EFFECTS OF A COMBINED RESISTANCE TRAINING AND DIET PROGRAM ON LIPOPROTEIN AND LIPID PROFILES: A COMPARISON BETWEEN MALES AND FEMALES

By

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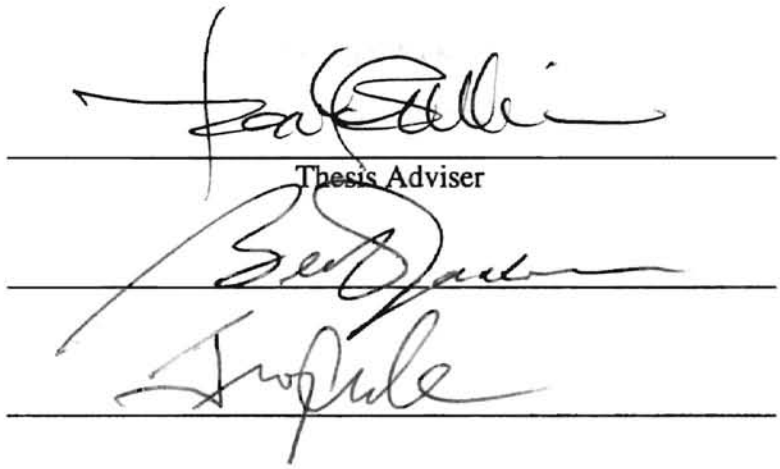

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CHAPTER I

INTRODUCTION

Every year over 100 million dollars are spent by Americans on new and so-called miraculous methods to lose weight, improve health status, and reduce risk for coronary heart disease (CHD) (Howley & Franks, 1986). Those in the weight-loss industry is continuously developing and marketing numerous so-called quick-fix remedies for public purchase. The consumer is bombarded with special foods, diets, physical devices, and programs promoting immediate results with little effort. The need for scientific data and research to support a product or program is becoming increasing crucial for it to win scientific support (Howley & Franks, 1986).

Biometrics One-on-One® program is a six-week personalized weight management program that has been studied and analyzed for its effects on functional capacity (VO2 max), body fat percentage, lean body mass, blood lipid profile and associated parameters. The main goal of this program is focused on preserving or increasing lean muscle tissue during weight loss (Biometrics, 1993). Even though, Biometrics One-on-One® program was not researched and developed to specifically improve lipoprotein-lipid profiles, the participants in the current study were examined for possible changes in total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C), total cholesterol/high-density lipoprotein cholesterol ratio (TC/HDL-C ratio), and triglycerides (TG).

It is of prime importance that guidelines and programs for managing lipids-lipoprotein levels through diet and exercise are available to the American public for reducing coronary risk (Ramsay, Yeo, & Jackson, 1991). Tyroler (1989) stated that for every one percent reduction in serum cholesterol, there is a one to two percent reduction in

the risk for coronary events exists; and each kilogram of maintained weight loss results in: 0.05 mmol/L drop in total cholesterol (TC), 0.02 mmol/L drop in low-density lipoprotein (LDL-C), and 0.009 mmol/L rise in high-density lipoprotein (HDL-C). Other well-documented research has led investigators to agree on the improved effects of lipoprotein-lipid profiles due to exercise and diet (Barnard, 1991; Dengel, Hagberg, Coon, Drinkwater, & Goldberg, 1994).

Justification

The combined effects of diet and aerobic (endurance) exercise have been studied and well-documented in an effort to determine an improvement on lipoprotein-lipid profiles (Wood, Stefanick, Williams, & Haskell, 1993; Nieman, Haig, Fairchild, DeGuia, Dizon, & Register, 1990). However, an area that has not been researched is the combined effects of resistance (strength) training and diet with respect to lipoprotein-lipid profiles in males and females (Kokkinos & Hurley, 1990). It remains to be determined if resistance training, combined with diet, is a beneficial approach for favorably improving lipid levels.

Statement of the Problem

The objectives of this study were to determine if a six-week resistance training program combined with a low-fat, high-fiber diet would significantly improve lipoprotein-lipid profiles and if there were significant differences in these results between the genders.

Hypothesis

1. There will be no significant ($p>0.05$) change in lipoprotein-lipid concentration in subjects after resistance training and diet (time interaction).
2. There will be no significant ($p>0.05$) gender difference in lipoprotein-lipid concentration levels after resistance training and diet (gender interaction).

