an accurate reading.
4. When testing for 1RM, subjects exhibited a maximal performance.

Summary

Cardiovascular diseases have been the number one cause of death since 1950. Much has been done to decrease the mortality rate, however, many questions remain unanswered as to the prevention of CHD. Research reports a decrease in CHD with a decreased plasma TC concentration. It has also been suggested that an increased plasma HDL-C concentration will decrease CHD. Aerobic activity (ie., running) and weight training have demonstrated slight decreases or no changes in plasma TC, as well as increases plasma HDL-C. This study compared the effects of a weight training regimen, an

aerobic exercise regimen, and a combined aerobic and weight training regimen on plasma TC and HDL-C concentrations. This study also looked at the relationships between changes in these variables: FFM, %BF, BW, and plasma TC, HDL-C, and the TC/HDL-C ratio.

CHAPTER II

REVIEW OF THE LITERATURE

Introduction

For more than 30 years scientists have been searching for insight into the complex etiology of coronary heart disease (CHD). CHD has been the leading cause of death in the United States during this time (Ross, 1986), causing 1.5 million myocardial infarctions and claiming over 500,000 lives annually (McCunney, 1987). The exact cause of atherosclerosis is still unknown, but numerous risk factors, especially smoking, hypertension, and hypercholesterolemia play significant roles in its development (Francis, 1980; Patsch & Patsch, 1984; McCunney, 1987).

A decline in CHD has occurred over the past several years. Increased medical care and technology have been important factors, but the healthy changes in the lifestyles of the American population which help to control the major risk factors may be more important in explaining this decline (Thom, Kannel, & Feinlab,1985).

Pathogenesis of Atherosclerosis

CHD results largely from the slow formation of plaques (deposition of cholesterol and cellular debris) on the arterial wall. This process of plaque formation is known as atherosclerosis. According to Brown and Goldstein's theoretical model (1984), it is damage to the thin layer of endothelial cells that lines an artery which initiates plaque formation. The damaged endothelium is injured at some point, allowing low-density lipoprotein-cholesterol (LDL-C) particles and blood platelets to penetrate the cell wall. Hormones (ie., platelet-derived growth factor, PDGF) are released, causing smooth-muscle cells in the layer below the endothelium to multiply and migrate into the damaged area. The smooth-muscle cells and macrophages are overwhelmed by the high levels of cholesterol, hence they begin to ingest and degrade LDL-C thereby forming cholesterol-

filled foam cells. High levels of LDL-C in the plasma cause excess cholesterol from the LDL-C to accumulate in and among the foam cells. The accumulated cholesterol, cells, and debris constitute an atheroma, which in time can narrow the channel of the artery. This results in a restriction of the blood flow to the body, which, in specific areas may cause a heart attack (heart) or stroke (brain).

Ross (1986) refined the concept of endothelial injury by proposing two pathways of proliferative lesions. The first pathway has been observed in experimentally induced hypercholesterolemia, and is similar to the theory of Brown and Goldstein (1984). Injury to the endothelium induces release of growth factors. Monocytes attach to the endothelium, thereby continuing the release of more growth factors. These monocytes migrate to the subendothelium, resulting in fatty streak formation and further growth factor secretion. Macrophages may stimulate or injure the overlying endothelium and may even lose their covering, causing platelet attachment. Thus, three sources of growth factor are possible - platelets, macrophages, or endothelium. Another possible source of growth factor may be smooth-muscle cells which form in the proliferative lesion itself.

An alternate pathway for development of atherosclerosis involves injury to the endothelium which nevertheless remains intact. Increased endothelial turnover results in growth factor formation. This stimulates migration of smooth-muscle cells from the media to the intima accompanied by endogenous production of growth factor (PDGF) from the smooth muscles. This could cause fibrous-plaque formation and further progression of the atherosclerotic process (Ross, 1986).

Further research is needed in this area of growth factor production and lesion formation. Continued testing of these hypotheses may enable researchers to determine which cells in lesions and adjacent tissues are releasing growth factors or inhibitors that may regulate lesion formation, progression, and regression.

Pathway of Cholesterol

Contrary to lay opinion, cholesterol is an essential component in the human body. It is not only an important building block for cell membrane (Dufaux, Assmann, & Hollimann, 1982; Patsch & Patsch, 1984), but also a necessary precursor for the manufacture of digestive bile acids and steroid hormones. This substance can be supplied by either exogenous (diet) or endogenous (produced within the body) sources. The liver produces about 80% of the body's cholesterol while the diet supplies the remaining 20%. Synthesized from acetic acid, cholesterol is found in greatest concentration in nervous tissue, the liver, and fat deposits (Garman, 1978).

The exogenous pathway begins as dietary cholesterol is absorbed in the intestine and packaged with triglyceride in the form of chylomicrons (Brown & Goldstein, 1984; Grundy, 1986). Lipoprotein lipase (LPL) aids in the removal of fatty acids as the chylomicrons travel through the capillaries. Upon reaching the liver, the cholesterol-rich remnants bind to specific receptors and are absorbed. The cholesterol is then either reabsorbed into the intestine (as bile acids) or repackaged with triglyceride to form very low-density lipoprotein (VLDL-C) particles and secreted back into the circulation to begin the endogenous cycle. Triglyceride is once again cleaved from the VLDL-C, leaving an intermediate-density lipoprotein (IDL-C) rich in cholesterol, which can be removed by the liver or converted to LDL-C. The latter is absorbed into the liver via LDL receptors or cleaved by the extrahepatic tissues. Some cholesterol is bound to high-density lipoprotein (HDL-C) and esterified by the enzyme lecithin cholesterol acyltransferase (LCAT), which allows it to be recycled back to IDL-C and LDL-C for uptake into the liver or other cells. The remaining cholesterol is carried out of the bloodstream and voided in the feces.

Cholesterol is transported through the plasma in combination with protein, triglyceride and phospholipids. These macromolecules are known as lipoproteins and

classified according to size and density. The higher the ratio of lipid to protein, the lower the density; the lower the density, the larger the particle (Eder, 1984). The major classes of lipoproteins are chylomicrons and VLDL-C, the principle component of both being triglycerides; LDL-C, composed mainly of cholesterol esters; and high-density lipoproteins (HDL-C), which contain primarily phospholipids and the highest amount of protein among the lipoprotein classes. HDL-C appears to play a critical role in the transport of cholesterol from the peripheral cells to the liver for catabolism and eventual excretion (Garman, 1978; Brandenberg et al., 1987).

Relationship between Plasma TC and CHD

It has been shown that increased levels of plasma total cholesterol (TC) and LDL-C are positively related to CHD (Castelli, 1983; Goldberg et al., 1984; Hartung, 1984). Plasma LDL-C, which accounts for approximately 50-70% of plasma TC (Francis, 1980), plays a major role in the transport of cholesterol from plasma to the peripheral cells for intracellular metabolism. Evidence suggests (Eder, 1984; Goldberg & Elliot, 1985) that LDL-C causes damage directly to the arterial endothelium, thus causing arterial smooth muscle to proliferate and accumulate large amounts of lipids, primarily cholesterol. According to Goldberg and Elliot (1985), this accumulation may be the mechanism which leads to formation of atherosclerotic plaques.

In the Framingham Heart Study, 2,253 men and 2,818 women were studied and 37 variables indicating high risk for CHD were determined. One of the top 3 factors was elevated blood cholesterol (Wood, Haskell, Blair, Williams, Krauss, Lindgren, Albers, Ping, & Farquhar, 1983). This study suggested that rates of CHD are relatively constant for plasma TC levels up to 200 to 220 mg/dl, but as levels above this range increased, the risk of CHD also increased. A common interpretation of these results suggests that little is to be gained by having plasma TC levels below 200 mg/dl. However, a curvilinear

relationship between plasma TC and CHD was seen in the Multiple Risk Factor Intervention Trial (MRFIT) (Stamler, Wentworth, & Neaton, 1986). A study of 356,222 men between the ages of 35 and 57 showed a continuous and graded relationship between plasma TC and CHD. When plasma TC levels were below 180 mg/dl, risk seemed unchanged; at higher levels of plasma TC, the risk factor for CHD was compounded. Of all CHD deaths, 46% were estimated to be excess deaths attributable to plasma TC levels above 180 mg/dl. Conversely, a 20 mg/dl decrease in plasma TC was found to decrease CHD mortality by 16%. The results of the Lipid Research Clinics Coronary Primary Prevention Trial (1984) yielded a similar report. It was suggested that on the average, each 1% decrease (approximately 2 - 3 mg/dl) in plasma TC results in an appropriate 2% decrease in the incidence of CHD.

Because of the positive correlation between plasma TC levels and risk of CHD, many investigators believe that the plasma TC levels should be as low as possible. However, this position does not specify desirable ranges or levels and thus, the question of what constitutes an ideal plasma TC level has arisen. The National Institutes of Health (NIH) (1985) and American Heart Association (AHA) (1984) have sought to answer this question recently. Both groups suggested guidelines for safe levels of plasma TC. The AHA recommended that plasma TC levels be contained below 200 mg/dl, while further suggesting that an ideal range falls between 130 and 190 mg/dl. The NIH conference arrived at a similar recommendation that plasma TC levels remain below 200 mg/dl.

In a 21-month study of the progression of atherosclerosis, 106 patients demonstrated angiographic evidence of atherosclerotic progression, while 13 patients showed none (Nash, Gensini, Simon, Arno, & Nash, 1977). Fifty-one percent of those showing atherosclerotic progression had plasma TC levels above 250, while 15% had values less than 200 mg/dl. Of those with no progression, no patients had values over 250

and 69% had values under 200 mg/dl. In light of this evidence, along with epidemiologic studies (Stamler, et al., 1986; Rifkind, 1984; Castelli, 1983) and recommendations of the AHA and NIH, it appears that plasma TC levels below 200 mg/dl are desirable and should be widely recommended, with levels below 180 mg/dl being optimal for reduced risk of CHD.

The Effect of Aerobic Exercise on Plasma TC

A variety of personal characteristics and environmental factors have been documented which influence the composition of plasma TC, including gender, age, body composition, dietary intake of fats, medications, family history, and exercise (Haskell, 1984; Carey, 1984). The effects of each of these factors interact with one another in a variety of ways, making a definitive statement regarding the independent effect of any one factor difficult, but not impossible. Many studies have controlled for dietary intake, age, gender, and medications while investigating the independent effect of exercise on plasma TC, thus, enabling the researcher to suggest strong correlations between exercise and plasma TC.

Observational studies of exercise effects on plasma TC have generally demonstrated that people who regularly perform vigorous, large-muscle, dynamic exercise for extended periods (endurance training) have similar or only slightly different plasma TC concentrations than less active people of similar ages. For example, some studies of long-distance runners report no differences in plasma TC (Lehtonen & Viikari, 1978; Thompson, Lazarus, Cullinane, Henderson, Musliner, Eshleman, & Herbert, 1983; Hagan & Gettman, 1983; Moore et al., 1983; Morgan, Cruise, Girardin, Lutz-Xchneider, Morgan, & Qi, 1986), while similar studies report either slightly lower (Wood, Haskell, Klein, Lewis, Stern, & Farquhar, 1976; Clarkson, Hintermister, Fillyaw, & Stylos, 1981) or higher (Lehtonen & Viikari, 1980; Hartung, Jackson, North, Reeves, & Foreyt, 1985)

values for endurance-trained athletes. No differences between trained athletes and sedentary controls have been reported for tennis players (Vodak, Wood, Haskell, & Williams, 1980), soccer players (Lehtonen & Viikari, 1980), or skaters (Farrell, Maksud, Pollack, Foster, Anholm, Hare & Leon, 1982).

Findings from prospective analyses of exercise and plasma TC levels have also been inconsistent. Lopez, Vial, Balart, and Arroyave (1974) studied plasma TC values on 13 medical students (mean age = 22 years) who participated in a 7 week training program. Treatment consisted of four 30-minute sessions per week of intense physical exercise (approximately 7 METS) involving 10 - 15 minutes of jogging, 10 - 15 minutes of bicycling, and 5 - 10 minutes of calisthenics. These tasks were performed to the subject's maximal tolerance. Plasma TC concentrations decreased significantly from 169 - 162 mg/dl ($p \leq .03$). Although this change was statistically significant, it was not clinically significant, due to the fact that the pretraining value was well within normal values. Dietary intake was individually adjusted to maintain caloric balance and body weight, but no control group was used, nor were other variables controlled, such as smoking or alcohol consumption.

In a study yielding similar results with 15 oarsmen and 21 controls, matched for age, smoking, and drinking habits (Danner, Wieling, Havekes, Leuren, Smit, & Dunning, 1984), plasma TC levels decreased significantly from 166 - 145 mg/dl during the 7 month training season ($p \leq .05$). In fact, after 2 weeks, plasma TC levels had fallen significantly below initial values to 155 mg/dl. Months 1 to 3 consisted of endurance training, months 4 to 6 involved interval training, and months 6 and 7 consisted entirely of rowing. Subjects trained 10 hours per week the first four weeks, and 14 hours per week thereafter, but the frequency, intensity and duration of each session were not discussed. Body weight remained constant in this study also, although skinfold thickness decreased significantly

from 30.7 - 22.2 mm ($p \leq 0.01$). These results suggested that diet did not influence plasma TC values. Although the frequency and intensity of each session was not discussed, plasma TC changes may have resulted from the number of hours spent exercising each week.

Other studies however, have failed to show a decrease in plasma TC after endurance training lasting several weeks or months. A study comparing a low intensity to a high intensity aerobic training program yielded no change in plasma TC values in either group after 18 weeks (Gaesser & Rich, 1984). Seventeen sedentary healthy, non-smoking males between 20 - 30 years participated in either a high intensity (H; n = 7) or low-intensity (L; n - 9) exercise training program. Both groups pedaled at 50 rpm 3 days per week on a cycle ergometer. H exercised 25 min/session at 80 - 85% measured VO2 max, while L exercised for 50 min at 45% measured VO2 max. The lack of change in plasma TC was expected by this researcher, due to the initially low values (H - 183 mg/dl; L - 182 mg/dl). Gaesser and Rich (1984) suggested that exercise training is unlikely to induce changes in plasma TC values which are low at the beginning of the study, however, the two previous studies which demonstrated significant decreases in plasma TC also had low initial values. Lack of change in this study may have been due to the duration of each session, rather than the low pre-training values. Dietary intake was not controlled, which may have confounded the results, also.

Blessing, Stone, Byrd, Wilson, Rozenek, Pushparani, & Lipner (1987) elicited similar results in plasma TC when they compared 11 subjects who jogged with 13 controls. The exercise program was conducted 3 times per week for 12 weeks. Joggers walked/jogged for 20 minutes at 60 - 75% of their predicted maximum heart rate (HR) for weeks 1-3, increasing to a 25 minute jog at 75 - 80% weeks 4-6, increasing still to 80 - 85% for 25 minutes, weeks 7-12. Plasma TC remained unchanged (231 - 232 mg/dl)

which may have been due to the low intensity and duration of the treatment of the first three weeks.

One of the few studies investigating the effects of endurance exercise on plasma TC in women also failed to induce a decrease in plasma TC (Rotkis et al., 1984). Nineteen healthy women who were already running between 15 and 25 miles per week participated in this study. Testing was performed when each subject had run 30 miles per week more than her baseline average weekly mileage (Δ30) for 2 consecutive weeks and 50 miles per week more than her baseline average weekly mileage (Δ50) for 2 consecutive weeks. The total length of the training time was 14.8 months (range = 13 -15 months). Plasma TC remained constant at each testing time (baseline = 185; Δ30 = 183; Δ50 = 185 mg/dl). Once again, initial plasma TC values were already low, which may in part explain the lack of change. There was however, a shift in the relative contribution of plasma HDL-C and LDL-C to plasma TC.

Thus far the effect of exercise on plasma TC concentration appears to be variable and inconsistent. Investigators have not always controlled for dietary intake and body weight in their analyses, both of which influence lipoprotein values. Seasonal variation, use of medication, and intensity and duration of training have also influenced the lipid and lipoprotein values. Another factor in the unchanged plasma TC values may be the concomitant increase in plasma HDL-C and decrease in LDL-C and VLDL-C (Wood & Haskell, 1979). This would cause plasma TC to remain relatively constant, yet decrease the plasma TC/HDL-C ratio, thus decreasing the risk of CHD (Gordon et al., 1977).

The Effect of Resistance Weight Training on Plasma TC

Exercise studies have primarily researched the effect of endurance training on lipoprotein levels. Few investigations have considered the consequence of exercise using resistance to muscular movement, despite evidence suggesting that periodic, intense

exercise of the heavy work occupations may reduce the incidence of CHD (Cassel, Heyden, Bartel, Tyroler, Cornoni, & Hames, 1971; Paffenbarger & Hale, 1975; Blessing et al., 1987). A longitudinal study by Paffenbarger & Hale (1975) assessed the role of physical activity in reducing coronary mortality among longshoremen. Cargo handlers, whose major duties included "repeated bursts of high-energy resistance exercise of lifting, pulling, and pushing cargo", similar to weight lifting, were the only group which showed a reduced mortality rate. Another group demonstrating a lower incidence of CHD were heavy-working farm laborers (Cassel et al., 1971). Both authors concluded that a critical threshold level of exercise intensity appeared to have been the most important factor in reducing CHD.

Body builders and powerlifters were compared to runners to examine the relationship between lipid profiles and type of weight training (Hurley, Seals, Hagberg, Goldberg, Ostrove, Holloszy, Wiest, & Goldberg, 1984). The powerlifters (PL) trained using heavy-resistance exercises with few repetitions, while the body builders (BB) trained using a moderate-resistance, high-repetition exercise regimen. When compared to the plasma TC of runners who trained 5 days a week and averaged 8 to 11 kilometers per 40 to 45-minute session (170 mg/dl), the BB had similar plasma TC values (172 mg/dl), while the PL had plasma TC values similar to sedentary controls (195 and 192 mg/dl, respectively). The data suggest that the intensity of a weight training program may be an important factor in lipoprotein alterations. Similar results were observed by Elliot, Goldberg, Kuehl, and Catlin (1987). They compared blood lipids in female bodybuilders and runners, finding that plasma TC values were lower for bodybuilders than runners (166 to 183 mg/dl, respectively). Both studies suggested that the effects of bodybuilding on lipid levels may be comparable with the effects of regular prolonged aerobic exercise.

Johnson, Stone, Byrd, & Lopez (1983) studied the effects of a 12-week weight training program on middle-aged men. The 24 subjects exercised 3 days per week for 45 - 60 minutes per session. Each session consisted of five sets of 10 repetitions at the following resistance: one set = light; one set = moderate; and three sets = heavy. The results showed a significant decrease in plasma TC (201 mg/dl - 192 mg/dl) for the experimental group with no decrease for the control group (188 - 185 mg/dl; $p < .05$). Goldberg et al. (1984) studied a group of young, healthy males (mean age = 33) and females (mean age = 27). Training consisted of three sets of repetitions for each of the seven or eight individual exercises on the Universal apparatus. Each exercise set was continued until a subject could not complete an additional repetition. No more than 8 or less than 3 repetitionss were allowed for each training set. After 16 weeks of progressive weight training (3 days per week, 45 - 60 minutes), females demonstrated a 9.5% reduction (198 - 179 mg/dl) ($p < .05$) in plasma TC, while males demonstrated a 6.8% reduction (209 - 194 mg/dl) ($p < .07$) in plasma TC. No dietary changes were reported among the subjects and body weight remained constant, however, skinfold measures decreased significantly for both males and females. Other variables, such as alcohol consumption, smoking, and medications were controlled, thus allowing the author to suggest that exercise was the factor which caused the reduction in plasma TC. It is interesting to note that although females had a lower initial plasma TC value, they nevertheless reduced those values to a greater extent than males. The mean age of the females was 27, whereas the mean age for males was 33 years of age. Age has been known to affect cholesterol levels (Haskell, 1984) and may have been a limiting factor in this study.

Contrary to the previous studies, Blessing et al. (1987) found no change in plasma TC after 12 weeks on a weight lifting regimen. The noncircuit resistive training program

included three sets of 10 reps with the maximum weight possible for the first 6 weeks, then decreased the repetitions but kept sets and weight constant. Plasma TC levels for the weight lifters decreased slightly, but nonsignificantly, from 229 - 227 mg/dl. The decrease in intensity may have been a factor in these nonsignificant results. Another study by Ullrich, Reid, and Yeater (1987), compared the effect of four constant resistance weight lifting programs: Endurance group - two sets of 15 RM, explosive group - one set of 15 RM, as quickly as possible, strength I - three sets of 6 RM, and strength II - one set of 10 RM 2 days per week and one set of 3 RM one day per week. Plasma TC did not change for any group after 8 weeks of training. Similarly, Kokkinos, Hurley, Vaccaro, Goldberg, and Ostrove (1986) found no significance after 10 weeks of weight lifting, however, plasma TC values were within normal limits before training began.

According to Johnson, Stone, Lopez, Herbert, Kilgore, & Byrd (1982), it appeared that the greatest changes in plasma TC occurred during the highest volume of training. It has also been suggested (Gaesser & Rich, 1984) that subjects having a low plasma TC level (below 180 mg/dl) at the onset of a training program would be the least likely to show any changes in plasma TC levels, due to the low initial values.

Differences Between Males and Females

There are differences in lipoprotein concentrations of males and females. Research supports the view that premenopausal females tend to have a slightly lower or similar plasma TC (Goldberg, 1984; Elliot et al., 1987) and a higher plasma HDL-C concentration than males of the same age (Gordon, et al., 1977; Carey, 1984). In fact, plasma HDL-C levels of sedentary females do not differ significantly from those of active males of similar age (Carey, 1984).

Kannel (1983) noted that excessive cholesterol concentrations contributed only slightly to increased risk among women when compared to men. However, Gordon et al.,

(1977) found low plasma HDL-C values to be a significant risk factor in females when associated with diabetes and obesity, raising the level of risk to that of males of similar age.

Plasma HDL-C consists of two subfractions: Plasma HDL_2 and HDL_3. The plasma HDL_2 are larger in size and contain more lipids than plasma HDL_3, thus allowing them to carry about twice as much cholesterol per unit as plasma HDL_3. Physical exercise predominantly increases plasma HDL_2 (Patsch & Patsch,1984). The major difference in lipoprotein levels between females and males lies in the plasma HDL_2 levels, which are three times higher in women. Having such a high percentage of plasma HDL-C carried by the plasma HDL_2 subfraction is one reason why women have a lower incidence of CHD than men.

Most researchers have found plasma TC levels to be similar in both sexes, however, plasma HDL-C levels are about 10 mg/dl higher in women (Moore et al., 1983; Elliot et al., 1987). Elliot measured the plasma HDL-C levels of male and female bodybuilders against similar runners. Both female bodybuilders and runners had plasma HDL-C values approximately 10 mg/dl higher than their matched male counterparts.

Data from studies looking at endurance exercise find that female as well as male endurance athletes have higher plasma HDL-C concentrations than their sedentary counterparts. The study by Wood et al., (1976) reported that plasma HDL-C values from male runners and sedentary controls were 64 and 43 mg/dl, respectively. Female runners and controls had plasma HDL-C values of 75 and 56 mg/dl, respectively.

The dose-response relationship between plasma HDL-C and exercise in active women is similar to that of men (Smith, Mendez, Druckenmiller, & Krisetherton,1982; Moore et al., 1983; Goldberg et al., 1984). Moore compared the average plasma HDL-C cholesterol concentration of inactive women, joggers, and long-distance runners with kilometers run per week. There was a moderate correlation between distance run per week

and plasma HDL-C values (r = 0.408). Along with these results were presented the findings of a previous study on men (Gordon et al., 1977). Females elicited plasma HDL-C values of 62, 70, and 78 mg/dl, at 0, 20, and 50 km/week,respectively. A similar pattern was seen in the results of the men with the exception of an average 10 mg/dl decrease in plasma HDL-C values at the same distance. As distance per week increased, plasma HDL-C values also increased, similar for both men and women. Goldberg et al., (1984) elicited the same response from males and females after a 16-week, progressive weight training program. Beginning plasma HDL-C values were 51 and 77 mg/dl for men and women, respectively, increasing in similar fashion to 59 and 81 mg/dl. Albeit, the changes after training were non-significant, the direction of change was similar for both groups.

Lipoprotein lipase (LPL), an enzyme necessary in the degradation of triglyceride-rich lipoproteins (VLDL-C) has been shown to increase after exercise training in females (Goldberg & Elliot, 1985). Females carry a greater amount of adipose tissue, plus, adipose tissue LPL activity is higher in females than males. Therefore, the higher plasma HDL-C levels in women may be due to increased LPL activity (Goldberg and Elliot, 1985). Moll, Sanders, Williams, Lester, Quarfordt and Wallace (1979) suggested two possibilities: (1) that exercise in males activates a pathway toward plasma HDL-C formation which is already maximally activated in females, or (2) exercise in males inhibits a pathway degrading plasma HDL-C that is already inhibited in females.

Relationship Between Plasma HDL-C and CHD

Plasma lipoproteins have long been considered a major factor in the pathogenesis of CHD. Plasma HDL-C has received considerable attention in recent years due to its potentially protective effect against the development of CHD. Plasma HDL-C is thought to mobilize free cholesterol both from the bloodstream and from atherosclerotic plaques for

metabolism by the liver (Eder, 1984; McCunney, 1987; Brandenberg et al., 1987). In this system called reverse transport, L-CAT, which is loosely associated with plasma HDL-C, functions to esterify free cholesterol on the surface of plasma HDL-C particles (Dufaux et al., 1982). The cholesterol becomes hydrophobic and migrates to the neutral core of the plasma HDL-C particle (Patsch & Patsch, 1984; Brandenberg et al., 1987). This action frees the cholesterol binding sites on the surface of the plasma HDL-C particle and thus permits them to pick up additional free cholesterol from the tissues or vessel walls and blood stream and transport them back to the liver for catabolism and excretion. It is proposed that this function of plasma HDL-C contributes to its property of being a negative risk factor for CHD.

An inverse relationship between plasma HDL-C and CHD was first observed in the early 50's (Barr, Russ, & Eder, 1951), and later confirmed in 1966 (Mills & Wilkinson, 1966). In 1975, Miller and Miller suggested the hypothesis that plasma HDL-C may play a protective role against the development of CHD and may well be a better predictor of CHD than plasma TC. Following that observation, epidemiological data were reexamined and it was found that patients with low levels of plasma HDL-C had considerably higher risk of CHD than patients with high levels of plasma HDL-C (Eder, 1984). In the Framingham study, for example, it appeared that for every 10 mg/dl decrease in plasma HDL-C, the CHD risk was increased twofold (Eder, 1984).

In a controlled study of 6,854 men and women of black, white, and Japanese descent (over 40 years old), from populations in Albany (NY), Framingham (Mass.), Evans Co. (GA), Honolulu, and San Francisco, the mean levels of plasma HDL-C in each study were lower in those with CHD than without the disease (Castelli, Doyle, Gordon, Hames, Hjortland, Hulley, Kagan, & Zukel, 1977). Studies have shown that patients who sustained a myocardial infarct also had significantly lower levels of plasma HDL-C than

controls (Enger, Hjermann, Foss, Helgeland, Holme, Leren & Norum, 1979; Havel, 1979). Among 104 patients with coronary angiography, those without CHD had average plasma HDL-C levels of 53.9 mg/100ml, which was 30% greater than those patients with abnormal coronary arteries (Tan, McIntosh, & Weldon, 1980). Similarly, an investigation involving 483 men and women showed that the plasma HDL-C level was inversely related to the degree of stenosis and the number of vessels affected (Pearson, Bulkley, & Achuff, 1979).

The ability of plasma HDL-C to predict the development of CHD has been estimated to be four times greater than plasma TC (Gordon et al., 1977). Two possible mechanisms for the connection between plasma HDL-C and CHD have been presented: 1) plasma HDL-C acts as a "scavenger" by transporting cholesterol from the peripheral cells to the liver for degradation and elimination, and 2) plasma HDL-C interferes with the uptake and degradation of LDL-C at extrahepatic tissue sites (Carew, Hayes, & Koschinsky,1976; Dufaux et al., 1982).

The Effect of Aerobic Training on Plasma HDL-C

Because high levels of plasma HDL-C are associated with low levels of CHD and the notion that plasma HDL-C may play a protective role in the prevention of CHD, researchers have investigated potential factors which may increase or decrease plasma HDL-C. Increased concentrations of plasma HDL-C have been associated with low body weight index, ethanol consumption, nicotinic acid, estrogen, various medications, and exercise (Fenman, Alaupavic, & Howard, 1967; Avogaro, Cazzdato, Bittolo, 1978; Ernst, Fisher, & Smith, 1980; Wallace, Hunninohake, Reiland, 1980; Dufaux et al., 1982; Haskell, 1984; Goldberg & Elliot, 1985; McCunney, 1987). Reduced levels of plasma HDL-C have been associated with high body weight index, high carbohydrate diets, androgens, cigarette smoking, increasing age, male gender, and physical inactivity (Dufaux

et al., 1982; Stubbe, Eskilsson, & Nilsson-Ehle,1982; Haskell, 1984; Goldberg & Elliot, 1985).

Recently, a great deal of interest has centered around the potential effects of physical activity (more specifically, aerobic exercise) upon plasma HDL-C levels. Generally, most studies have found an increase in plasma HDL-C levels following various training programs, although no definitive conclusions have been drawn regarding the intensity, frequency, or duration necessary for exercise to positively influence plasma HDL-C levels.

Cross-sectional Studies on Plasma HDL-C

Cross-sectional studies performed on previously active individuals appear to agree on one important factor: active individuals had higher plasma HDL-C levels than inactive subjects. Plasma HDL-C levels in a group of runners were compared to those in a group of professional golfers and inactive men (Hartung et al., 1985). The runners had significantly higher plasma HDL-C concentrations than both the golfers or controls. Plasma TC in the runners was slightly higher, but a greater percentage of plasma TC was carried by HDL-C. Similarly, Farrell et al. (1982) compared the blood levels of plasma HDL-C in 11 candidates for the 1980 U.S. Olympic speed skating team, and 11 untrained men. The speed skaters had significantly higher plasma HDL-C levels (53.7 mg/dl) than the control group (47 mg/dl), further supporting the hypothesis that those who train aerobically tend to have higher plasma HDL-C levels.

Clarkson et al.(1981) compared 6 trained distance runners (involved in competitive running for several years, averaging 70 miles/week), and 17 untrained subjects who were uninvolved in any type of exercise program. No significant differences in plasma HDL-C were found between the 2 groups, although the percentage of plasma HDL-C was significantly higher in runners (43.7%) than controls (32.0%). Since the runners had such

low plasma TC levels (126 mg/dl) compared to the controls (165 mg/dl), it is not surprising that the plasma HDL-C levels were about the same. What is important is that the runners had an increased proportion of their plasma TC carried in the plasma HDL-C fraction.

Seven female competitive swimmers, 11 synchronized swimmers, and 6 sedentary females were studied for possible differences between plasma HDL-C levels (Smith et al., 1982). Plasma TC was not significantly different, although plasma HDL-C levels of the competitive swimmers were 17% and 22% higher than those of the synchronized swimmers and controls, respectively. In 1983, Moore et al., measured plasma HDL-C in 45 long-distance runners (at least 26 miles per week), 49 joggers (6 miles per week) and 47 inactive women (no activity for at least 6 months preceding the study). Pairwise comparisons revealed significant differences between all groups, with the plasma HDL-C in women running 31 miles per week being 16 mg/dl higher than average control levels. Elevated plasma HDL-C levels were associated with increased exercise levels. The plasma HDL-C levels increased as the weekly mileage increased. A study by Hartung and Squires in 1980 on men demonstrated a similar increase in plasma HDL-C concentration with increased mileage, although the levels for women were higher than men for all distances.

Numerous other cross-sectional studies have demonstrated increased plasma HDL-C levels in those involved in endurance exercise such as cross country skiing (Enger, 1977), endurance running (Adner & Castelli, 1980; Lehtonen & Viikari, 1978; Wood et al., 1976, Wood, Haskell, Stern, Lewis, & Perry, 1977; Morgan et al., 1986), soccer players (Lehtonen & Viikari, 1980); and tennis players (Vodak et al., 1980).

Longitudinal Studies on Plasma HDL-C

The general consensus of longitudinal studies has demonstrated an increase in plasma HDL-C with endurance exercise. Those individuals not revealing an increase in

plasma HDL-C were generally those with the highest initial values (Haskell, 1984; Goldberg & Elliot, 1985; Blessing et al., 1987). For the most part, changes in plasma HDL-C have been dependent upon levels prior to exercise conditioning. Subjects with lower initial plasma HDL-C values showed the greatest increases after training.

A group of 19 women participated in an endurance training program for 15 months (Rotkis et al., 1984). Training consisted of an individualized, progressive running program in which each woman ran 5 - 6 days per week and was to slowly increase her mileage without regard to speed. Plasma HDL-C was measured prior to training, after each woman had run 30 miles per week more than her baseline average weekly mileage (Δ30) for 2 consecutive weeks, and 50 miles per week more than her baseline average weekly mileage (Δ50) for 2 consecutive weeks. No changes were seen in plasma TC (185 mg/dl, 184 mg/dl, and 185 mg/dl) or BW (56.8 kg), however plasma HDL-C progressively increased from 59.6 mg/dl (baseline) to 63.7 mg/dl (Δ30), and 69.4 mg/dl (Δ50). Lean weight also increased from 42.5 to 43.8 to 44.6 kg ($p \leq .001$). The authors suggested that training duration and/or frequency resulted in the elevated plasma HDL-C levels. But it is improbable that the average jogger would care to invest this much time and effort in exercise in order to elicit more desirable plasma HDL-C values. It is more realistic that a minimum number of miles per week and days per week be determined so as not to discourage potential exercisers before they even begin.

It was interesting to see plasma HDL-C levels increase when they were high initially (59.6 mg/dl). It appears that the number of miles ran per week influenced even these high plasma HDL-C levels. Those individuals with the lowest baseline plasma HDL-C values had the largest increases both at Δ30 and Δ50.

Gaesser & Rich (1984) were unable to elicit any change in plasma HDL-C levels after an 18 wk high- (H) and low-intensity (L) training program. Subjects pedaled on a

cycle ergometer 3 days per week at 80 - 85% for 25 minutes (H) or at 45% for 50 minutes (L). There was no dietary control, however, BW did not change. %BF decreased significantly ($p \leq .05$), for both groups. Pretraining plasma HDL-C values were relatively high for men (42 and 45 mg/dl) and therefore, not expected to change significantly. The plasma TC levels were also low (182 - 183 mg/dl), resulting in a high percentage of cholesterol carried by plasma HDL-C. According to Gaesser, when initial plasma TC values are low and plasma HDL-C values are high, rarely does any treatment change this. An exception to this theory was seen in a study by Rotkis et al., (1984). Subjects increased running mileage by 30 miles per week after 7.5 months, and further increased mileage per week to 50 miles above average baseline values. Plasma TC remained stable while significant increases in plasma HDL-C were seen from 59.6 - 63.7 - 69.4 mg/dl ($p < .05$). There was no change in body weight, yet a significant decreases in %BF, thus demonstrating that body weight did not influence the plasma HDL-C increase.

A group of 48 previously sedentary males participated in a running program lasting one year (Williams et al., 1982). Initially, the training program met 3 days per week and ran 20 minutes with a 5-min warm-up and cool-down period. After 2 - 3 weeks, subjects added an additional day, and by wk 10 were running 5 days per week. Gradually, duration increased also, up to 45 minutes of running. An intensity of 70 -85% was maintained during the entire training period. A slight decrease was seen in plasma TC at 9 - 13 miles; increases were seen in plasma HDL-C values at 10 miles per week. According to the researcher, failure to achieve a significant decrease in plasma HDL-C was due to the large proportion of the exercise group (> 50%) that failed to reach and/or maintain the necessary level of exercise (> 10 miles per week). Nonetheless, this study showed that number of miles run and duration of exercise training were directly related to changes in plasma HDL-

C (r = .48). Plasma HDL-C did not increase until an average exercise training level of 10 miles per week for nine months of training was achieved.

In a 7-month study of 15 oarsmen and 21 controls matched for age, smoking, and drinking habits, a significant increase in plasma HDL-C and decrease in plasma TC was seen (Danner et al., 1984). During the first four months, non-specific endurance training was prescribed for 10 hours per week; the last 2 - 3 months involved rowing for 14 hours a week. Plasma HDL-C values increased significantly ($p \leq .05$) from 47.7 - 51 mg/dl in the oarsmen compared to a nonsignificant drop in controls from 50 - 49.6 mg/dl. An unexpected interruption in the training program during the third and fourth month caused plasma HDL-C values to return to their pre-training levels, although they increased significantly during the last 3 months. This may suggest that either rowing itself is more beneficial in affecting plasma HDL-C values or the time involved exercising caused the significant increase.

Recently, a 12-week study comparing 11 joggers and 13 controls found an increase in plasma HDL-C without a concomitant change in plasma TC (Blessing, et al., 1987). Training for the joggers consisted of a 20 min walk/jog at 70 - 75% predicted max HR for weeks 1-3. The level increased to 75 - 80% during the second 3 weeks (duration = 25 minute jog) and 80 - 85% for 25 minutes for weeks 7 - 12. While plasma TC remained stable for joggers (231 - 232 mg/dl), plasma HDL-C levels increased from 38 - 44 mg/dl (no change for control group). The results did not indicate whether the post-training values differed significantly from pre-training values. However, it was noted that post-training values for the joggers were significantly different from those in the C group, suggesting that exercise was responsible for the change in plasma HDL-C. Body weight did not change, which was a function of increased LBM and decreased %BF. Once again, the

author did not indicate whether the post-training changes were significant from pretest values.