3. There will be no significant ($p>0.05$) interaction differences of lipoprotein-lipid concentration levels based on time and gender.

Extent of Study

Limitations of the Study

1. The sample only encompassed apparently healthy adult males and females.
2. No attempt was made to randomly select the sample subjects.
3. Information on each participants' dietary compliance of the meal plan was self-reported.
4. No attempt was made to correct for possible changes in blood volume of subjects.
5. No attempt was made to separate the possible effect(s) of resistance exercise and diet.
6. A control group was not used.

Assumptions

1. Subjects completed the Biometrics "One-on-One" Health Questionnaire honestly and accurately.
2. Subjects' information concerning dietary adherence was honest and accurate.
3. Specific data (blood sample) relative to the present study was accurately collected and analyzed.

Definitions of Terms

The following are terms that are used in the study:

American Heart Association Step I Diet (AHA) - Dietary approach to reduce high serum cholesterol consisting of: <30% total fat, <10% saturated fat, ≤ 10% polyunsaturated fat, 10-15% monounsaturated fat, 50-60% carbohydrate, 10-20% protein, & <300 mg/day of cholesterol to achieve and maintain a desirable weight. It is identical to the NCEP Step I Diet to reduce high serum cholesterol (Pate & Burgess, 1993; Dalen, 1991).

AHA Step II Diet - Dietary approach to reduce high serum cholesterol consisting of: <30% total fat, <10 saturated fat, ≤ 10% polyunsaturated fat, 10-15% monounsaturated fat, 50- 60% carbohydrate, 10-20% protein, & <200 mg/day of cholesterol to achieve and maintain a desirable weight. It is identical to the NCEP Step II Diet to reduce high serum cholesterol (Pate & Burgess, 1993; Dalen, 1991).

Circuit Training - A sequence of exercises done one after the other in the same workout with short periods of rest in between exercises (Howley & Franks, 1986).

High-Density Lipoprotein (HDL-C) - A plasma lipid-protein complex containing relatively more protein and less cholesterol and triglycerides. It is considered the "good" type of cholesterol because it serves as a scavenger to remove cholesterol from the periphery and returns it to the liver to be excreted as bile. Desirable levels are ≥ 35 mg/dL (Howley & Franks, 1986; Kenney & Humphrey, 1995).

Hypercholesterolemia - Elevated cholesterol levels found in the blood (Howley & Franks, 1986).

Hyperlipidemia - Elevated levels of fats (triglycerides & cholesterol) found in the blood (Golding, Myers, & Sinning, 1989).

Lipids - Any of the free fatty acid substances in the blood. They are stored in the body and serve as energy reserves. Types of lipids are cholesterol, triglycerides, and fatty acids (Anderson, K.N., Anderson, L.E., & Glanze, 1994).

Lipoproteins - A complex consisting of fat and protein molecules bound together; cholesterol and triglycerides are transported in the bloodstream as part of the lipoprotein structure. There are four major types: HDL-C, LDL-C, VLDL-C, and chylomicrons (Pate & Burgess, 1993).

Low-Density Lipoprotein (LDL-C) - A plasma lipid-protein complex containing relatively more cholesterol and triglycerides than protein. It is considered the "bad" cholesterol because it delivers cholesterol and lipids to body tissues. Desirable levels are <130 mg/dL (Howley & Franks, 1986; Kenney & Humphrey, 1995).

milligrams per deciliter (mg/dL) - A measure of the concentration (amount in weight) of cholesterol (mg) per unit volume (dL) of blood. To convert values from mg/dL to mmol/L; for cholesterol, high-density lipoprotein, and low-density lipoprotein; divide by 38.67.

millimoles per liter (mmol/L) - A measure of the molar concentration (number of millimoles, 1000 millimoles=1 mole) of cholesterol per unit volume (L) of blood. To convert values from mmol/L to mg/dL; for cholesterol, high-density lipoprotein, and low-density lipoptrotein, multiply by 38.67.

megajoule (MJ) - A measure of a unit of energy equilavent to 4.148 MJ/1000 kcal. To convert values from MJ to Kcal; for cholesterol, high-density lipoprotein, and low-density lipoprotein, multiply by 4.148. To convert values from Kcal to MJ, divide by 4.148.