It does appear that increased levels of physical exercise have positive effects on plasma HDL-C, which in turn, may slow the progression of CHD. Several aspects of the training program are necessary in order to achieve this effect. The length of a training program is an important factor in affecting lipoprotein values. Moll et al., (1979) failed to achieve an increase in plasma HDL-C after 6 weeks of endurance training, while Farrell and Barboriak (1980) were able to see significant changes in plasma HDL-C following an 8 week exercise training program. Likewise, Ullrich et al.(1987) likewise saw a significant increase in plasma HDL-C after 8 weeks of endurance training. However, Williams et al., (1982) exercised their subjects for a year and did not begin to see changes in plasma HDL-C until 9 months of exercise training. Conversely, because two studies demonstrated a return to baseline plasma HDL-C values after terminating their exercise programs, it may be suggested that a lifetime fitness program be incorporated in order to consistently control levels of plasma TC and HDL-C.

Not only is the length of the study important, but also the frequency, intensity, and duration of each exercise session. While Williams et al., (1982), Wood et al., (1983), and Rotkis et al., (1984) suggested training 5 days per week, Blessing et al., (1987), utilizing only 3 days per week, saw an increase (NS) in plasma HDL-C from 38 - 44 mg/dl, which improved the plasma TC/HDL-C ratio from 6.1 - 5.5. Dufaux et al. (1982), Patsch & Patsch (1984), and Brandenburg et al., (1987) likewise suggested 3 - 4 days per week.

The duration or miles run during each session are interrelated with the intensity, or percentage of maximum heart rate. The American College of Sports Medicine (1985) suggests at least 30 - 60 minutes at 70 - 85% of maximal HR. Myhre, Mjos, Bjorsvik, & Stromme (1981), Dufaux et al., (1982), Patsch & Patsch (1984), and Brandenburg et al.,

(1987) suggested a lower limit of 30 minutes at 70 - 80% max HR was adequate to achieve improved lipoprotein levels. Paffenberger, Wing, and Hyde (1978) investigated the number of kcalories expended and suggested that greater than 2000 kcal per week above normal levels of activity be incorporated. This approximates 20 miles per week, which is in agreement with Superko ,Wood, & Haskell (1985). However, Kavanaugh , Shephard, Lindley, & Pieper (1983) induced significant increases in plasma HDL-C levels at 12.4 miles per week, and Wood et al., (1983) and Williams et al., (1982) indicated that if an average of 10 miles per week were maintained, increases in plasma HDL-C might result.

The amount of exercise necessary to result in increased plasma HDL-C levels is an important "dose-response" issue. The above investigations show that exact guidelines have not been agreed upon, however they have shown that before a beneficial effect on plasma HDL-C can be achieved from exercise, a threshold needs to be surpassed that may be defined by type, frequency, intensity, and duration of activity as well as kcalories expended. It has also been suggested that the changes in lipoprotein concentrations lasted only as long as the training period (Danner et al., 1984). Therefore, a lifetime program of physical activity is recommended.

Most of the studies that did not induce changes in plasma HDL-C levels studied young, healthy men or women whose plasma HDL-C values were initially high and therefore, not subject to a great deal of change (Gaesser & Rich,1984; Schwartz, 1987). Goldberg and Elliot (1985) suggested that changes in plasma HDL-C were dependent upon levels prior to exercise conditioning. It would appear reasonable to investigate the effects of exercise on those subjects to whom an increase in plasma HDL-C would be beneficial as a risk factor of CHD, such as those with hypercholesterolemia.

The Effect of Weight Training on Plasma HDL-C

Previous studies on the effects of exercise on plasma HDL-C levels have been largely confined to investigations of endurance exercise. Little attention has been directed toward the possibility that weight training could improve lipoprotein levels. Weight training is one of the fastest growing physical activities (Stone & Wilson,1985) and is widely used to enhance various aspects of physical fitness as well as a training component for many sports. Because a large segment of the population engages in this activity, it would appear that more extensive research on the effects of weight training on a broad sample of measures would be beneficial.

What little research that has been done on weight lifting has resulted in controversy. Cross-sectional studies of young adult male weight lifters revealed no differences in lipid profiles between weight lifters and sedentary subjects (Clarkson et al., 1981; Farrell et al., 1982). Neither study reported training volumes or intensities, nor did they describe the type of exercises performed. Additionally, they did not control for androgen usage, which is common among weight lifters and is known to depress plasma HDL-C levels (Stone & Wilson, 1985).

Beneficial effects of weight training on plasma HDL-C levels suggested by longitudinal studies have shown more promising results. Ullrich et al., (1987) studied the effects of weight training on plasma HDL-C in 4 different groups: endurance - two sets of 15 RM; explosive group - one set of 15 RM, as quickly as possible; strength I - 3three sets of 6 RM; and strength II - one set of 10 RM 2 days per week and one set of 3 RM one day per week. Training continued for 8 weeks. A significant improvement in plasma HDL-C (38.8 - 44.0 mg/dl) occurred in all subjects, although plasma TC did not improve. Similarly, Kokkinos et al., (1986) studied 29 healthy, untrained college males for 10 weeks. A low repetition group trained using 4 - 6 RM and a high repetition group trained

using 14 - 16 RM. Kokkinos et al., found no significant difference in plasma HDL-C after training, but attributed it to the already high plasma HDL-C values.

By weight training young men and women for 16 weeks, Goldberg et al., (1984) found a non-significant increase in plasma HDL-C levels for both men (50.6 - 58.6) and women (77.4 - 81.1 mg/dl). It should be noted that plasma TC was relatively low for both groups initially, thus keeping the ratio of plasma TC to plasma HDL-C low (4.2 - 3.3 for men; 2.6 - 2.2 for women; $p > .05$). Blessing et al., (1987) compared weight training to a jogging program in middle-aged males. Plasma HDL-C values increased for both treatment groups (38 - 42 mg/dl for weight trainers; 38 - 44 mg/dl for runners), while the control group remained the same. Statistical analyses revealed significant differences in the posttest plasma HDL-C values between the experimental and control groups. He reported that the plasma HDL-C changes were "meaningful because the alterations were significant and in directions connected to lower risk for CVD". However, he did not report whether the changes from pre- to posttest were significant in the treatment groups.

Though it seems that weight training may not cause as great a change in plasma HDL-C as endurance-type activities, the direction of change is favorable. Resistance exercise may, therefore, be an important measure in the reduction of CHD, although more extensive research is necessary in order to determine the specific exercise prescription and length of time necessary to effect a change in blood lipids.

The Relationship Between HDL-C and Body Composition

Recent research has investigated the relationship between body composition and plasma HDL-C. Several questions surface in this area: Does an increase in LBM or a decrease in %BF cause a concomitant increase in plasma HDL-C or are these 2 variables unrelated? At what point does the increase/decrease of one affect the other and do the initial values of either variable affect their ability to improve?

Percent body fat is that part of the body composed only of fatty tissue, while LBM consists of muscle, bone, and other tissue, as well as an essential amount of lipid that is necessary for nerve, membrane, and other physiological functions (Buskirk & Mendez, 1984). Fat-free mas is total body weight minus fat weight. Most analytical techniques used to assess body composition (i.e., hydrostatic weighing and skinfold measures) are measuring FFM rather than LBM. However, researchers continue to use the incorrect term - LBM. In this review, the same terms will be utilized as the individual researchers.

While %BF is normally expressed in percentage form, LBM is usually expressed in kilograms in the cholesterol literature. When %BF decreases, LBM increases if body weight is held constant. Throughout the current literature, both LBM and %BF are used frequently. The general trend seen in cholesterol and CHD research is a decrease in %BF and an increase in LBM while BW is held constant. A change in body weight will affect cholesterol levels, thus researchers attempt to control for that variable in order not to confound the results.

In a cross-sectional study by Moore et al., (1983), she measured %BF and plasma HDL-C in three groups of women ages 24 - 58 years: inactive women, joggers (at least 6 miles per week), and long distance runners (at least 26 miles per week). Pearson correlation showed %BF to be the variable most highly correlated to plasma HDL-C ($r = -0.491$). Distance run per week was also related to plasma HDL-C ($r = 0.408$). It appears from these results that for these women the further one ran, the leaner she was, and the higher the plasma HDL-C levels. Therefore, the author suggested a probable relationship between plasma HDL-C and LBM.

Another cross-sectional study of 7,338 males and 6,768 females found an inverse relationship between body mass index (BMI) and plasma HDL-C (Forde, Thelle, Arnesen, & Mjos,1986). Both variables demonstrated a linear relationship; plasma HDL-C being

higher in those with lower relative body weight and lower plasma HDL-C levels in those subjects with a higher relative body weight.

Rotkis et al., (1984) found a significant correlation between the changes in plasma HDL-C and LBM ($r = 0.65$; $p < 0.002$), after female subjects had increased running distance by 50 miles per week. There was no change in BW, although a decrease in %BF was noted, as well as a progressive increase in plasma HDL-C levels. Goldberg et al., (1984) found similar results in a study of males and females who weight trained for 16 weeks. Although there was no change in BW, a significant decrease in skinfold thickness ($p < 0.05$) suggests that LBM increased. Males increased plasma HDL-C levels from 50.6 mg/dl to 58.6 mg/dl ($p > 0.05$; 15.8% increase) and females demonstrated a 4.8% increase in plasma HDL-C values (77.4 - 81.1 mg/dl; $p > 0.05$). Although nonsignificant for both genders, the direction of change in plasma HDL-C values along with stable weight and loss of body fat indicated a relationship between LBM and plasma HDL-C. Had a greater number of subjects participated (females, $n = 8$; males, $n = 6$) or a longer exercise training period been utilized, a greater rise in plasma HDL-C may have occurred (Goldberg et al., 1984).

Similarly, Blessing et al., (1987) studied 33 males who jogged or weight-trained for 12 weeks. He found no change in BW, a decrease in %BF, and an increase in plasma HDL-C from 38 - 44 mg/dl and 38 - 42 mg/dl for joggers and weight-trainers, respectively. Thompson, Cullinane, & Eshleman (1984) studied the effect of exercise cessation on plasma HDL-C in trained individuals and concluded that exercise not weight, adiposity, or caloric intake was the most important determinant of the high plasma HDL-C seen in endurance athletes.

In contrast to these studies, Weltman, Matter, & Stamford (1980) reported that a 10-week program of mild aerobic exercise resulted in a substantial drop in the plasma

TC/HDL-C ratio, but incurred little change in BW or %BF. In addition, a 10-week program of caloric restriction by Weltman et al., (1980) resulted in a substantial loss in fat but little change in the plasma TC/HDL-C ratio. Thus, exercise rather than body fatness was suggested to be a key factor in alterations of plasma TC/HDL-C levels. Weltman, et al., (1980) and Clarkson et al., (1981) observed no relationship between BW and plasma TC or HDL-C; likewise, Ullrich, et al., (1987) and Schwartz (1987) found no relationship between plasma HDL-C and %BF.

Gaesser & Rich (1984) also failed to find a relationship between %BF and plasma HDL-C. He had males, ages 20 - 30 years train for 18 weeks on a bicycle ergometer (one group at a high intensity of 75 - 80%, one group at a lower intensity of 45%). There was no change in BW or plasma HDL-C levels, but significant decreases were seen in %BF. No correlational analyses were run between these variables, but Gaesser and Rich suggested that exercise training induced changes in plasma HDL-C were dependent upon pre-training levels rather than any losses in body fat.

Since a relationship between plasma HDL-C and LBM or %BF has been noted by many researchers (Rotkis et al., 1984; Moore et al., 1983; Goldberg et al., 1984; Blessing et al., 1987), it would be helpful to know the mechanism behind this linear, inverse relationship. However, little research has addressed this issue. Nikkila, Taskinen, Rehunen, & Harkonen (1978) found an increased level of lipoprotein lipase (LPL) activity in the skeletal muscle and adipose tissue of endurance-trained athletes. LPL has been found to degrade the triglyceride-rich lipoproteins (VLDL-C, LDL-C, and chylomicrons) (Goldberg & Elliot,1985). During exercise, the increase in blood flow causes large amounts of VLDL-C, LDL-C, and chylomicrons to mesh with LPL. Theoretically, more triglyceride could be hydrolyzed, thus breaking down LDL-C and forming more plasma

HDL-C (Defaux et al., 1982). It has been suggested that this may be a key mechanism in which exercise affects lipoprotein values.

Weight loss or reduced body fat may also be responsible for elevated levels of LPL in endurance-trained athletes (Goldberg & Elliot,1985). LPL in adipose tissue has been found to increase after weight loss, a finding also associated with increased plasma HDL-C values (Schwartz, 1987). It is still unclear, however, if exercise alone induces increased enzyme activity.

Summary

Cholesterol has been documented as one of the major risk factors of CHD. A positive relationship has been reported between plasma TC and CHD and a negative correlation reported between plasma HDL-C and CHD. Therefore, efforts to decrease plasma TC and increase HDL-C concentrations have been investigated. It appears that exercise can usually increase plasma HDL-C if the duration of training and the intensity is high enough, however, plasma TC, for the most part is difficult to decrease. Most studies have found that if the plasma HDL-C increases, that may be adequate in itself, since it is believed that a lower (below 5.0) plasma TC/HDL-C ratio contributes to a lower CHD risk.

CHAPTER III

JOURNAL MANUSCRIPT

THE EFFECT OF AN ENDURANCE AND WEIGHT TRAINING PROGRAM ON TOTAL CHOLESTEROL AND HIGH-DENSITY LIPOPROTEIN CHOLESTEROL

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THE EFFECT OF AN ENDURANCE AND WEIGHT TRAINING PROGRAM ON PLASMA TOTAL CHOLESTEROL AND HIGH-DENSITY LIPOPROTEIN CHOLESTEROL

by

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(ABSTRACT)

Research has reported that increased levels of plasma total cholesterol (TC) are directly related, while low levels of plasma high-density lipoprotein-cholesterol (HDL-C) are inversely related, to coronary heart disease. Regular physical exercise has been suggested as a method for reducing plasma TC and increasing plasma HDL-C. Thirty-one healthy, sedentary women (ages 18-30) were studied to determine the effects of a jogging, weight training, or a combined jogging and weight training program on plasma TC, HDL-C, and body composition. Experimental subjects were randomly assigned to the treatment conditions. The subjects trained three days a week for nine weeks. The R group ran for 30 minutes a session at 75% predicted maximum HR. The W group trained with weights utilizing exercises to strengthen all major muscle groups for one hour at 60% one repetition maximum the first 3 weeks and 75% one repetition maximum weeks 4 - 9. The RW group ran for 25 minutes a session at 75% predicted maximum HR, then lifted weights using the leg-strengthening exercises for 30 minutes, similar to the W group. Preceding and following the treatment period, plasma TC, HDL-C, body weight, and percent body fat was assessed for all four groups. Plasma TC was not significantly altered, although a downward trend was observed for all three treatment groups. Plasma HDL-C did not change over the treatment period for any group. The plasma TC/HDL-C ratio changed significantly among groups over the treatment period, with the R group decreasing their ratio from 3.5 to 2.9 ($p < .05$). No changes were noted in percent body fat, fat-free mass,

or body weight for any of the groups. The Pearson product-moment correlations performed between the changes in blood lipids and the changes in body composition found no significant relationships. The results of this study indicate that an exercise program consisting of endurance training for 30 minutes, 3 times per week, or weight training for one hour, 3 times per week, or a combination aerobic/weight training program 3 times per week is not adequate to significantly improve plasma TC or HDL-C in young females over a nine week period. However, significant improvements may be made in the plasma TC/HDL-C ratio which may decrease the risk for CHD.

Introduction

Coronary heart disease (CHD) has become the leading cause of death among the American population in this generation. Recently, there has been a decline in CHD mortality rates, however, deaths due to CHD continue to remain significantly above those due to cancer, the second leading cause of death. One of the primary risk factors contributing to this disease is hypercholesterolemia. Evidence from epidemiologic studies and the Lipid Research Clinics Coronary Primary Prevention Trial (1) indicate that the risk for CHD is directly related to plasma levels of total cholesterol (TC) and inversely to high-density lipoprotein-cholesterol (HDL-C) concentrations (2,3).

Research has indicated that the manner in which cholesterol is transported in the blood may be more critical in the development of CHD than plasma TC (4). Populations having higher levels of plasma HDL-C seem to have a lower incidence of CHD, whereas elevated levels of plasma low-density lipoprotein-cholesterol (LDL-C) seem to be related to a higher incidence of CHD (5, 6) While the exact mechanisms of action remain unclear, plasma LDL-C may contribute to CHD by its ability to infiltrate the arterial intima (7). Conversely, plasma HDL-C may exert its protective effect by transporting cholesterol away from the arterial wall to the liver for catabolism and excretion (7).

Physical activity has been proposed to have a possible protective effect against the development of coronary heart disease by lowering plasma TC and increasing concentrations of plasma HDL-C (8). Cross-sectional studies of the effects of exercise on plasma TC have generally demonstrated that people who regularly exercise aerobically have similar or only slightly lower plasma TC concentrations than less active people of similar ages (9, 10, 11), although others have reported significantly lower differences between active and inactive people (12). Power-trained athletes have demonstrated similar variations, in that some trained studies reported no significant differences in plasma TC

(13, 14), while other studies showed somewhat higher plasma TC values in the power-trained athlete (15, 16).

The effects of exercise on plasma HDL-C is also inconclusive. Increased plasma HDL-C levels have been seen in many studies when endurance- or weight-trained subjects were compared with inactive controls (10, 11, 12, 15, 17). However, other studies have reported no changes in plasma HDL-C (18, 19, 20, 21, 22)

This study sought to investigate not only the effects of an aerobic exercise program and the effects of a weight-training program on plasma TC and HDL-C, but also the effects of an exercise regimen which combined the two activities.

Methodology

Subjects. Prior to subject selection, permission to conduct the study was obtained from the Institutional Review Board for Research Involving Human Subjects. The 26 experimental subjects who agreeed to participate in this study were among volunteers who registered for a physical fitness class entitled "Bodybuilding and Fitness". The 5 subjects in the Control (C) group were volunteers from leisure sports activity classes, i.e., bowling and golf, and fitting the same criteria as the experimental subjects. Selection criteria included the following: (1) females, 18 - 30 years, (2) generally healthy, without any known health-related diseases, (3) sedentary for past 6 months, (4) non-smokers, (5) on no medication known to alter lipoprotein-cholesterols and (6) on no weight-loss program during participation in this study.

All subjects were asked to complete a detailed questionaire concerning medical history, family history, demographic information, cigarette smoking, alcohol consumption and physical activity. From the screening questionaire, it was determined that alcohol use was moderate, no subject was taking anabolic steriods, and none were smokers. Some were taking oral contraceptives. Diet was not assessed; however, subjects were asked to

maintain typical eating patterns throughout the course of the study in order to maintain body weight. The twenty-six subjects participating in the experimental treatments were randomly assigned to one of three groups and each group was randomly assigned to a treatment: running (R; n=8), weight training (W; n=8), or a combination of running and weight training (RW; n=10).

Laboratory procedures. All subjects were given both a written and an oral explanation of the study, including its risks, benefits, and procedures, prior to any testing. During the initial visit, height, weight, percent body fat (%BF) via hydrostatic weighing, and blood pressure were measured on each participant.

Pre-treatment values were obtained on each subject for the following variables: plasma TC, HDL-C, TC/HDL-C ratio, %BF via hydrostatic weighing, body weight (BW) and fat-free mass (FFM). Values for fat-free mass (FFM) were calculated using the %BF criterion score from hydrostatic weighing.

Blood samples were drawn from subjects prior to the treatment period and 48 hours following the last workout of week 9. Subjects were instructed not to eat for at least 3 hours prior to blood sampling. They were also given written instructions specifying recommended meals (i.e., whole-grain breads, fresh fruit and vegetables, low-fat milk, cheese, yogurt, etc.,) as well as foods to avoid (fried foods, fatty meats, mayonnaise, etc.). None of the subjects had been engaged in physical activity during the 12 hours immediately prior to blood sampling. Blood was drawn into vacutainers containing dry sodium ethylenediamine tetraacetic acid (EDTA). Blood was separated within 1 hour of collection by centrifugation. Plasma TC and HDL-C were determined according to enzymatic methods (23); plasma HDL-C was measured in the supernatant after precipitation of VLDL-C and LDL-C with a heparin and manganese-chloride solution (24). All analyses were performed in duplicate; additional assays were performed if values differed by more

than 5% for plasma TC and 10% for plasma HDL-C. Reliability estimates between duplicate pretest measures were performed for plasma TC and HDL-C (plasma TC - r = .99; plasma HDL-C r = .97). A known external standard control (Sigma) was utilized with each assay run to ensure reliability of measures (r = .89).

Subjects were weighed weekly to the nearest .25 kg on a calibrated scale. Hydrostatic weighing to determine body composition was performed as described by Katch and McArdle (26) in a rectangular, stainless steel tank. Underwater weight was measured while supine on a basket which was supported in the water by four load cells. Correction was made for residual volume as determined by the oxygen rebreathing technique of Wilmore, Vodak, Parr, Girandola, and Billing (27).

Exercise protocol. Workouts for the experimental groups were performed at the same time three days per week. Subjects assigned to R trained aerobically using a jogging program under direct supervision. Subjects began with a 20 minute jog, progressing to 25 minutes during the third week and 30 minutes for weeks 4 - 9. Due to low initial fitness levels, approximately 50% of the subjects walked during the first 3 weeks. Two subjects continued to walk/jog until they were continuously jogging by the fifth week. To keep subjects in a target heart range of 70 - 80%, heart rate was taken three times during exercise at ten-minute intervals, as well as before and after each exercise session.

The W and RW groups performed the first two out of four phases of Stone and O'Bryant's (28) periodization model on the Universal and Nautilus weight lifting apparatus. Only two phases were utilized (hypertrophy and basic-strength) in order to avoid undue variation in the exercise routine during the 9-week treatment period. The hypertrophy or adaption phase consisted of a program of high volume and low intensity (high repetitions and low weights). The basic-strength phase involved moderate volume and high intensity (fewer repetitions at a greater workload). One repetition maximum (1

RM) on the leg extension and leg press machines were taken on the W, RW, and C groups prior to and immediately following the treatment period. Biweekly measures were taken on the R and RW groups in order to assess changes in strength during the treatment period.

The W group lifted weights on the Universal and Nautilus machines for 50 - 60 minutes. Weeks 1 and 2 consisted of two to three sets of 8 - 12 repetitions of 60% of one repetition maximum for each of the exercises. Weeks 4 - 9 included three to four sets of 4 - 6 repetitions at 75% 1RM for each exercise. Microcycles were incorporated into the treatment program. On Mondays and Fridays of the adaption phase, subjects performed three sets of eight to twelve repetitions on the leg apparatus (Table 1) and two sets of eight to twelve repetitions on the arm apparatus. Wednesdays consisted of two sets on the leg apparatus and three sets on the arm apparatus. Weeks 3 - 9, subjects decreased repetitions to four to six and performed four sets on the leg apparatus and three sets on the arm apparatus on Mondays and Fridays, while lifting three sets on the legs and four sets on the arms on Wednesdays.

Subjects in the RW group ran during the first half of each exercise session. Progression from a 20 minute walk/jog to a 25 minute jog was similar to the R group. Subjects then lifted weights during the second half of each session. They performed the same leg strengthening exercises as the W group and 4 pieces of arm apparatus of their choice to complete 30 minutes of weight training. Attendance for all groups was greater than 90%.

Statistical procedures. All data were assessed using a two-factor (group and time) analysis of variance (ANOVA) with repeated measures. When statistical significant F values were noted, the appropriate Tukey post hoc test was utilized to identify which groups differed across time. A Pearson product-moment correlation analysis was performed on the changes in plasma TC, HDL-C, TC/HDL-C and the changes in BW,

%BF, and FFM to determine the relationship between variables. Statistical significance was set apriori at the 0.05 level. All data was analyzed using the Statistical Analysis System (SAS).

Results

Physical characteristics of age and height, and the dependent variables are presented in Tables 2 and 3. One-way analysis of variance (ANOVA) was performed on the pretest values for all dependent measures and indicated that the groups were similar except for plasma HDL-C. The Tukey post-hoc test revealed that the C group had a significantly greater plasma HDL-C than the R group at pre-test ($p < .05$).

There was a wide variation in plasma HDL-C (82 - 27 mg/dl). Only four of the 31 subjects had plasma HDL-C values below 50 mg/dl, while only two subjects had plasma TC values above 200 mg/dl. Subject 16 had a plasma TC level of 216 mg/dl, but she had a plasma HDL-C value of 74 mg/dl, which resulted in a low plasma TC/HDL-C ratio (2.9). Subject 21 had a plasma TC value of 226 mg/dl and a plasma HDL-C value of 50.5 mg/dl, which resulted in a plasma TC/HDL-C ratio of 4.5. This was the highest ratio seen in any group. It is evident from the data that these subjects were at a low-risk for CHD when considering plasma cholesterol.

Table 3 shows the means for %BF, FFM, and BW. There were no significant changes across time for any group and no significant interaction between treatment groups across time.

The averages for plasma HDL-C before and after the experimental period are shown in Figure 1. Two-way ANOVA (Table 5) indicated that a significant group effect was seen for plasma HDL-C. A follow-up test using the Tukey procedure indicated that the R group was significantly lower than the other 3 groups ($p < .05$) attributable to pre-test differences.

No significant differences were found among the remaining 3 groups, nor did ANOVA indicate significant group interaction across time during the treatment period.

The pre- to posttest mean changes for plasma TC are shown in Figure 2. The decrease in plasma TC for all groups combined was significant over time ($p < .05$). Exploring the differences between pretest to posttest for the individual groups via the Tukey post hoc test resulted in no significant differences. It was noted that while the Tukey test demonstrated no significance, a downward trend was seen for the experimental groups. The plasma TC changes from pre- to posttest were 8.2% for the R group, 7.3% for the W group and 4.2% for the RW group. There was no significant group interaction across time during the treatment period.

Group means for the plasma TC/HDL-C ratio are presented in Figure 3. A significant interaction among groups over time was noted ($p < .05$). Further testing by means of the Tukey procedure revealed significant differences in the response of plasma TC to treatment in the R group. The ratio for the R group decreased 17% (3.5 - 2.9; $p < .05$), while the ratio for the C group increased 13% (2.3 - 2.6; $p > .05$). No changes were found for the W and RW groups.

Correlational analyses were performed between the changes in blood lipids and changes in body composition. The change in plasma TC was positively related to the change in HDL-C ($r = .38$; $p < .05$) and the change in plasma TC/HDL-C ratio ($r = .39$; $p < .05$). The change in plasma HDL-C was positively related to the change in the ratio ($r = .60$; $p < .05$). When FFM changes were related to plasma TC and HDL-C, no significant correlations were found.

Discussion

The present investigation was designed to determine the effect of running, weight lifting, and a combination of running and weight lifting on plasma TC and HDL-C , and to

investigate the relationship between changes in plasma TC, HDL-C, TC/HDL-C and changes in BW, %BF, and FFM. Neither BW, %BF , nor FFM changed significantly in any of the exercising groups in this study. Many studies examining the effects of weight training on women have reported a significant decrease in percent body fat and increase in FFM with no corresponding changes in body weight (19, 21, 22, 29, 30). These studies have suggested that exercise was the factor which affected body composition by increasing lean weight and decreasing fat weight. It does not appear that the exercise treatments in the present study had any effect on body composition.

Neither epidemiologic nor prospective studies have produced a clear consensus as to a direct relationship between plasma TC and exercise (3,8,18, 21, 31). Although plasma TC of the treatment groups exhibited a downward trend, there were no significant differences at the posttest between the three experimental groups and the C group, which was in agreement with other longitudinal studies (17, 19, 21, 29).

Some cross-sectional studies have noted a lower plasma TC in individuals who are physically active as compared to inactive people. Wood et al., (12) demonstrated significantly lower, although not marked, plasma TC values for runners (200 mg/dl) when compared to matched sedentary controls (210 mg/dl, $p < .05$). Clarkson et al., (13), Hartung & Squires (32), and Martin, Haskell, & Wood (33) also noted significantly lower plasma TC values for physically active people involved in endurance exercise when compared to sedentary controls. However, these results were confounded by controls who were more obese (32, 33) or had dissimilar dietary habits (32) from the groups who were physically active. In another study, plasma TC was compared in runners and body builders matched for age and %BF to that of controls (14). It was shown that the two exercising groups had lower plasma TC values than the controls. On the other hand, others

have failed to show a significant difference in plasma TC concentration between matched sedentary and physically active groups (10, 14).

Findings from prospective studies have also generated varied results. While some have reported significant decreases in plasma TC after exercise (15, 22, 34, 35), others have noted little or no changes in plasma TC (14, 17, 20, 21, 29, 30, 36, 37). The present study demonstrated similar results to that of the latter studies, with no significant differences between pre- and posttest plasma TC values for any of the exercising groups.

Although there were no significant decreases in plasma TC, a downward trend was seen for all three experimental groups (a decrease of 8.2%, 7.3%, and 4.2% for R, W, and RW groups, respectively). It has been suggested that the decreases in the lipoprotein-cholesterol values are partly due to weight or fat loss (38). The Zutphen Study (38) concluded that every 1 kg change in body weight is accompanied by a 2 mg/dl change in the plasma TC level. However,neither body weight nor body fat changed for any of the groups in the present study.

An issue in this study not addressed in previous studies involves the RW group. No research has combined the 2 exercise regimens while investigating plasma TC and HDL-C. Many studies have seen decreases in plasma TC while exercising aerobically and many have seen plasma TC decrease after weight lifting. In agreement with, and in addition to these studies, the present study noted an apparent decrease in plasma TC with the combined exercise regimen. Because the decrease (4.2%) was less than the decrease seen for the R group (8.2%) or the W group (7.3%), it appears that the addition of aerobic activity to a weight training program would not enhance the possibility of lowering plasma TC values. Likewise, it appears that incorporating a weight training program into an aerobic workout could diminish the positive effect of aerobic activity on plasma TC.

A lack of change seen in the plasma HDL-C values may be attributed to several factors. Recently, total plasma HDL-C has been divided into two major subclasses: plasma HDL_2 and HDL_3. Plasma HDL_2 has been shown to be strongly correlated to reduced CHD incidence and coronary occlusion (39), whereas plasma HDL_3 seems to have no relation to CHD. Evidence suggests that physical activity alters the plasma HDL_2, but not plasma HDL_3 as seen in studies by Ballantyne, Clark, Simpson, and Ballantyne (40) and Wood et al., (20). Increased plasma HDL_2 values were seen after exercise training in both healthy (20) and CHD patients (40). Hence, it is possible that changes occurred in this study in the subclasses of plasma HDL-C which were not reflected in the total plasma HDL-C value. This lack of change is consistent with another study which reported plasma HDL-C subfraction changes for 17 sedentary, middle-aged men who exercised for 10 weeks without change in concentrations of total plasma HDL-C (41).

Plasma HDL-C values remained constant for all three treatment groups while decreasing for the C group. Most prospective studies have noted significant increases (15, 17, 19, 29) or slight increases (20, 21, 22) in plasma HDL-C after exercise. The experimental groups differed significantly from the C group, which can be attributed to significant pretest differences. The C group began the study with unusually high values of plasma HDL-C (69 mg/dl) for sedentary individuals, while the R group began with much lower values (49 mg/dl) than similar studies using young, sedentary females (18, 22, 29). By the end of the study, plasma HDL-C values for the C group had apparently dropped from 69 mg/dl to 58 mg/dl, no longer significantly different from the experimental groups. Similarly, other studies have demonstrated stable plasma HDL-C values throughout the duration of an exercise program (20, 21, 22). However, none of these studies have seen a decrease of plasma HDL-C in the C group, with no change in the experimental groups.

Recently, researchers have suggested that the plasma TC/HDL-C ratio is a better

indicator of CHD risk than plasma TC (2). The ratio was calculated in the present study and significant differences were seen between the R group and all other groups. While the ratio significantly decreased for the R group ($p < .05$), no changes were seen in the RW, W, and C groups. This decrease in the ratio was due primarily to the decrease in plasma TC rather than any improvement in plasma HDL-C, a result seen in other studies (22, 36).

Many studies have seen an increase in plasma HDL-C with a concomitant increase in muscle mass and no change in BW (15, 19, 29, 30). Nikkila (42) found an increase in LPL activity in skeletal muscle tissue in endurance-trained athletes. It appears that when muscle mass increases with physical activity, there is also an increase in LPL activity, which enhances catabolism of VLDL-C (10, 43). During VLDL-C catabolism, there is a "release of surface components (unesterified cholesterol, phospholipids, and C apolipoproteins) which enter the plasma HDL-C density range and by association with plasma HDL-C_3 or with plasma HDL-C precursors secreted by the liver and intestine, form plasma HDL-C_2" (43). These processes are mediated by lecithin cholesterol acyltransferase (LCAT) although the exact details of the reaction are unknown. In the present study, no change was seen in muscle mass for any group, which may have limited any increase in skeletal muscle LPL activity. Consequently, there was not the increased catabolism of triglyceride lipoprotein-cholesterols and the subsequent formation of additional plasma HDL-C (42, 44).

It has been suggested that changes in cholesterol concentrations are dependent upon initial pre-test values (19). In other words, those individuals exhibiting initially low plasma TC values usually fail to change significantly. It is not clear whether the reciprocal suggestion stands for plasma HDL-C. If so, since plasma HDL-C values were already high in the present study, further increases may not have been observed.

The present study demonstrated decreases in plasma TC for the exercise groups, while plasma HDL-C levels remained the same. According to Wood et al (20), when exercise groups exceeded 4 miles per week, decreases were seen in plasma TC. It was not until the subjects ran between 8 and 13 miles per week that increases in plasma HDL-C were seen. However, these changes were reported after subjects had been exercising for one year. The R and RW groups in the present study were estimated to run approximately 9 and 7 1/2 miles per week for nine weeks, respectively. Significant increases in plasma HDL-C may have been demonstrated had both groups been able to either increase weekly mileage or increase the length of the training program. Another factor may have been the fact that these subjects had low initial fitness levels and were therefore, unable to run continuously for 30 and 25 minutes until the fourth week. Therefore, they ran at this weekly mileage for only 6 weeks. Moll et al., (18) also failed to induce significant changes in female runners after 6 weeks of running 5 days per week for 30 - 45 minutes. Farrell & Barboriak (19) induced significant increases in plasma HDL-C in female runners after 8 weeks of training. They ran 4 days per week for 30 minutes per day, which added at least 3 miles per week to the training regimen. This increase in mileage, plus the two extra weeks of training may explain the differences in the results between the two studies. Results from studies by Williams et al., (45) demonstrated the need for long-term exercise in order to induce changes in plasma HDL-C cholesterol.

Forty-eight previously sedentary men (30 - 55 years) participated in a running program lasting one year. Initially, subjects jogged for 20 minutes at 70 - 85% max HR achieved during a baseline treadmill test. During the third week, subjects added a fourth day , and then increased to 5 days per week around the seventh week. At approximately six weeks, the exercise group was encouraged to increase the duration of running gradually

until each session reached 45 minutes. Approximately nine months were required before significant increases in plasma HDL-C were observed..

Although statistical significance was not observed between groups, it is important to note the apparent effectiveness of the exercise programs in increasing strength and aerobic capacity. Strength increases were noted in both the W and RW groups; the W group increasing leg strength via the leg press by 56% and the RW group increasing leg strength by 67%. Likewise, improvements in aerobic capacity were noted in the R and RW groups by means of a 12-minute run performed prior to and following the 9 week exercise program. The R group increased the distance run by 40% and the RW group increased by 51%. Because of these changes in leg strength and aerobic capacity, it would seem apparent that these programs, while not finding statistical significance for blood lipids or body composition, were nevertheless, rigorous enough to bring about changes in the parameters to which each exercise program was intended.

Researchers examining the relationship between blood lipids and body composition have noted varied results. Weltman et al., (46) found that a 10-week program of mild aerobic exercise resulted in a substantial drop in the plasma TC/HDL-C ratio, but incurred little change in BW or %BF. In addition, a 10-week program of caloric restriction resulted in a substantial loss in fat but little change in the plasma TC/HDL-C ratio (46). Thus, exercise rather than body fatness was suggested to be a key factor in alterations of plasma TC/HDL-C levels. Weltman et al., (46) and Clarkson, et al., (13) observed no relationship between BW and plasma TC or HDL-C, which was in agreement with the present study. Likewise, Ullrich, et al., (30) and Schwartz (47) found no relationship between plasma HDL-C-and %BF. However, many studies did observe significantly positive correlations between plasma TC and %BF and negative correlations between plasma HDL-C and %BF (10, 20, 29). Thompson, Cullinane, & Eshleman (48) studied the effect of exercise

cessation on plasma HDL-C in trained individuals and concluded that exercise not weight, adiposity, or caloric intake was the most important determinant of the high plasma HDL-C seen in endurance athletes.

In summary, the results of the present study suggest that the three training programs tended to decrease plasma TC, though non-significantly. No changes in plasma HDL-C were noted among the three training groups, while the C group decreased over the experimental period. The plasma TC/HDL-C ratio indicated significant differences between the R and all other groups across time. The ratio for the R group decreased while no changes were noted for the the W, RW, and C groups. There were no significant differences over time for BW, %BF, or FFM. In the present study, it could not be shown that aerobic exercise was any more or less effective than the weight training program. The relationship between blood lipids and body composition remains speculative, with further research necessary to clarify the relationship between these variables.

Table 1. Weight training Apparatus utilized for W and RW groups

Universal Apparatus	Nautilus Apparatus
Lower Body	Lower Body
Leg press	Calf raises
Leg extension	
Leg curls	
Calf-raises	
Upper Body	Upper Body
Arm Curls	Chest flies
Bench press	Chest press
Tricep press	Shoulder flies
Shoulder shrugs	Military press
Latissimus dorsi pulldowns	Abdominals
Military press	

Table 2. Subject characteristics.

Variable	**R**	**W**	**RW**	**C**
n	8	8	10	5
Age (yrs)	21.1	21.0	20.1	24.3
	(1.0)	(0.5)	(0.3)	(1.5)
Height (cm)	165.4	164.7	165.5	162.0
	(2.8)	(2.5)	(2.2)	(2.2)

Values expressed as group means with SEM in parentheses
R = Running; W = Weight training; RW = Running/Weight training; C = Control group

Table 3. Group means for plasma TC, HDL-C, TC/HDL-C, %BF, FFM, and BW.