National Cholesterol Education Program Step I Diet (NCEP) - Dietary approach to reduce high serum cholesterol consisting of: <30% total fat, <10% saturated fat, ≤ 10% polyunsaturated fat, 10-15% monounsaturated fat, 50-60% carbohydrate, 10-20% protein, & <300 mg/day of cholesterol to achieve and maintain a desirable weight (Pate & Burgess, 1993).

NCEP Step II Diet - A dietary approach to reduce high serum cholesterol consisting of: <30% total fat, <10 saturated fat, ≤ 10% polyunsaturated fat, 10-15% monounsaturated fat, 50- 60% carbohydrate, 10-20% protein, & <200 mg/day of cholesterol to achieve and maintain a desirable weight (Pate & Burgess, 1993).

Resistance (Strength) Training - Exercise using a wide variety of muscular strength and power-building methods and modes to increase muscular size, strength, and endurance. The performance of various exercises not only using free weights and machines, but the use of resistance provided through hydraulics, elastic bands, springs, and isometrics, involving concentric and eccentric contractions (Pate & Burgess, 1993; Howley & Franks, 1986; Stone & Wilson, 1985).

Total Cholesterol (TC) - Fat-like substances found in animal tissue. Desirable plasma levels are considered to be <200 mg/dL; borderline to high cholesterol is 200-239 mg/dL; high cholesterol level is >240 mg/dL (Howley & Franks, 1986; Kenney & Humphrey, 1995).

TC/HDL-C Ratio - The ratio between the total cholesterol concentration in plasma and the concentration of cholesterol bound to high-density lipoprotein. Desirable levels are ≤5.0. (Howley & Franks, 1986; Pate & Burgess, 1993).

Triglycerides (TG) - A type of lipid (fat) found in the body, otherwise referred to a devired fat, representing more than 90% of the fat stored in the body. They are composed of three fatty acids and a glycerol molecule, and is important of the storage and transport of fatty acids. Desirable levels are <200 mg/dL (Pate & Burgess, 1993; Kenney & Humphrey, 1995; Howley & Franks, 1986).

CHAPTER II

REVIEW OF THE LITERATURE

Epidemiological data show that elevated blood cholesterol is a major risk factor in development of CHD and more than 50% of the adult population (>20 yrs.) in the United States have blood cholesterol exceeding suggested-desirable levels (<200 mg/dL) (Bell, Hectorn, Reynolds, & Hunninghake, 1990).

Data from the third National Health and Nutrition Examination Survey (NHANES III) in 1992 indicated that 29% of all adult men and women (>20 yrs.) combined, 27% of all adult women (>20 yrs.), 32% of all adult men (>20 yrs.), and 50% of all men and women, aged 55 to 74 years, were candidates for dietary intervention based on elevated blood cholesterol levels defined as TC ≥6.21 mmol/L, HDL-C <0.90 mmol/L, and LDL-C >4.14 mmol/L (Kris-Etherton & Krummel, 1993).

It has been well-documented, in controlled-studies, that diets low in fat, high in carbohydrate, and fiber are associated with significant changes in TC, LDL-C, and HDL-C levels in males and females (Dengel et al., 1994; Cobb, Greenspan, Timmons, & Teitelbaum, 1993). In addition, Dengel et al. (1994) and Cobb et al. (1993) agree that dietary modification can also reduce this risk factor for coronary complications. Another investigator, Synder (1990) agrees that diet therapy should always be the first consideration in the attempt to lower serum cholesterol levels.

Research on resistance training, with or without diet manipulation is limited. A review of literature revealed that several studies regarding specific lipid changes have provided conflicting information (Kokkinos & Hurley, 1990; Stone, Fleck, Triplett, & Kraemer, 1991; Verrill, Shoup, McElveen, Witt, & Bergey, 1992). However, the following literature review contains pertinent research on the topic of concern.

Changes in Total Cholesterol

Diet Only

Anderson (1993) compared the effects of the American Heart Association (AHA) Step I Diet (30% fat, 55% carbohydrate, 15% protein, 300 mg dietary cholesterol/day, and polyunsaturated fat to saturated fat ratio 1:1) for eight weeks on male (N=99) and female (N=64) participants. All subjects (30-70 yrs.) had serum cholesterol levels between 200-300 mg/dL, serum triglycerides levels <300 mg/dL, and ≤130% of desirable weight. Complete blood lipid profiles were obtained bi-weekly after a 12-hour fast. Dietary intake was monitored at four-week intervals using a three-day food record. Serum TC decreased from 263±4 to 238±4 mg/dL ($p \leq 0.0001$) for women, and from 248±3 to 230±3 mg/dL ($p \leq 0.0001$) for men, a 9.1% and 7.3% decrease for women and men, respectively.

Long-term study also done by Anderson (1993) compared a control group with two separate groups using different types of cholesterol-lowering diets. Male (N=87) and female (N=59) subjects (30-50 yrs.), combined, had a serum cholesterol of 200-300 mg/dL, body weight 80-120% of desirable weight, and were in good general health. The AHA Phase II-type diet (AHA) consisted of 25% fat, 55% carbohydrate, 20% protein, <200 mg dietary cholesterol/day, polyunsaturated fat to saturated fat ratio 1:4. The AHA Phase II-type diet with additional intakes of dietary fiber (35-40 g) (AHA-F) also consisted of 25% fat, 55% carbohydrate, 20% protein, <200 mg dietary cholesterol/day, and polyunsaturated fat to saturated fat ratio 1:4. Diet and blood samples were collected at zero and 12 months. The AHA-F group had a significantly ($p \leq 0.004$) greater reduction in TC than did the control group. There were also significant ($p \leq 0.05$) differences in TC reduction between the AHA-F group and the AHA group. TC concentrations were not significantly ($p > 0.05$) different between the AHA group and control group. Average reductions in TC for the control, AHA group and AHA-F group were: -16.3 mg/dL (-7%), -22.8 mg/dL (-10%), and -30.6 mg/dL (-13%); respectively.

Long term (1 year) effects of dietary fat intake on lipoprotein metabolism were investigated in healthy women (N=72) (>18 yrs.) who were at risk for breast cancer. Dietary intervention group received a 15% fat diet (LFD) and the control group received a nonintervention usual diet (NIUD). All lipoprotein data were obtained at baseline and at three month intervals. Two blood samples were taken after a 14-hour fast. The LFD group experienced a significant ($p \leq 0.05$) reduction in TC at three (4.83±0.17 mmol/L) and six (4.80±0.16 mmol/L) months compared to baseline values (5.21±0.18 mmol/L). No further decreases were demonstrated at nine and 12 months. The NIUD group had no significant change in TC throughout the dietary period (Kasim et al., 1993).

Sandstrom, Marckmann, and Bindslev (1992) investigated the effects of an eight-month low-fat, high-fiber diet on young, (20-30 yrs.) healthy, Danish males (N=18) and females (N=12). All subjects were consuming a typical Danish diet with a dietary fat intake of approximately 35%. Tobacco consumption was less than ten cigarettes per day prior to the study. The dietary intervention group (DI) received a diet consisting of <30% of calories from fat, <10% of calories from sugar, and a minimum fiber content of 3g/MJ (12.4g/1000 Kcal), while the control group consumed their normal dietary intake. Blood samples were taken at baseline, one, three, five, and eight months during the DI period. Significant ($p \leq 0.001$) decreases in TC were experienced at the end of the eight-month period for both sexes in the DI group. TC levels at baseline and after eight months were 4.61±0.59 and 3.89±0.61 mmol/L for the females, and 4.21±0.61 and 3.80±0.62 mmol/L for the males. No significant ($p > 0.001$) changes in TC were observed for the CG throughout the study.

Ginsberg and associates (1990) investigated the effects of an average American diet (AAD) versus an AHA step I diet, and a monounsaturated fatty acid-enriched AHA step I diet on TC on a group of healthy males (N=36) (22-32 yrs.). Each subject consumed an AAD (38% fat; 18% saturated fatty acids, 10% monounsaturated fatty acids, 10% polyunsaturated fatty acids; and 500 mg of cholesterol/day) for 10 weeks. After

randomization, one third of the subjects continued to consume the AAD, one third consumed the AHA Step I diet, and one third switched to a monounsaturated fatty acid-enriched modification of the Step I diet (Mono diet) (38% fat; 10% saturated fatty acid, 18% monounsaturated fatty acid, 10% polyunsaturated fatty acid; 250 mg of cholesterol/day) for an additional 10 weeks. Twelve-hour fasting blood samples were taken at weeks two, four, six, and nine while all subjects consumed the AAD, and were also taken at weeks two, four, seven, and 10 during the randomized-diet period. Plasma cholesterol levels remained stable during the AAD period. During the randomized-diet period, the reduction in plasma TC levels in both the Step I diet (-14.3±10.4 mg/dL) and the Mono diet (-17.8±13.9 mg/dL) were statistically significant ($p \leq 0.025$), as compared with the reduction of TC in the AAD (-1.9±13.9 mg/dL). These reductions were -8%, -10.4%, and -1.1%, respectively. There were no significant ($p > 0.025$) differences in TC reductions of the Mono diet, as compared to the Step I diet.