Variable	R		W		RW		C	
	pre	post	pre	post	pre	post	pre	post
Plasma TC (mg/dl)	158.3 (10.8)	144.9 (11.8)	172.7 (4.6)	160.1 (6.2)	165.6 (10.7)	158.7 (9.7)	147.7 (9.6)	148.3 (12.6)
Plasma HDL-C (mg/dl)	49.1[a] (4.4)	50.6 (4.5)	63.1 (3.7)	63.0 (2.8)	61.5 (3.1)	63.9 (3.0)	68.5[a] (6.3)	58.4 (2.7)
Plasma TC/HDL ratio (mg/dl)	3.5 (.41)	2.9+ (.26)	2.8 (.19)	2.6 (.09)	2.7 (.19)	2.6 (.24)	2.3 (.25)	2.6 (.23)
%BF Hydrostatic (%)	25.7 (2.4)	24.1 (3.1)	25.4 (3.5)	25.6 (3.5)	24.5 (1.9)	24.8 (1.5)	22.8 (3.9)	22.4 (3.6)
FFM (kg)	48.4 (1.0)	49.1 (1.1)	42.9 (1.3)	43.6 (1.3)	46.5 (1.6)	46.8 (1.5)	44.2 (3.0)	44.3 (2.9)
BW (kg)	64.4 (1.7)	64.4 (1.9)	58.7 (3.7)	59.7 (3.4)	62.0 (2.9)	62.7 (2.9)	57.7 (1.7)	57.6 (1.6)

Values expressed as group means with SEM in parenthesis
*p < .05 between HDL pretest scores across groups
[a]pretest values in same row with same letter are significantly different
+indicates significant change from pre- to posttest
R = running group; W = weight training group; RW = running/weight training group; C =control group
TC = total cholesterol, HDL-C = high-density lipoprotein-cholesterol; TC/HDL-C = total cholesterol/high-density lipoprotein-cholesterol; %BF = percent body fat; FFM = fat-free mass; BW = body weight

Table 4. Statistical Summary of Analysis of Variance

Variable		TC	HDL-C	TC/HDL-C	%BF	FFM	BW
Source	df	ms	ms	ms	ms	ms	ms
Group	3	1058.7	680.8*	1.55	19.18	91.6	126.7
$Error_1$	27	1335.5	180.2	6.9	120.01	37.9	152.2
Time	1	947.7*	36.9	0.33	1.78	2.95	2.36
Group/Time	3	126.0	95.9	0.36*	3.47	0.26	0.92
$Error_2$	27	201.9	43.2	0.088	4.25	2.6	1.09

*$p < .05$
TC = total cholesterol;
HDL-C = high-density lipoprotein cholesterol
TC/HDL-C = total cholesterol/high-density lipoprotein cholesterol ratio
%BF = percent body fat
FFM = fat-free mass
BW = body weight

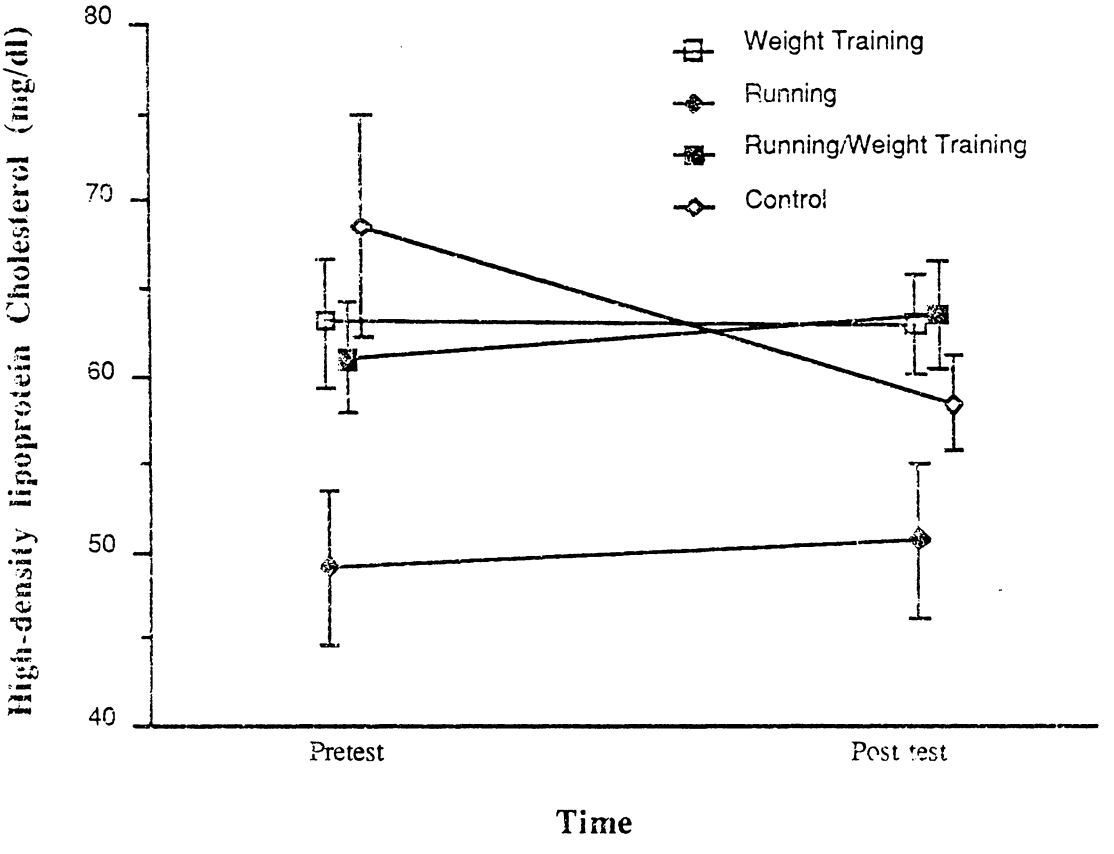

Figure 1. Group means and SEM for plasma high-density lipoprotein cholesterol for the Running, Weight Training, Running/Weight Training and Control groups.

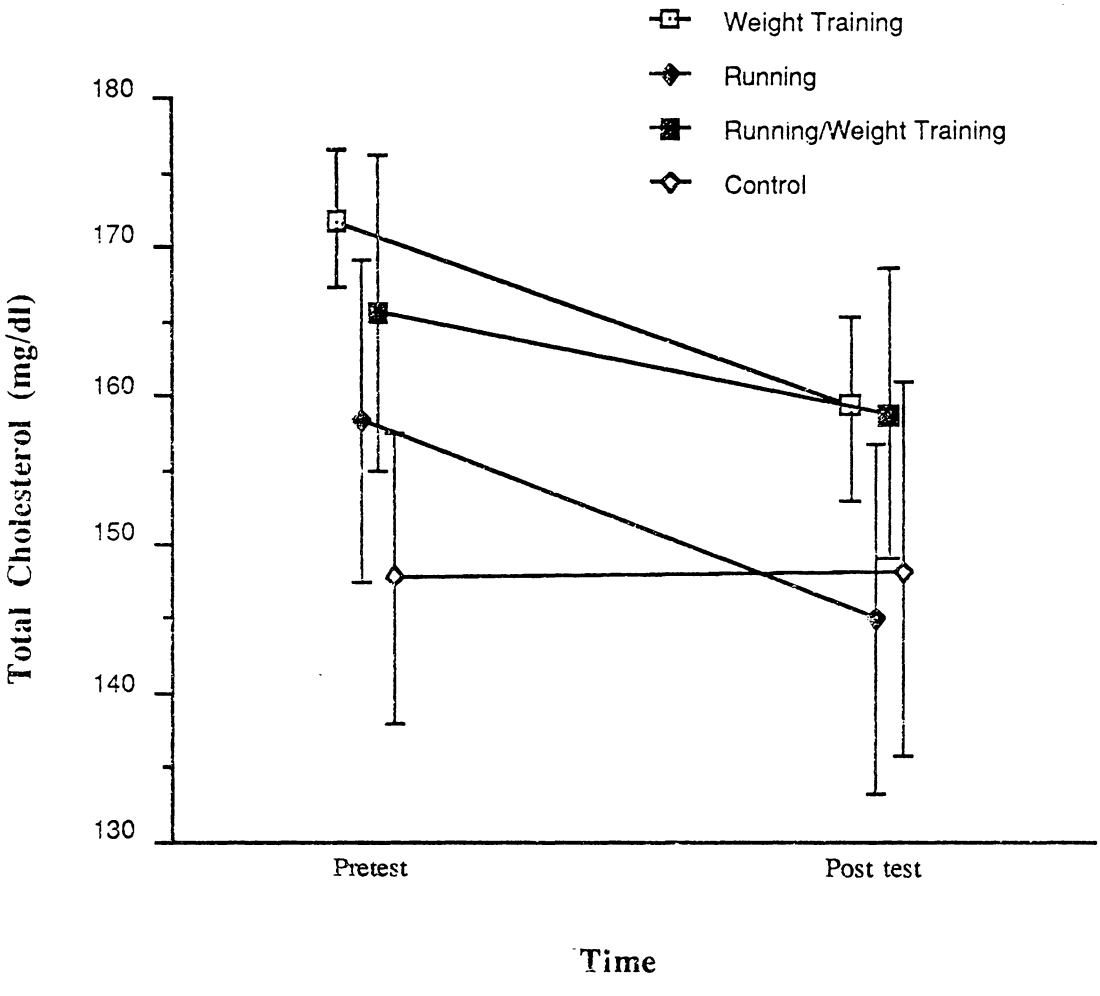

Figure 2. Group means and SEM for plasma total cholesterol for the Running, Weight Training, Running/Weight Training, and Control groups.

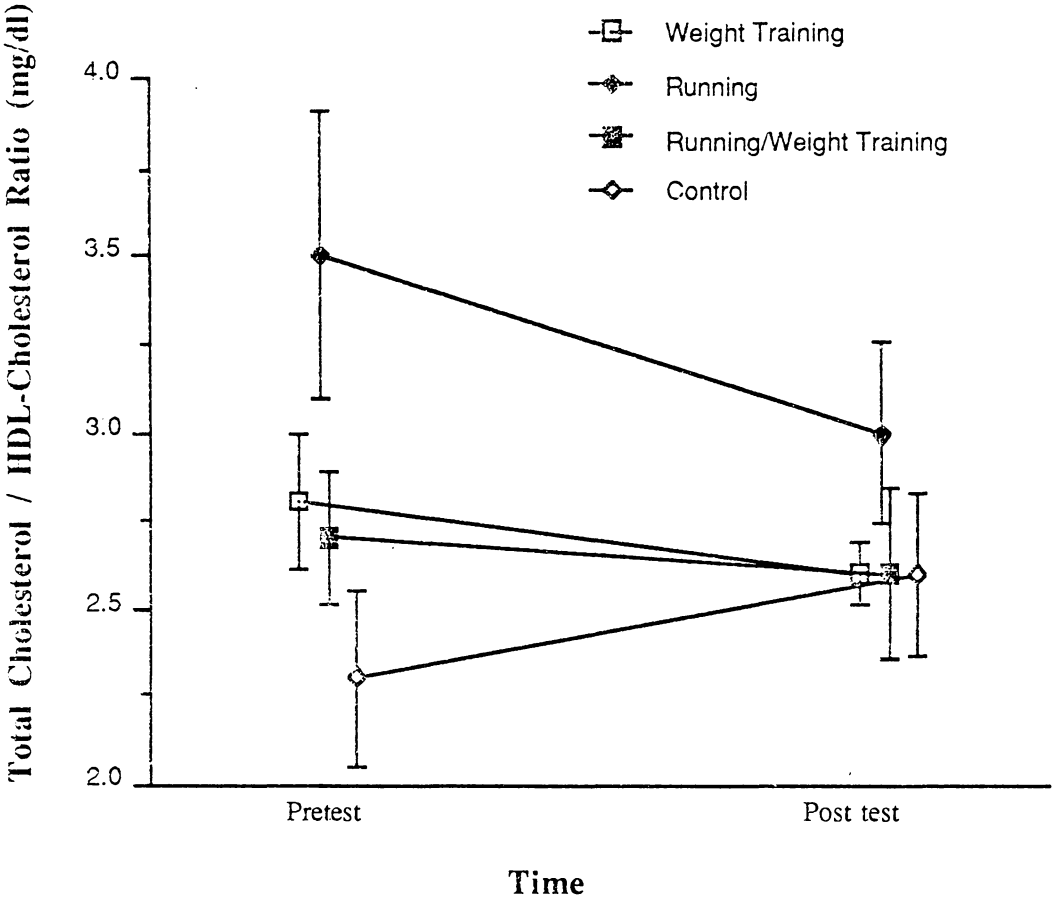

Figure 3. Group means and SEM for plasma total cholesterol / high-density lipoprotein-cholesterol ratio for the Running, Weight Training, Running/Weight Training, and Control groups.

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Chapter IV

Summary and Research Recommendations

Summary

Coronary heart disease has become the leading cause of death among the American population in this generation. There has been a decline in CHD mortality rates, however, deaths due to CHD continue to remain significantly above those due to cancer, the second leading cause of death. One of the primary risk factors contributing to this disease is hypercholesterolemia. Evidence from epidemiologic studies and the Lipid Research Clinics Coronary Primary Prevention Trial (1984) indicate that the risk for CHD is directly related to plasma levels of total cholesterol (TC) and inversely to high-density lipoprotein - cholesterol (HDL-C) concentrations (Lipinska & Gurewich, 1982; McCunney, 1987).

Research has indicated that the manner in which cholesterol is transported in the blood may be more critical in the development of CHD than plasma TC (Miller & Miller, 1975). Populations having higher levels of plasma HDL-C seem to have a lower incidence of CHD, whereas elevated levels of low-density lipoprotein-cholesterol (LDL-C) seem to be related to a higher incidence of CHD (Gordon et al., 1977; Enger et al., 1979;) While the exact mechanisms remain unclear, plasma LDL-C may contribute to CHD by its ability to infiltrate the arterial intima (Ross, 1986). Conversely, plasma HDL-C may exert its protective effect by transporting cholesterol away from the arterial wall to the liver for catabolism and excretion (Ross, 1986).

Physical activity has been proposed to have a possible protective effect against the development of coronary heart disease by lowering plasma TC and increasing concentrations of plasma HDL-C (Goldberg & Elliot, 1985). Cross-sectional studies of the effects of exercise on plasma TC have generally demonstrated that people who regularly exercise aerobically have similar or only slightly lower plasma TC concentrations than less active people of similar ages (Adner & Castelli, 1980; Moore et al., 1983; Morgan et al.,

1986), although others have reported significant differences between active and inactive people (Wood et al., 1976). Power-trained athletes have demonstrated similar variations, in that some studies reported no significant differences in plasma TC (Clarkson et al., 1981; Hurley et al., 1984), while other studies showed somewhat higher plasma TC values in the power-trained athlete (Johnson et al., 1983; Farrell et al., 1982).

The effects of exercise on plasma HDL-C are also inconclusive. Increased plasma HDL-C levels have been seen in many studies when previously sedentary endurance- or weight-trained subjects were compared with inactive controls (Wood et al., 1976; Johnson et al., 1983; Moore et al., 1983; Morgan et al., 1986; Blessing et al., 1987). However, other studies have reported no changes in plasma HDL-C (Moll et al., 1979; Farrell & Barboriak, 1980; Wood et al., 1983; Gaesser & Rich, 1984; Goldberg et al., 1984)

The purpose of this study was to investigate not only the effects of an aerobic exercise program and the effects of a weight-training program on plasma TC and HDL-C, but also the effects of an exercise regimen which combined the two activities. Four groups of females, 18-30 years of age, participated in a nine week exercise program. The four groups were as follows: 1) running (R), 2) weight training (W), 3) running/weight training (RW), and 4) control (C). The three treatment groups exercised three times per week. The R group ran for 30 minutes at an intensity corresponding to 75% of their predicted maximum HR. Five HRs were taken during each training session: pre-, post-exercise, and three times during the session, approximately 10 minutes apart. The W group lifted weights for one hour at 60% of their 1-RM for the first two weeks of the study, and 75% of their 1-RM for weeks three through nine. They performed various leg and arm exercises on the Universal and Nautilus apparatus. The RW group ran for 25 minutes at a HR of 75% of their predicted maximum HR. The second half of their workout consisted of weight training at the same percentages as the W group using the same

exercises. However, they performed all the leg exercises and only enough arm exercises to fill the 30 minute time period. Attendance for the experimental groups was greater than 90%.

A two factor (group and time) repeated measures analysis of variance (ANOVA) was conducted to measure pre- and post-test differences on plasma TC, HDL-C, TC/HDL-C, percent body fat (%BF), fat-free mass (FFM), and body weight (BW) for R, W, RW, and C. ANOVA was also computed for weekly BW measures for the three treatment groups. Differences between the groups over time were assessed using a two-factor (group and time) analysis of variance (ANOVA) with repeated measures. When statistically significant F values were noted, the appropriate Tukey post hoc test was utilized to identify which groups differed across time. A Pearson product-moment correlational analysis was performed on changes in plasma TC, HDL-C, and TC/HDL-C ratio and changes in %BF, FFM, and BW. Statistical significance was set apriori at the 0.05 level. All data were analyzed using the Statistical Analysis System (SAS).

One-way (ANOVA) performed on the pre-test values for all dependent measures indicated that the groups were similar except for plasma HDL-C. The Tukey post-hoc test revealed that the C group had a significantly greater plasma HDL-C than the R group ($p < .05$). Percent body fat, FFM, and BW demonstrated no significant changes across time for any group and no significant interaction between treatment groups across time.

A two-way ANOVA indicated a significant group effect for plasma HDL-C. A follow-up test using the Tukey procedure indicated that the C group was significantly higher than the other 3 groups ($p < .05$) attributable to pretest differences. No significant differences were found among the remaining 3 groups, nor did ANOVA indicate significant group interaction across time during the treatment period.

The decrease in plasma TC for all groups combined was significant over time ($p < .05$). Exploring the differences between pretest to posttest for the individual groups via the Tukey post hoc test resulted in no significant differences. It was noted that while the Tukey test demonstrated no significance, a downward trend was seen for the experimental groups. The plasma TC changes from pre- to posttest were 8.2% for the R group, 7.3% for the W group and 4.2% for the RW group. There was no significant group interaction across time during the treatment period.

A significant interaction among groups over time was noted for plasma TC/HDL-C ratio ($p < .05$). Further testing by means of the Tukey procedure revealed significant differences in the response of plasma TC to treatment in the R group. The ratio for the R group decreased 17% (3.5 - 2.9; $p < .05$), while the ratio for the C group increased 13% (2.3 - 2.6; $p < .05$). No changes were found for the W, RW, or C groups.

Correlational analyses were performed between the changes in blood lipids and the changes in body composition. The change in plasma TC was positively related to the change in plasma HDL-C ($r = .38$; $p < .05$) and the change in plasma TC/HDL-C ratio ($r = .39$; $p < .05$). The change in plasma HDL-C was positively related to the change in the ratio ($r = .60$; $p < .05$). When FFM changes were related to lipoprotein-cholesterol changes, no significant correlations were found.

It is interesting to look at the individual changes from the beginning to the end of the treatment period. Five of eight subjects in the R group decreased plasma TC, while five increased plasma HDL-C levels. The plasma TC/HDL-C ratio decreased for six subjects. The W group demonstrated similar changes. Seven of eight subjects decreased plasma TC, while four subjects increased plasma HDL-C. Five of the eight decreased their plasma TC/HDL-C ratio. The RW group noted a decrease in plasma TC in four of ten subjects while reporting an increase in plasma HDL-C values in six subjects. On the average, a

greater number of subjects improved blood lipid values than those not demonstrating changes. Had the treatment period been longer or more miles run per week, even greater changes may have occurred. There were no changes in %BF, with the exception of Subject 13. Her percentage of body fat decreased from 17.6 - 9.8. There was a concomitant decrease of 3 kg in BW; however, it seem unlikely that the subject lost 7.8 %BF, since the loss of BW was that small.

Research Implications

Plasma TC is positively related and HDL-C is negatively related with the incidence of atherosclerosis and CHD, therefore, researching methods for decreasing plasma TC levels are crucial. If an effective program designed to decrease plasma TC and increase plasma HDL-C were determined, the risk of disease could be greatly reduced.

The results of this study suggest that short-term exercise programs, whether aerobic or weight training, are ineffective for inducing statistically significant changes in lipoprotein-cholesterol values in young females. However, an apparent trend for lower plasma TC was observed in all three treatment groups. Plasma HDL-C remained the same, but because plasma TC decreased (NS), the plasma TC/HDL-C ratio for the R group improved significantly, thereby, decreasing the risk for CHD.

Although statistical significance was not observed between groups, it is important to note the apparent effectiveness of the exercise programs in increasing strength and aerobic capacity. Strength increases were noted in both the W and RW groups; the W group increasing leg strength via the leg press by 56% and the RW group increasing leg strength by 67%. Likewise, improvements in aerobic capacity were noted in the R and RW groups by means of a 12-minute run performed prior to and following the 9 week exercise program. The R group increased the distance run by 40% and the RW group increased by 51%. Because of these changes in leg strength and aerobic capacity, it would seem

apparent that these programs, while not finding statistical significance for blood lipids or body composition, were nevertheless, rigorous enough to bring about changes in the parameters to which each exercise program was intended.

Although no improvements were seen in %BF, FFM, or BW, there is the possibility that improvements would have occurred if the frequency and duration of the exercise programs were increased. The amount of exercise necessary to result in increased plasma HDL-C levels is an important "dose-response" issue defined by type, frequency, intensity, and duration of activity as well as kcalories expended. The research confirms that exact guidelines have not been agreed upon, however they have shown that before a beneficial effect on plasma HDL-C can be achieved from exercise, a threshold needs to be surpassed. A running program of 3-4 days per week at 70-85% intensity would seem beneficial in improving lipoprotein values, if mileage exceeded at least 12 miles per week. This would approximate 1,200 kcal, which was similar to the lower threshhold suggested by Superko et al., (1985). It is difficult to say how long an individual should continue an exercise program before lipoprotein improvements are found. If an individual exercises consistantly, changes may be seen after 12 weeks of aerobic training, however, it may take as long as nine months (Williams et al., 1982). It has been suggested that the changes in lipoprotein concentrations last only as long as the training period (Lopez et al., 1974; Danner et al., 1984). Therefore, a lifetime program of physical activity is recommended in order to hold plasma TC to a lower limit, with an accompanying high plasma HDL-C level.

Recommendation and Future Research

This investigation leaves many unanswered questions about the effects of exercise on plasma TC and HDL-C. The following recommendations for further study have been made to supplement the results of this investigation:

1. Determination of other lipoprotein-cholesterols such as LDL-C, VLDL-C, and the subfractions plasma HDL_2 and HDL_3 would give greater insight into the details of changes occurring within the lipoproteins.

2. A great deal of research has looked at endurance exercise and cholesterol. However, conclusive evidence on the frequency, intensity, and duration of exercise necessary to induce improvements in lipoprotein-cholesterol levels is still lacking. A study using groups which ran different miles per week (i.e., group 1 = 8 miles per week; group 2 = 16 miles per week; group 3 = 20 miles per week) may be able to better define the lower limits for inducing changes in cholesterol values. Further research on duration of an exercise program would also be beneficial. A follow-up of this study investigating how lipoprotein-cholesterol reacts to the termination of an exercise program would be beneficial in supporting a lifetime exercise program as a means for decreasing cholesterol levels.

3. More research is necessary regarding the effects of changes in plasma HDL-C to determine if risk of disease is actually reduced as plasma HDL-C levels increase.

4. Research on weight training versus plasma lipoprotein-cholesterol has produced varied results. Continued research on the type, intensity, frequenc, and duration of a weight-training regimen is needed to further define the benefits to lipoprotein-cholesterols.

5. The majority of current literature has focused on individuals with normal levels of cholesterol. Since individuals with abnormal levels of cholesterol are at a higher risk for CHD, investigation of the effect of exercise on those people would be beneficial.

6. The present study attempted to keep the caloric expenditure constant. Similar plasma TC and HDL-C changes were seen in all of the treatment groups. However, those who weight trained had to work out for one hour, while the R group only exercised for 30 minutes. Would any differences be noted in plasma TC and HDL-C between R and W if runners exercised for one hour, also?

7. Prior to this experiment, no research had been done on the effects of a combined running/weight training program on plasma HDL-C. Looking at the physiological benefits of this training program as compared to either program alone may uncover additional benefits to lipoprotein cholesterol levels and patterns not observed with the either of the separate programs (i.e., runners including weight training might expect strength gains; likewise, weight trainers adding a running segment to their exercise routine might increase cardiovascular parameters).

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APPENDIX A

METHODOLOGY

METHODOLOGY

Plasma total cholesterol (TC) and high-density lipoprotein-cholesterol (HDL-C) levels were measured at the beginning and end of a nine week treatment period. During this period, the subjects participated in one of three treatments: weight lifting (W), running (R), or running and weight lifting (RW). The control group (C) was composed of volunteers from the university family which met the specified criteria.

SELECTION OF SUBJECTS

Prior to the selection of subjects, permission from the Institutional Review Board for Research Involving Human Subjects was obtained to conduct this study (Appendix B). The 26 students participating in the three treatment groups were vounteers who registered for a university physical education course (Bodybuilding and Fitness for Beginners). The subjects were selected according to the following criteria:

1. Females, ages 18-30;
2. Generally healthy individuals without any known health-related problems such as CHD, diabetes, hypertension (>140/90), gout, or orthopedic problems;
3. Uninvolved in a weight training program for at least the previous 6 months;
4. Uninvolved in a strenuous aerobic conditioning program for at least 6 months (ie., no more than 2 times per week and 20 minutes in duration);
5. Nonsmokers (ie., never smoked or refrained from any type of smoking (cigarettes, cigar, pipe, chew) for 6 months or more (Stubbe, Eskilsson, & Nilsson-Ehle, 1982);
6. On no medication known to alter plasma concentrations of lipoproteins (Glueck, 1985);

7. On no type of active weight-loss program during their participation in the study;

SAMPLING PROCEDURES

All students were given an oral explanation of the testing and the treatment procedures. Those students consenting to participate completed a general medical questionaire (Appendix C) describing their present health status, and a time was arranged when pretest, baseline measures could be taken. All potential subjects were given written instructions (Appendix E) concerning pretesting measures (ie., appropriate attire, fasting time for blood, etc). Eligible subjects were randomly assigned to one of the three groups which were randomly assigned to three treatments: weight lifting, running, or weight lifting and running.

The control group consisted of 5 females who fit the same criteria as the experimental groups. The same pre- and post-test measures were also administered to both control and experimental groups.

A pilot study was conducted in order to allow the researcher and assistants to become familiar with the testing procedures. Blood samples were tested for plasma TC and HDL-C the final week of the pilot study to establish technique and a reasonable level of consistency in data collection and analysis.

EXPERIMENTAL PROCEDURES

Subjects came to the testing site (Human Performance Lab, War Memorial Gym) where they first read and signed an informed consent form (Appendix D). Baseline measures were taken at this time which included height, weight, blood pressure (using a stethoscope and a mercurial sphygmomanometer), %BF (via hydrostatic weighing), and 1RM on the Universal leg press and leg extension apparatus. Blood was drawn in order to measure plasma TC and HDL-C levels. To test reliability, subjects were retested on leg

strength approximately 72 hours after initial measures. Test administrators remained the same between pre- and posttest measures.

Percent body fat was estimated by means of hydrostatic weighing (Katch & McArdle, 1983), using a water-filled stainless steel weighing tank. Calibration within the tank was measured by means of placing a known weight (4.21 kg) in the basket and turning on the recorder. The calibration line was recorded and used to determine scores. Subjects were first weighed dry in only a swimsuit, then weighed wet by laying in a supine position on the basket which was suspended in the tank by 4 load cells. Underwater weight was recorded on a recorder and by digital display. After the subject had expelled all her air and was laying still in a supine position, the readings were recorded and the trial was completed. Eight trials were recorded. The 3 highest scores were averaged and used as the criterion score. Body weight underwater was calculated using the recorder displacement from the calibration weight in a ratio to calculate kilograms and then converted to pounds. The following equation was utilized: BD = BW/BW - UWW/H2O density - RLV. (BD = body density; BW = body weight; UWW = underwater weight; H2O density=water density; and RLV=residual lung volume) (Keys and Brozek, 1953). Percent BF was calculated using the Siri (1961) equation where %BF = (4.95/BD - 4.50) x 100.

To complete the equation for %BF, residual volume was measured (Wilmore, Vodak, Parr, Girandola, & Billing,1980). Vital capacity was first measured in order to estimate maximal exhalation. Three trials were taken, with the highest value being utilized. This value was multiplied by .85, which indicated the volume of O2 that was needed to fill the anesthesia bag. In a prone position, each subject was fit with a mouthpiece attached to an anesthesia bag. A metronome was set at 60 and used to maintain the breathing rhythm equivalent to 1 breath/2 seconds. After approximately one minute of breathing room air, the subject exhaled as much air from the lungs as possible. She then signaled to the

technician who switched to a bag filled with 100% oxygen. Again, the subject was to breathe normally for about 6-7 breaths, followed by another maximal exhalation after which the technician switched the air in the bag back to room air. Oxygen and carbon dioxide percentages of the air in the anesthesia bag were analyzed and used to calculate residual volume.

Seven milliliters of blood were drawn into vacutainers containing dry ethylenediamine tetracetic acid (EDTA). Vacutainers were inverted for thorough mixing, and refrigerated until spun in an IEC HN-S Centrifuge for 20 minutes (speed = 1/2) to separate the plasma from the red blood cells. Plasma was placed into a 12 x 75mm capped culture tube, labeled, and refrigerated. Assays were run within one week of collection. plasma TC was determined by enzymatic methods using Sigma Cholesterol Kit #351-50 (Allain, Poon, Chan, Richmond, & Fu,1974); the cholesterol component of plasma HDL-C was determined from the supernatant obtained after precipitation of VLDL-C and LDL-C by a heparinized manganese-chloride solution, according to the method of Albers, Warnick, and Chenng (1978). Duplicate assays were run for each subject on plasma TC and HDL-C; additional assays were performed if the values were more than 5% or 10%, respectively.

DETERMINATION OF PLASMA TOTAL CHOLESTEROL

1. To BLANK test tube, add 0.02 ml distilled water.
2. To STANDARD test tubes, add 0.02 ml each of 100, 200 and 400 mg/dl cholesterol calibrator. (These were used for the TC standard curve.) Run duplicates.
3. To TEST test tube, add 0.02 ml plasma. Run duplicates.
4. To each test tube, add 1.0 ml cholesterol reagent*. Cover with parafilm and invert several times to mix well.

5. Incubate samples at 37 degrees C for 15 minutes.
6. Add 1.0 ml physiological saline to all samples, mixing well.
7. Read and record absorbance of STANDARD AND TEST vs. BLANK as reference at 500 + 15 nm. Complete readings within 30 minutes.
8. Calculate plasma TC as follows:

$$\text{Plasma TC (mg/dl)} = \frac{\text{TEST}}{\text{STANDARD}} \times 100$$

*Reagent contains phosphate buffer, 0.1 mol/L, 4-aminoantipyrine, 0.8 mmol/L, p-hydroxybenzenesulfonate, 20 mmol/L, cholesterol oxidase (microbial), >200 U/L, esterase (microbial), >150 U/L, and peroxidase (horseradish), >25,000 U/L, with stabilizers and fillers.

DETERMINATION OF PLASMA HDL-C

Reagents:

1. Manganese chloride solution 1.06M: (MNCL2 4H2O - MW = 197.91) Weigh out 209.78 g MnCl2 H2O and dissolve in a small amount of distilled water. Dilute to 1 liter volume with distilled water. This solution is thought to be stable indefinitely. Invert. Place in opaque storage container.
2. Sodium chloride 0.15M (physiological saline): Weigh out 8.77 g NaCl and dissolve in about 500 ml distilled water. Bring up to 1 liter volume with distilled water. Place in opaque storage container.
3. Heparin 40,000 units/ml: Weigh out 0.280 g heparin and dissolve in 1 ml 0.15 M saline. Use a very small glass vial to prepare solution, since volume is so slight. Vortex vigorously. Let set in order to ensure all the heparin is in solution. PREPARE FRESH FOR EACH RUN.

4. Combined heparin-manganese reagent: Add 0.6 ml 40,000 units heparin/ml to 10 ml 1.06M $MnCl_2 4H_2O$. PREPARE FRESH FOR EACH RUN.

PLASMA HDL-C PROCEDURE

1. Warm plasma samples to room temperature.
2. Mix plasma samples well. Using a calibrated Eppendorf macropipet, transfer 1 ml plasma into small disposable test tubes. Run duplicates.
3. Mix combined heparin-manganese reagent (See #3 and 4 above). Vortex well. Using a calibrated Eppendorf micropipet, transfer 0.1 ml combined reagent into each tube. A precipitate will form immediately.
4. Vortex each sample lightly and cover with Parafilm.
5. Allow the samples to stand at room temperature for 10 minutes.
6. Centrifuge samples in the Sorvall RT6000 refreigerated centrifuge for 30 minutes (speed = 3100 rpm; temperature = 15 degrees C). A hard pellet will form on the bottom of the tube.
7. Transfer most of the supernatant with a disposable pipet into a 7 ml test tube. Label and cover with Parafilm.
8. Treat supernatant like unfractionated plasma to determine HDL-C concentration. EXCEPTION: Add EDTA to cholesterol reagent as follows: 1.) Add 5.844 g EDTA per 100 ml distilled water, 2.) add 40 microliters EDTA solution per 1 ml of reagent.

EXPERIMENTAL PROCEDURES

Subjects assigned to the W group participated in a 9-week exercise program of progressive weight training using Universal and Nautilus machines. Subjects exercised three days per week for approximately 50 - 60 minutes each session. Training on the

Universal apparatus included leg curls, leg press, leg extension, bench press, bicep curls, lat pulls, and tricep press, while chest curls, chest press, shoulder flies, military press, calf raises, and abdominals were performed on the Nautilus. Weeks 1 - 3 consisted of two - three sets of 8 - 12 repetitions at 60% 1 RM for each of the exercises. Weeks 4 - 9 included three - four sets of four - six repetitions at 75% 1RM for each of the exercises. A maximum rest period of 2 minutes was allowed between sets. 1 RM measures were taken pretreatment, bi-weekly (leg press and leg extension only), and posttreatment.

Subjects assigned to the R group ran three times per week. During the first five sessions, the runners jogged (or jog/walked, according to their initial fitness level) for 20 minutes at 70% age-predicted maximum HR. Each of the following sessions included a progressively longer jogging period of 1 - 2 minutes until the subjects were jogging (or jog/walking) 30 minutes at 75 - 80% age-predicted maximum HR by the fourth week. Five heart rates (HR) were taken and recorded by each subject during each exercise session: one at rest, one at cooldown, and one every 6 - 8 minutes during the run.

Those subjects randomly assigned to the RW group ran during the first half of each session. They progressed from an initial 20-minute jog (jog/walk, if necessary) to a 25-minute continuous jog, similar to the R group. Five heart rates were also taken at the same time as the R group. After the run, the remaining 30 minutes were spent lifting weights. The same apparatus and techniques were utilized by the RW group as by the W group. Subjects in the RW group were to perform all of the leg exercises during the available time, and with the time left, they could work out on the other apparatus.

During the first session, subjects in the R and RW groups were instructed on correct techniques for assessing heart rate (HR) via radial or carotid pulse. All subjects were given the chance to practice during rest and exercise. Target HR was estimated by

means of the Karvonen method ({220 - age} - RHR X 100 + RHR) (Karvonen, Kentala, & Musta,1957).

Maximal strength for each subject in the W and RW groups was assessed for each piece of equipment. After an initial warmup, each subject was instructed to lift the given weight one time. Weight was increased until the subject could no longer lift it. The greatest weight lifted for each muscle mass was determined as the criterion score. Three trials were given to determine the maximal repetition, with a short rest between each lift. A percentage of the1 RM then functioned as the daily weight lifted.

STATISTICAL PROCEDURES

Reliability Estimate

To estimate the consistency of the measurement techniques designed to assess the subject's initial pre-test values, a correlation coefficient was computed on the data from the subject's two pre-treatment scores on %BF, plasma TC and HDL-C. The measures were reliable if the r value was 0.70 or greater. The calculated r values were: %BF = 0.97; plasma TC = 0..99; and plasma HDL-C = 0.97.

Validity Estimates

External Validity. The characteristics of the subjects (sedentary females 18 - 30, non-smoking, etc.) limited the results of this study to populations possessing similar characteristics.

Internal Validity. Variance was minimized by the following:1) familiarizing the subjects and test administrators with the testing equipment and protocol prior to the initial endurance and strength tests, and 2) utilizing identical testing procedures during both the pre- and post-training exercise tests.

Research Design

Group differences across time were assessed using a two-factor analysis of variance (ANOVA) with repeated measures. The independent variables were group (weight lifting, aerobic exercise (jogging), and a combination of both) and time (pretest and posttest). The dependent variables were plasma TC, HDL-C, TC/HDL-C ratio, %BF and FFM.

The Tukey post hoc test was applied to groups with significant differences between means. The relationship between plasma concentrations of plasma TC and FFM, plasma HDL-C and FFM, and plasma TC/HDL-C and FFM were evaluated using Pearson product-moment correlation analysis. The level of significance was 0.05. All data were analyzed by computer by means of the Statistical Analysis System (SAS).

Group	Pretest	Treatment	Posttest
Control (C)	O		O
Running (R)	O	X	O
Weight Training (W)	O	X	O
Combination (RW)	O	X	O

Summary ANOVA tables for all the pre- and post-testing measures and weekly measures may be found in Appendix F. One-way (ANOVA) performed on the pre-test values for all dependent measures indicated that the groups were similar except for plasma HDL-C. The Tukey post-hoc test revealed that the C group had a significantly greater plasma HDL-C than the R group ($p<.05$). Percent body fat, FFM, and BW demonstrated no significant changes across time for any group and no significant interaction between treatment groups across time.

Two-way ANOVA indicated that a significant group effect was seen for plasma HDL-C. A follow-up test using the Tukey procedure indicated that the R group was significantly lower than the other 3 groups ($p<.05$) attributable to pre-test differences. No significant differences were found among the remaining 3 groups, nor did ANOVA indicate significant group interaction across time during the treatment period.