Boyd and associates (1990) observed changes in serum cholesterol in women (N=206) (≥30 yrs.) with mammographic dysplasia (examined by mammography within three months of entry into the study and found to have ≥50% of the breast volume occupied by the radiologic changes of dysplasia) who participated for 12 months in a randomized, controlled trial of a low-fat, high-carbohydrate diet. Total fat intake was reduced from an average of 37% to 21%, and carbohydrate was increased from 44% to 51% of calories. Blood samples were obtained at zero, four, eight, and 12 months for both the intervention (IG) and control groups (CG). Serum cholesterol in the IG fell -8% at four months (4.45±0.85 mmol/L), -6% at eight months (4.55±0.87), and -4% at 12 months (4.63±0.81), as compared to baseline values. The mean value of serum cholesterol in the IG was significantly lower at four ($p \leq 0.006$), eight ($p \leq 0.06$), and 12 ($p = 0.07$) months, as compared to the control group. Serum cholesterol was unchanged from baseline to 12 months in the control group.

Anderson and associates (1990) studied initial TC levels in response to a cholesterol-lowering diet. A group of males (N=99) and females (N=64) were investigated to determined the effects of a 6-week AHA Phase I Diet on TC levels. All subjects had serum cholesterol levels >200 mg/dL prior to the study. The group was subdivided based on sex and initial TC levels. Tertiles for women were as follows: 418-273 mg/dL (high), 272-248 mg/dL (intermediate), 247-207 mg/dL (low); and for men were 348-256 mg/dL (high), 255-237 mg/dL (intermediate), 236-201 mg/dL (low), respectively. Females in the high and intermediate tertiles experienced significant ($p \leq 0.001$) percent reductions of -12.1% and -11.3%, respectively. Males also experienced significant percent reductions in the high (-10.2%, $p \leq 0.001$), intermediate (-8.2%, $p \leq 0.001$), and low (-3.5%, $p \leq 0.022$) tertiles.

In summary, dieatry modification is of prime importance and undoubtedly can lower TC levels, as shown by the previous investigations. All the previously mentioned dietary intervention studies demonstrated that a modified diet can beneficially affect TC levels and modify risks of cardiovascular disease.

Resistance Training Only

Manning et al. (1991) studied healthy sedentary obese women (N=16) to determine if 12 weeks of strength training would result in blood lipid improvements, as well as, improvements in strength gains. The subjects exercised three days per week for approximately 60 minutes. Each subject completed eight types of exercises per training session. Week one involved two sets of six to eight repetitions at 60-70% of 1 repetitons maximum (RM) for each exercise. During the next 11 weeks, subjects performed three sets per exercise. Sets one and two invovled eight repetitions at 60-70% of 1 RM, and set three involved the greatest weight possible, so that failure occurred between six to eight repetitions. The fasting concentration of TC taken 72 to 96 hours after the last training

session showed no significant ($p>0.05$) change in TC over the duration of the training period.

Kokkinos and colleagues (1988) examined the effects of the degree of resistance and the number of repetitions as important factors in establishing the effects of resistive training on lipoprotein-lipid profiles. A ten week low- (four to six repetitions maximum) versus high- (14 to 16 repetitions maximum) repetition resistive training program was used to study 37 healthy untrained males. Subjects were encouraged not to change dietary intakes throughout the training period. All fasting blood measurements were taken at 24 and 72 hours after last exercise bout. The concentrations of TC, in the low-repetition group, was 150±7 mg/dL at baseline and 144±7 mg/dL after the ten weeks. The high-repetition group showed TC concentration of 148±6 mg/dL and 158±6 mg/dL, pre-and post-test, respectively. At the end of the training period, no significant ($p>0.05$) TC changes were demonstrated with either of the two training intensities.