The pre- to posttest mean changes for plasma TC are shown in Figure 2. The decrease in plasma TC for all groups combined was significant over time ($p < .05$). Exploring the differences between pretest to posttest for the individual groups via the Tukey post hoc test resulted in no significant differences. It was noted that while the Tukey test demonstrated no significance, a downward trend was seen for the experimental groups. The plasma TC changes from pre- to posttest were 8.2% for the R group, 7.3% for the W group and 4.2% for the RW group. There was no significant group interaction across time during the treatment period.

A significant interaction among groups over time was noted for plasma TC/HDL-C ratio ($p < .05$). Further testing by means of the Tukey procedure revealed significant differences in the response of plasma TC to treatment in the R group. The ratio for the R group decreased 17% (3.5 - 2.9; $p < .05$), while the ratio for the C group increased 13% (2.3 - 2.6; $p<.05$). No significant changes were found for the W, RW, and C groups.

Correlational analyses were performed between the changes in blood lipids and the changes in body composition. The change in plasma TC was positively related to the change in plasma HDL-C ($r = .38$; $p < .05$) and the change in plasma TC/HDL-C ratio ($r = .39$; $p < .05$). The change in plasma HDL-C was positively related to the change in the ratio ($r = .60$; $p < .05$). When FFM changes were related to lipoprotein-cholesterol changes, no significant correlations were found.

Conclusions

Based upon the results of this study, the researcher retains the following null hypotheses:

Ho: There is no significant difference between individuals who weight train for one hour each session three times a week, individuals who run 30 minutes three times a week, and those who combine 25 minutes of running and 30 minutes of weight training in the following measures:

1. Plasma total cholesterol
2. Plasma high-density lipoprotein-cholesterol
3. Plasma total cholesterol/high-density lipoprotein cholesterol ratio
4. Percent body fat
5. Fat-free mass
6. Body weight

APPENDIX B

REQUEST TO HUMAN SUBJECTS COMMITTEE

CERTIFICATE
OF
APPROVAL FOR RESEARCH
INVOLVING HUMAN SUBJECTS

Division of HPER

The Human Subjects Committee of the Division of Health, Physical Education and Recreation has reviewed the research of

Janet L. Walberg, Ph.D. and Stella L. Volpe

entitled Effect of endurance running on strength gains and muscle cell damage in women participating in a weight lifting program.

The members have judged the subjects participating in the related experiment (not to be at risk) as a result of their participation.

(If a risk proposal) Procedures have been adopted to control the risks at acceptably low levels. The potential scientific benefits justify the level of risk to be imposed.

Members of Divisional
Human Subjects Committee

_____	_____
Chairman	Date
_____	_____
	Date
_____	_____
	Date
_____	_____
	Date

REQUEST FOR APPROVAL OF RESEARCH PROPOSAL
IN THE DIVISION OF HPER

Submitted to

Charles Baffi
Chairman, Division Human Subjects Committee and/or
Chairman, Institutional Review Board

by

Janet L. Walberg, Ph.D. and Stella L. Volpe
Principal Investigator

TITLE: Effect of endurance running on strength gains and muscle cell damage in women participating in a weight lifting program.

BACKGROUND/SCIENTIFIC JUSTIFICATION: Weight lifters avoid exercise due to testimonials that it decreases gains in strength and bulk. Theoretically, endurance exercise may increase breakdown of body protein for fuel and/or cause damage to muscle cells (as evidenced by and increase in serum creatine kinase, CK). However, aerobic exercise is beneficial for overall health plus dramatically enhances fat utilization. The latter would aid development of the lean, cut look desired by weight lifters and bodybuilders. There have been few scientific investigations to determine the positive or negative effects of aerobic exercise on the adaptations caused by resistance weight lifting.

PURPOSE(S): This study will assess the differences in strength gains, muscle hypertrophy, body fat and muscle cell damage in females during weight training alone or weight training plus endurance running.

EXPERIMENTAL METHODS & PROCEDURES; Subjects will be assigned to either a control (C), weight training only (WT), or weight training plus running (WT+RN) group. Initial measurements will be made of body weight, body composition (hydrostatic and skinfold), and exercise-induced muscle cell damage (5 ml blood sample assayed for CK immediately before and 24 hours after respective work out). WT subjects will participate in a supervised 3 d/wk, 1 hr/d weight training program (legs and upper body). WT+RN will participate in a weight training for legs only plus 1/2 hr of continuous running at 75% predicted maximal heart rate. All dependent measures will be reassessed at the end of the ten wk period.

STATEMENT DESCRIBING LEVEL OF RISK TO SUBJECTS; Subjects may experience muscle soreness due to exercise programs and temporary discomfort during blood sampling.

PROCEDURES TO MINIMIZE SUBJECT RISK (IF APPLICABLE): All exercise training will be supervised by at least two individuals. Subjects will be instructed on proper technique and injury prevention.

RISK/BENEFIT RATIO (IF RISK PROJECT): Subjects will be expected to benefit physically with an increase in strength and/or endurance exercise, depending on treatment group. Their knowledge of their body composition and of exercise principles will also be expected to increase.. The risks of participation are minimal due to supervision of the training programs and utilization of a trained phlebotomist for the infrequent, small quantity blood sampling.

The principle investigators in this study were Kelsie R. Webb and Stella L. Volpe. Because I (Kelsie R. Webb) joined the study at a later date than Stella, she had already written and submitted the above request. We used the same subjects and many of the same methods and procedures, therefore, the request was adequate without submitting another one.

APPENDIX C

MEDICAL HISTORY QUESTIONAIRE

LAST NAME FIRST NAME INITIAL

DATE OF BIRTH SEX HOME PHONE

ADDRESS CITY, STATE ZIP

SS NUMBER WORK PHONE FAMILY MD

SECTION A

1. When was the last time you had a physical examination?

2. If you are allergic to any medications, foods, or other substances, please name them.

3. If you have been told that you have any chronic or serious illnesses, please list them.

4. Give the following information pertaining to the last three times you have been hospitalized. (Women: do not list normal pregnancies).

HOSPITALIZATION NUMBER 1	HOSPITALIZATION NUMBER 2	HOSPITALIZATION NUMBER 3
TYPE OF OPERATION		
MONTH AND YEAR HOSPITALIZED		
NAME OF HOSPITAL		
CITY AND STATE		

SECTION B

DURING THE PAST TWELVE MONTHS. . .

(PLEASE ANSWER YES OR NO)

1. Has a physicain prescribed any form of medication for you?
2. Has your weight fluctuated more than a few pounds?
3. Did you attempt to bring about this weight change through diet and/or exercise?
4. Have you experienced any faintness, lightgheadedness, or blackouts?
5. Have you occasionally had trouble sleeping?
6. Have you experienced any blurred vision?
7. Have you had any severe headaches/
8. Have you ever experienced chronic morning cough?
9. Have you ever experienced any temporary change in your speech pattern such as slurring or loss of speech?
10. Have you ever felt unusually nervous or anxious for no apparent reason?
11. Have you ever experienced unusual heartbeats such as skipped beats or palpitations?
12. Have you ever experienced periods in which your heart felt as though it were racing for no apparent reason?

AT PRESENT. . .

(PLEASE ANSWER YES OR NO)

1. Do you experience shortness of breath or loss of breath while walking with others your own age?
2. Do you experience sudden tingling, numbenss, or loss of feeling in your arms, hands, legs, feet, or face?
3. Have you ever noticed that your hands and feet sometimes feel cooler than other parts of your body?
4. Do you experience swelling of your feet and ankles?
5. Do you get pains or cramps in your legs?
6. Do you experience any pain or discomfort in your chest?
7. Do you experience any pressure or heaviness in your chest?
8. Have you ever been told that your blood pressure is abnormal?
9. Have you ever been told that your serum cholesterol or triglyceride level was high?
10. Do you have diabetes?
    If yes, how is it controlled?
    ___Dietary means ___Insulin injection
    ___Oral medication ___Uncontrolled
11. How often would you characterize your stress levels as being high?
    ___Occasionally ___Frequently ___Constantly
12. Have you ever been told that you have any of the following illnesses?
    ___Myocardial infarction ___Arteriosclerosis ___Heart disease
    ___Coronary thrombosis ___Rheumatic heart ___Heart attack
    ___Coronary occlusion ___Heart failure ___Heart murmur
    ___Heart block ___Aneurysm ___Angina

SECTION C

Has any member of your immediate family been treated for or suspected to have any of these conditions? Please specify relationship to you (father, mother sister, brother, etc.)

A. Diabetes
B. Heart Disease
C. Stroke
D. High Blood Pressure

SMOKING HABITS

(PLEASE ANSWER YES OR NO)

1. Have you ever smoked cigarettes, cigars, or a pipe?
2. Do you smoke presently?
   Cigarettes _____per day
   Cigars _____per day
   Pipefuls _____per day
3. At what age did you start smoking? _____years
4. If you have quit smoking, when did you quit? _____

DRINKING HABITS

1. During the past month, how many days did you drink alcoholic beverages? _____days
2. During the past month, how many times did you have five or more drinks per occasion? _____times
3. On the average, how many glasses of beer, wine, or highballs do you consume per week?
   Beer_____glasses or cans
   Wine_____glasses
   Highballs_____glasses
   Other_____glasses

EXERCISE HABITS

(PLEASE ANSWER YES OR NO)

1. Do you exercise vigorously on a regular basis?
2. What activities do you engage in on a regular basis?
3. If you walk, run, or jog, what is the average number of miles you cover per week? _____miles
4. How many minutes on the average is each of your workouts? _____minutes.
5. How many workouts per week do you participate in on the average? _____workouts

6. Is your occupation:
   _____Inactive (e.g., desk job)
   _____Light work (e.g., housework, light carpentry)
   _____Heavy work (e.g., heavy carpentry, lifting)
7. Check those activities that you would prefer on a regular exercise program for yourself:
   _____Walking/running/jogging _____Handball/racquetball/squash
   _____Stationary running _____Basketball
   _____Rope jumping _____Swimming
   _____Bicycling _____Tennis
   _____Stationary cycling _____Aerobic dance
   _____Other (specify) _____

DIETARY HABITS

1. What is your current weight?_____lb height?_____in
2. What would you like to weigh?_____lb
3. What is the most you ever weighed as an adult?_____lb
4. What is the least you ever weighed as an adult?_____lb

APPENDIX D

INFORMED CONSENT

HUMAN PERFORMANCE LABORATORY

Division of Health, Physical Education and Recreation
Virginia Polytechnic Institute and State University

INFORMED CONSENT

I, _____, do hereby voluntarily agree and consent to participate in a testing program conducted by the personnel of the Human Performance Laboratory of the Division of Health, Physical Education and Recreation of Virginia Polytechnic Institute and State University.

Title of Study: Effect of endurance running on strength gains and muscle cell damage in women participating in a weight lifting program.

The purposes of this experiment include: This study will assess the differences in strength gains, muscle growth, body and muscle cell damage in females during weight training alone or weight training plus running.

I voluntarily agree to participate in this testing program. It is my understanding that my participation will include: A weight training program or a weight training plus running program. Pretesting will include measurement of body fat (under water weighing), thigh girth (tape measure), and leg strength (Universal equipment), and muscle cell damage (blood sample taken immediately before and 24 hours after one exercise session). Training will take place three times per week for approximately one hour per session. Leg strength gains will be measured biweekly. Body fat, girth, and muscle cell damage measurements will be repeated at the end of the ten week training period.

I understand that participation in this experiment may produce certain discomforts and risks. These discomforts and risks include: Temporary muscle soreness may be a consequence to strength testing and training. Slight discomfort may be experienced during blood sampling.

Certain personal benefits may be expected from participation in this experiment. These include: Subjects may expect to increase their fitness plus acquire increased knowledge of their body fat, and exercise training principles.

Appropriate alternative procedures that might be advantageous to you include:

I understand that any data of personal nature will be held confidential and will be used for research purposes only. I also understand that these data may only be used when not identifiable with me.

I understand that I may abstain from participation in any part of the experiment or withdraw from the experiment should I feel the activities might be injurious to my health. The experimenter may also terminate my participation should he feel the activities might be injurious to my health.

I understand that it is my personal responsibility to advise the researchers of any preexisting medical problem that my affect my participation or of any medical problems that might arise in the course of this experiment and that no medical treatment or compensation is available if injury is suffered as a result of this research. A telephone is available which would be used to call the local hospital for emergency service.

I have read the above statement and have had the opportunity to ask questions. I understand that the researchers will, at any time, answer my inquiries concerning the procedures used in this experiment.

Scientific inquiry is indispensable to the advancement of knowledge. Your participation in this experiment provides the investigator the opportunity to conduct meaningful scientific observations designed to make significant educational contribution.

If you would like to receive the results of this investigation, please indicate this choice by marking in the appropriate space provided below. A copy will then be distributed to you as soon as the results are made available by the investigator. Thank you for making this important contribution.

_____ I request a copy of the results of this study.

Date _____ Time _____am/pm

Participant Signature _____

Witness _____

Project Director _____ Telephone _____

HPER Human Subjects Chairman Dr. Charles Baffi Telephone _____

Dr. Charles Waring, Chairman, International Review Board for Research Involving Human Subjects. Phone 961-5283.

APPENDIX E

PRETESTING INSTRUCTIONS

INSTRUCTIONS: WITHDRAWING BLOOD

Seven milliliters of blood will be drawn on Friday, 4/3/87. This is a small amount, therefore, you will not feel faint, dizzy, or light-headed. However, it is important that you refrain from eating anything within 3 hours of your drawing. A nourishing breakfast or brunch would be advisable before 12:00 noon.

Please avoid food that has been fried, coated in a batter, or highly processed since they will contain a high percentage of fat. Carbohydrates are good choices. Sample menu ideas follow:

*whole-grain bread

*fruit juices

*fresh fruit and vegetables

*low-fat milk, cheese, yogurt

*high-fiber, low-sugar cereal

*low-fat salads

AVOID:

*mayonnaise

*fried foods

*ice cream

*butter or margarine

*red meat

Whatever you eat, please keep it low in fat content and calories. You may get hungry before class. Bear with it. It is imperative that you do not eat, otherwise you may confound the results of the study.

Blood will be drawn in Room 230, Human Performance Lab. Please arrive a few minutes ahead of your scheduled time to ensure smooth flow of so many people. Blood will be drawn from the left arm. Wear clothing which will allow easy access to the elbow area.

Thank you for your cooperation. The phlebotomist is very good at her job. We have done all that is possible to make this as painless as possible.

INSTRUCTIONS FOR HYDROSTATIC WEIGHING

Arrive at the Human Performance Lab, Gym 230, at the assigned time and check in with the technician. Be sure that you have fasted for at least 2 hours prior to the weighing. You will be weighed in you bathing suit, so bring one to change into or wear one under your street clothes.

The following list is what you will be asked to do: 1. First, your residual volume will be measured. This is used in the calculation of your percent fat. It measures the amount of air that is remaining in your lungs after a maximum expiration. You will be asked to blow out as much air as you can into a blue tank (spirometer). This measures your vital capacity, which is the maximum amount of air you can blow out of your lungs after a maximal inspiration. In a prone position on the cot, a noseclip will be fitted and an anesthesia bag with a mouthpiece attached will be placed in the mouth. Always keep a tight seal around the mouthpiece. After about one minute of breathing room air, you will be asked to exhale as much air from your lungs as possible. Signal the technician when you have done this, and the valve will be switched to the bag filled with 100% oxygen. Again, you will breathe normally for about a minute. Once again, you will be asked to breathe out as much air as possible into the bag. Your oxygen and carbon dioxide will be analyzed, and then you will be set to get into the water tank.

2. First, you will be weighed. Next, you will be asked to gently get into the tank. Remove all air bubbles from your suit and hair. Next, you will place small weights around your ankles and on your chest. The test administrator will ask you to slowly lay back and breathe out as you go down. Remain as still as possible for a few seconds. An administrator will tap on the side of the tank to signal you to come up. Be sure you come up sooner if you have to do so. This exercise will be performed 7 - 8 times. (If you would feel more comfortable with a noseclip or holding your nose, tell the technician).

3. After you are dried, change into dry clothes, shorts and a short-sleeved shirt, a test administrator will measure your percent fat with the skin calipers. All measurements will be taken on the right side of your body at the back of the arm, above the hip, and the thigh.

INSTRUCTIONS FOR MEASUREMENT OF 1-RM

When being measured for 1-repetition maximum (1-RM), which is the maximum amount of weight you can lift once on each weight training apparatus, be sure that you arrive at the weight training room in War Memorial Gym at the assigned time. Report to the technician. Wear clothing that you can move easily in (i.e., shorts, sweatpants, t-shirt, etc.). You will be asked to warm up on the specified piece of equipment at very low weights. Following this, you will perform three trials of lifting weights. The third trial will be your 1-RM. A fourth trial will be performed only in 1-RM was not achieved by the third trial.

BODY WEIGHT

Body weight will be measured on a weekly basis. It is preferable that you do not eat at least two hours prior to being weighed. Of great importance, is the fact that we do not want you to change your diet during this study!

APPENDIX F

STATISTICAL ANALYSES

Summary ANOVA - plasma TC

Source	df	SS	MS	F	p
Group	3	1058.7	31.16	.78	.52
Error I	27	1335.5	36057.3	---	---
Time	1	947.7	947.7	4.69	.04
Group*Time	3	126	377.9	.62	.61
Error II	27	201.9	5450.1	---	---

TC = Total cholesterol

Summary ANOVA - plasma HDL-C

Source	df	SS	MS	F	p
Group	3	2042.4	680.8	3.78	.02
Error I	27	4866.6	180.2	----	---
Time	1	36.9	36.9	0.86	.36
Group*Time	3	287.6	95.9	2.22	.11
Error II	27	1166.3	43.2	----	---

HDL-C = High-density lipoprotein-cholesterol

Summary ANOVA - plasma TC/HDL-C Ratio

Source	df	SS	MS	F	p
Group	3	4.64	1.55	1.72	.19
Error I	27	24.31	0.9	----	---
Time	1	0.29	0.29	3.25	.08
Group*Time	3	1.07	0.36	4.03	.02*
Error II	27	2.38	0.09	----	---

TC/HDL-C Ratio = Total cholesterol/High-density lipoprotein-cholesterol

*p<.05

Summary ANOVA - Percent Body Fat

Source	df	SS	MS	F	p
Group	3	57.53	19.18	0.16	.92
Error I	27	3240.24	120.01	----	---
Time	1	1.78	1.78	0.42	.52
Group*Time	3	10.41	3.47	0.82	.50
Error II	27	114.76	4.25	----	---

Summary ANOVA - Fat-free Mass

Source	df	SS	MS	F	p
Group	3	274.79	91.60	2.42	.09
Error I	27	1023.68	37.91	----	---
Time	1	2.95	2.95	1.13	.30
Group*Time	3	0.78	0.26	0.10	.96
Error II	27	70.42	2.61	----	---

Tukey Procedure for time effect - plasma TC

Difference between means	Q
R: pre- to posttest	2.64
W: pre- to posttest	2.48
RW: pre- to posttest	1.36
C: pre- to posttest	0.12

*$p < .05$, $Q_{cv} = 2.91$ for 2 and 27 df
TC = total cholesterol
R = running group; W = weight training group; RW = running/weight training group;
C = control group

Tukey Procedure for time effect - plasma HDL-C

Difference between means	Q
R: pre- to posttest	0.64
W: pre- to posttest	0.04
RW: pre- to posttest	1.02
C: pre- to posttest	4.30*

*$p < .05$, $Q_{cv} = 2.91$ for 2 and 27 df
HDL-C = high-density lipoprotein cholesterol
R = running group; W = weight training group; RW = running/weight training group;
C = control group

Tukey Procedure for time effect - plasma TC/HDL-C ratio

Difference between means	Q
R: pre- to posttest	5.64*
W: pre- to posttest	1.88
RW: pre- to posttest	0.94
C: pre- to posttest	2.82

*$p < .05$, $Q_{cv} = 2.92$ for 2 and 27 df
TC/HDL-C = total cholesterol / high-density lipoprotein-cholesterol
R = running group; W = weight training group; RW = running/weight training group;
C = control group

Tukey Procedure for group effect - plasma TC

Difference between means	Q
W & RW	0.83
W & R	2.91
W & C	3.54
RW & R	2.09
RW & C	2.72
R & C	0.63

*$p < .05$, $Q_{cv} = 3.88$ for 4 and 27 df
TC = total cholesterol
R = running group; W = weight training group; RW = running/weight training group;
C = control group

Tukey Procedure for group effect - plasma HDL-C

Difference between means	Q
C & W	0.17
C & RW	0.34
C & R	5.79*
W & RW	0.17
W & R	5.62*
RW & R	5.45*

*p < .05, Q_{CV} = 3.88 for 4 and 27 df
HDL-C = high-density lipoprotein-cholesterol
R = running group; W = weight training group; RW = running/weight training group;
C = control group

Tukey Procedure for group effect - plasma TC/HDL-C ratio

Difference between means	Q
R & W	4.71*
R & RW	5.28*
R & C	7.45*
W & RW	0.57
W & C	2.73
RW & C	2.17

*p < .05, Q_{CV} = 3.88 for 4 and 27 df
TC/HDL = total cholesterol / high-density lipoprotein-cholesterol
R = running group; W = weight training group; RW = running/weight training group;
C = control group

Tukey Procedure for plasma HDL-C

Difference between pretest means	Q
C & W	1.33
C & RW	1.72
C & R	4.77*
W & RW	0.39
W & R	3.44
RW & R	3.05

*$p < .05$, $Q_{CV} = 3.88$ for 4 and 27 df
HDL-C = high-density lipoprotein-cholesterol
R = running group; W = weight training group; RW = running/weight training group; C = control group

APPENDIX G

RAW DATA TABLES

Table of Pre- and Posttest Plasma Total Cholesterol Measures (mg/dl)

SUBJECT	GROUP	PRETEST	POSTTEST
1	W	185.0	163.5
2	W	155.0	180.0
3	W	188.0	178.5
4	W	173.5	161.5
5	W	152.5	127.0
6	W	182.5	161.0
7	W	173.5	165.0
8	W	171.5	144.0
9	R	164.0	141.0
10	R	151.0	162.0
11	R	159.5	134.0
12	R	142.5	121.0
13	R	193.0	145.0
14	R	107.0	106.0
15	R	144.0	134.0
16	R	205.0	216.5
17	RW	123.0	136.5
18	RW	158.0	142.5
19	RW	147.0	145.0
20	RW	146.0	165.0
21	RW	249.5	226.0
22	RW	153.5	122.0
23	RW	184.5	193.0
24	RW	169.0	163.5
25	RW	152.5	154.0
26	RW	173.0	139.0
27	C	178.0	138.0
28	C	145.0	161.5
29	C	139.5	145.0
30	C	120.0	110.5
31	C	156.0	186.5

Table of Pre- and Posttest Plasma HDL-Cholesterol Measures (mg/dl)

SUBJECT	GROUP	PRETEST	POSTTEST
1	W	75.0	75.0
2	W	55.0	72.5
3	W	54.5	62.0
4	W	54.0	60.0
5	W	70.5	53.0
6	W	51.0	53.0
7	W	76.0	65.0
8	W	69.0	63.5
9	R	49.5	49.0
10	R	50.0	53.5
11	R	27.5	30.0
12	R	38.5	40.5
13	R	50.0	51.5
14	R	68.5	57.5
15	R	48.5	49.0
16	R	60.5	74.0
17	RW	55.0	54.5
18	RW	78.0	79.5
19	RW	49.5	64.0
20	RW	58.5	69.0
21	RW	64.0	50.5
22	RW	63.5	58.0
23	RW	54.5	63.5
24	RW	75.0	76.0
25	RW	50.5	55.5
26	RW	65.5	68.0
27	C	78.0	59.0
28	C	73.0	54.5
29	C	47.0	50.5
30	C	82.0	63.0
31	C	62.5	65.0

Table of Pre- and Posttest Plasma TC/HDL Ratio Measures

SUBJECT	GROUP	PRETEST	POSTTEST
1	W	2.5	2.2
2	W	2.8	2.5
3	W	3.4	2.9
4	W	3.2	2.7
5	W	2.2	2.4
6	W	3.6	3.0
7	W	2.3	2.5
8	W	2.5	2.7
9	R	3.3	2.9
10	R	3.0	3.0
11	R	5.8	4.5
12	R	3.7	3.0
13	R	3.9	2.8
14	R	1.6	1.8
15	R	3.0	2.7
16	R	3.4	2.9
17	RW	2.2	2.5
18	RW	2.0	1.8
19	RW	3.0	2.3
20	RW	2.5	2.4
21	RW	3.9	4.5
22	RW	2.4	2.1
23	RW	3.4	3.0
24	RW	2.3	2.2
25	RW	3.0	2.8
26	RW	2.6	2.0
27	C	2.3	2.3
28	C	2.0	3.0
29	C	3.0	2.9
30	C	1.5	1.8
31	C	2.5	2.9

Table of Pre- and Posttest Hydrostatic Weighing Data (kg)

SUBJECT	GROUP	PRETEST	POSTTEST
1	W	27.6	27.0
2	W	16.6	18.2
3	W	44.6	44.8
4	W	25.9	24.3
5	W	22.7	21.2
6	W	27.1	29.8
7	W	10.2	11.1
8	W	28.6	29.6
9	R	18.7	17.6
10	R	23.9	22.3
11	R	29.4	27.3
12	R	24.1	22.8
13	R	17.6	9.8
14	R	39.3	39.6
15	R	26.1	22.1
16	R	26.5	31.1
17	RW	25.2	25.1
18	RW	25.1	25.1
19	RW	31.6	28.9
20	RW	22.7	17.9
21	RW	26.7	23.6
22	RW	25.0	29.9
23	RW	19.0	20.5
24	RW	17.0	20.4
25	RW	35.2	33.4
26	RW	17.2	22.8
27	C	16.5	13.8
28	C	33.7	32.4
29	C	25.4	24.9
30	C	11.7	14.3
31	C	26.6	26.5

Table of Pre- and Posttest Fat-free Mass Measures (kg)

SUBJECT	GROUP	PRETEST	POSTTEST
1	W	38.7	39.8
2	W	39.2	40.1
3	W	43.8	43.1
4	W	45.9	49.9
5	W	42.5	44.9
6	W	48.8	46.3
7	W	44.9	45.3
8	W	39.8	39.4
9	R	47.2	49.4
10	R	49.1	49.7
11	R	54.4	49.4
12	R	47.8	49.4
13	R	47.8	53.9
14	R	44.0	43.8
15	R	48.0	51.4
16	R	48.5	44.5
17	RW	51.6	50.2
18	RW	42.7	44.2
19	RW	51.3	52.6
20	RW	37.9	39.4
21	RW	44.7	47.4
22	RW	53.3	51.2
23	RW	41.3	40.5
24	RW	50.6	50.5
25	RW	46.7	48.6
26	RW	44.7	43.2
27	C	38.4	39.2
28	C	38.5	39.1
29	C	54.8	54.9
30	C	45.9	45.2
31	C	43.2	43.1

Table of Pre- and Posttest Skinfold Measures (%)

SUBJECT	GROUP	PRETEST	POSTTEST
1	W	20.6	20.6
2	W	14.8	16.0
3	W	31.7	30.1
4	W	21.7	25.7
5	W	19.5	21.7
6	W	26.6	26.6
7	W	17.2	16.0
8	W	25.7	26.6
9	R	19.5	22.7
10	R	26.1	27.5
11	R	31.6	30.4
12	R	26.6	26.6
13	R	21.2	17.2
14	R	35.0	33.2
15	R	28.4	29.3
16	R	24.2	25.7
17	RW	26.6	26.6
18	RW	21.1	19.5
19	RW	25.7	23.7
20	RW	19.5	18.3
21	RW	22.2	22.5
22	RW	27.1	28.4
23	RW	20.6	19.5
24	RW	18.3	19.5
25	RW	29.3	30.1
26	RW	23.2	23.7
27	C	18.9	18.3
28	C	28.5	29.8
29	C	24.7	22.7
30	C	17.8	19.5
31	C	22.5	21.9

Table of Pre- and Posttest Body Weight Measures (kg)

SUBJECT	GROUP	PRETEST	POSTTEST
1	W	53.5	54.5
2	W	47.0	49.0
3	W	79.0	78.0
4	W	62.0	66.0
5	W	55.0	57.0
6	W	67.0	66.0
7	W	50.0	51.0
8	W	56.0	56.0
9	R	58.0	60.0
10	R	64.5	64.0
11	R	68.0	68.0
12	R	63.0	64.0
13	R	58.0	55.0
14	R	72.5	72.5
15	R	65.0	66.0
16	R	66.0	66.0
17	RW	69.0	67.0
18	RW	57.0	59.0
19	RW	75.0	74.0
20	RW	49.0	48.0
21	RW	61.0	62.0
22	RW	71.0	73.0
23	RW	51.0	51.0
24	RW	61.0	63.5
25	RW	72.0	73.0
26	RW	54.0	56.0
27	C	46.0	45.5
28	C	58.0	58.0
29	C	73.5	73.0
30	C	52.0	53.0
31	C	59.0	58.5

Table of Subjects' Height and Age

SUBJECT	GROUP	HEIGHT (cm)	AGE (yrs)
1	W	156.0	21
2	W	164.5	22
3	W	158.5	22
4	W	172.0	22
5	W	173.6	19
6	W	170.5	19
7	W	156.0	22
8	W	166.5	21
9	R	164.5	19
10	R	157.5	21
11	R	172.0	27
12	R	169.0	20
13	R	167.0	20
14	R	150.0	22
15	R	171.0	18
16	R	172.5	22
17	RW	176.0	19
18	RW	164.0	19
19	RW	168.5	20
20	RW	169.5	21
21	RW	157.5	20
22	RW	166.5	21
23	RW	170.5	20
24	RW	154.0	19
25	RW	158.0	20
26	RW	170.0	22
27	C	156.5	21
28	C	163.0	28
29	C	159.5	22
30	C	158.3	22
31	C	163.5	27

Table of Weekly Body Weight Measures (kg)

SUBJECT	GROUP	1	2	3	4	5	6	7	8	9	10	11
1	W	53.5	53.0	55.0	55.0	55.0	55.0	55.0	55.0	55.0	55.0	54.5
2	W	47.0	49.0	49.0	49.0	49.0	49.0	50.0	51.0	51.0	51.0	49.0
3	W	79.0	81.0	81.0	80.0	80.0	81.0	80.0	79.0	78.0	79.0	78.0
4	W	62.0	63.0	64.0	64.0	64.0	63.0	64.0	64.0	65.0	65.0	66.0
5	W	55.0	56.0	56.0	56.0	55.0	55.0	56.0	57.0	57.0	58.0	57.0
6	W	67.0	70.0	68.0	68.0	69.0	69.0	69.0	67.0	68.0	67.0	66.0
7	W	50.0	52.0	51.0	51.0	51.0	53.0	52.0	53.0	53.0	52.0	51.0
8	W	56.0	58.0	58.0	57.0	58.0	58.0	58.0	58.0	57.0	57.0	56.0
9	R	58.0	61.0	61.0	61.0	62.0	60.0	62.0	61.0	61.0	60.0	60.0
10	R	64.5	65.0	64.5	64.5	64.0	64.5	64.0	65.5	64.5	64.5	64.0
11	R	68.0	70.0	70.0	70.0	71.0	70.0	69.5	70.0	70.0	68.5	68.0
12	R	63.0	64.5	64.5	64.0	64.0	65.0	64.0	64.0	64.0	64.5	64.0
13	R	58.0	58.5	59.0	59.0	60.0	59.0	57.5	57.5	57.0	56.0	55.0
14	R	72.5	75.0	75.0	75.0	74.0	73.5	73.5	73.5	73.0	73.0	72.5
15	R	65.0	67.5	67.0	66.0	68.0	67.5	68.0	68.0	68.0	67.0	66.0
16	R	66.0	65.5	65.5	65.5	66.0	65.5	65.5	65.5	65.5	65.5	66.0
17	RW	75.0	77.0	76.0	76.0	77.0	77.0	75.0	74.0	76.0	76.0	74.0
18	RW	49.0	50.0	50.0	50.0	50.0	49.0	50.0	49.0	50.0	49.0	48.0
19	RW	61.0	64.0	64.0	64.0	63.5	63.5	64.0	62.0	63.0	63.0	62.0
20	RW	69.0	70.0	68.0	67.0	68.0	67.0	67.0	67.0	67.0	67.0	67.0
21	RW	71.0	75.0	74.0	74.0	74.0	75.0	74.0	75.0	73.5	74.0	73.0
22	RW	57.0	59.0	59.0	59.0	59.0	61.0	59.0	60.0	59.0	60.0	59.0
23	RW	51.0	53.0	53.0	53.0	53.0	53.0	53.0	52.0	53.0	54.0	51.0
24	RW	61.0	62.0	62.0	63.0	63.5	63.5	64.0	64.0	64.0	65.0	63.5
25	RW	72.0	75.0	74.0	73.0	74.0	74.0	73.5	73.0	74.0	75.0	73.0
26	RW	54.0	55.0	55.0	55.0	56.0	56.0	57.0	57.0	56.0	56.0	56.0

R=running; W=weight training; RW=running/weight training

APPENDIX H

DATA SHEETS

FEMALE SKINFOLD DATA

SKINFOLD DATA--PRE-EXPERIMENTAL

SUBJECT:_____ DATE_____

AGE:_____

TEST ADMINISTRATOR:_____

	TRICEP	SUPRAILIAC	THIGH
TRIAL 1	_____	_____	_____
TRIAL 2	_____	_____	_____
TRIAL 3	_____	_____	_____
TRIAL 4	_____	_____	_____
TRIAL 5	_____	_____	_____

SUM OF 3 SITES (avg. mm):_____ PERCENT FAT: _____

SKINFOLD DATA--POST-EXPERIMENTAL

TEST ADMINISTRATOR:_____

	TRICEP	SUPRAILIAC	THIGH
TRIAL 1	_____	_____	_____
TRIAL 2	_____	_____	_____
TRIAL 3	_____	_____	_____
TRIAL 4	_____	_____	_____
TRIAL 5	_____	_____	_____

SUM OF 3 SITES (avg. mm):_____ PERCENT FAT: _____

HYDROSTATIC WEIGHING DATA

CIRCLE ONE: PRE-EXPERIMENTAL POST-EXPERIMENTAL

SUBJECT:_____ DATE:_____

TEST ADMINISTRATOR:_____

WEIGHT (Ma):_____kg

WATER TEMPERATURE (C):_____

RESIDUAL VOLUME (RV):_____

Value of 10 lb. weight in tank (VWT):_____

TRIALS (#g) (First line is equal to the # of boxes on the graph, the second line is equal to the digital display reading).

1	_____	_____	5	_____	_____
2	_____	_____	6	_____	_____
3	_____	_____	7	_____	_____
4	_____	_____	8	_____	_____

{4.2 kg/VWT = x/#g} = Mh_20 = _____

Dh_20 = (look in chart from Katch and McArdle) Dh_20 = _____

Body Density (BD) = Ma / [{Ma - Mh_20} / Dh_20] - RV

BD = _____/[{_____ - _____} / _____] - _____

BD = _____

% FAT = 4.95 / BD - 4.50

% FAT = _____%

DATA FOR 1-RM LIFTS

SUBJECT:_____ AGE: _____

WEIGHT (LB): _____ (kg):_____

TEST ADMINISTRATORS:_____

DATE:_____

CIRCLE ONE:

PRE-EXPERIMENTAL

WEEK 2

WEEK 4

WEEK 6

WEEK 8

POST-EXPERIMENTAL

	TRIAL 1	TRIAL 2	TRIAL 3
BENCH PRESS	_____	_____	_____
MILITARY PRESS	_____	_____	_____
ARM CURLS	_____	_____	_____
TRICEP PRESS	_____	_____	_____
LAT PULLS	_____	_____	_____
LEG CURLS	_____	_____	_____
LEG PRESS	_____	_____	_____
LEG EXTENSION	_____	_____	_____

CALCULATIONS FOR RESIDUAL VOLUME

SUBJECT:_____ DATE:_____

TEST ADMINISTRATOR:_____

CIRCLE ONE: PRE-EXPERIMENTAL POST-EXPERIMENTAL

VITAL CAPACITY VALUES (VC) (LITERS):

TRIAL 1 _____

TRIAL 2 _____

TRIAL 3 _____

**********TAKE GREATEST VITAL CAPACITY**********

AMOUNT OF OXYGEN TO PUT IN ANESTHESIA BAG:

85% X _____ (VC) = _____ LITERS

REBREATHING VALUES:

OXYGEN:_____% CO_2:_____

RESIDUAL VOLUME (RV) = VO_2 (L) x (b - a) / (c - d)

WHERE:

VO_2 = Volume of O2 in bag

a = 0%

b = 100 - total = % N2 (total = O2 + CO2)

c = 80%

d = .2% + value of b

RV = _____L x (_____ - _____) / (_____ - _____)

RV = _____L

RESULTS OF THE STUDY

Thank you for participating in this study. We hope that the training and educational sessions were beneficial to you. The following are your personal results. Thanks again!

SUBJECT'S NAME_____

PRE-EXPERIMENTAL RESULTS

PERCENT FAT

HYDROSTATIC WEIGHING:_____% CALIPERS_____

GIRTH MEASUREMENTS

RIGHT THIGH (midpoint):_____inches

RIGHT THIGH (3" above knee):_____inches

LEG STRENGTH

LEG PRESS:_____lbs. TC:_____mg/dl

LEG EXTENSION:_____lbs. HDL-C:_____mg/dl

POST-EXPERIMENTAL RESULTS

PERCENT FAT

HYDROSTATIC WEIGHING:_____% CALIPERS_____

GIRTH MEASUREMENTS

RIGHT THIGH (midpoint):_____inches

RIGHT THIGH (3" above knee):_____inches

LEG STRENGTH

LEG PRESS:_____lbs. TC:_____mg/dl

LEG EXTENSION:_____lbs. HDL-C:_____mg/dl

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