A more recent study done by Kokkinos and associates (1991) determined the effects of 20 weeks of strength training on lipoprotein-lipid profiles in 16 untrained males. All subjects had abnormal lipoprotein-lipid profiles and at least two other risk factors for CHD. The investigators were trying to determine if subjects at risk for CHD could show improvements in their lipid profiles with strength training. Subjects were periodically reminded not to change their dietary habits throughout training. Subjects trained three times per week using 12 different exercises. The training program consisted of two sets of 12-15 RM per set. Once the training period was completed, two blood samples were taken at 20±5 hours and 46±7 hours, respectively. No significant ($p>0.05$) changes were shown in TC for either the training or control group.

Research was also developed to specifically evaluate the effects of heavy-resistance training (three days/week, 1hour/session, 15 exercises, 12 repetitions maximum, three to seven sets/exercise) on strength and blood lipid levels in men (N=8) with Type I Diabetes Mellitus. Blood lipid profiles were tested, at least 24 hours after the last training session,

three times during the program for each of the two groups. Group A participated in the training program as follows: initial blood draw prior to training, ten-week training period, second blood draw was administered, six-week rest period (no activity), final blood draw was administered. Group B participated in the training program as follows: initial blood draw prior to training, six-week rest period (no activity), second blood draw was administered, ten-week training period, final blood draw was administered. Group A and Group B, combined, experienced significant ($p=0.015$) reductions in the TC mean values after 10-week resistance training period. Pre- and post-training values were 194±41 and 178±34 mg/dL. There were no measurements of TC mean values after the six-week rest period (Durak, Jovanovic-Peterson, & Peterson, 1990).

Smutok and colleagues (1993) compared the effects of strength training (ST), aerobic training (AT), and inactive controls (IC) for CHD risk factor intervention for 20 weeks. All subjects were untrained males (N=37) possessing two of the three following risk factors: abnormal lipid profile, abnormal blood glucose level (either from impaired glucose tolerance or type II diabetes), or hypertension. Twelve to 14-hour fasting blood samples were obtained before training for all subjects. After training, blood samples were obtained once for control subjects, and at 20±5 hours and 46±7 hours after the last exercise session in the training groups. The ST program exercised 3 days/week, consisting of two sets using the maximum amount of weight that could be lifted 12-15 times, with 11 different exercises , and modified sit-ups. The AT program walked/jogged on the treadmill 30 minutes/day, 3 days/week. Exercise intensity was 50-60% of maximum HRR during weeks 1-2, and increased to 75-80% during weeks 3-20. The IC program consisted of no regular exercise during the 20-week period. There were no significant ($p>0.05$) differences in the TC levels after training with either of the training groups. There were also no significant ($p>0.05$) differences found in the IC group after the 20-week period.

Boys (N=32), ages 6 to 11, were studied (Weltman, Janney, Rians, Strand, & Katch, 1988) over a 14-week period to determine the effects of a strength training program

on TC levels and were compared to a control group. Each strength training session was conducted for appproximately 45 minutes, three days per week. Participants performed a circuit weight training method using eight hydraulic-resistance machines, and two additional stations were also included. One circuit consisted of continuous exercise for 30 seconds with a 30-second rest between stations at each of the ten stations. Fasting blood samples were taken prior to training and at least 24 to 48 hours after the final exercise bout. TC levels decreased significantly ($p \leq 0.05$) in the strength training group as compared to the control group. Pre- and post-training means were 197.2±39.3 mg/dL and 166.3±26 mg/dL, a -15.7% change in the strength training group; and 159.9±21.5 mg/dL and 154.8±23.7 mg/dL, a -3.2% change in the control group.

Blessing and colleagues (1987) evaluated the effect of 12 weeks of jogging or weight training on TC blood lipid changes in healthy, sedentary males (N=33). The joggers and weight trainers exercised 3 days/week for approximately 45 minutes. Weight trainers performed three sets of ten repetitions using 12 exercises during weeks one through six, and performed three sets of five repetitions, using the same 12 exercises, during weeks seven through 12. All exercises were performed with maximum weight possible. Twelve-hour fasting blood samples were taken in the morning on two alternate days at zero, six, and 12 weeks. No significant ($p > 0.05$) differences were experienced in either the jogging (231±37, 238±35, & 232±36 mg/dL), weight training (229±40, 222±47, & 227±44 mg/dL), or control groups (208±28, 226±38, & 227±40 mg/dL) at baseline, six or 12 weeks. On the other hand, both experimental groups demonstrated significantly ($p \leq 0.05$) different patterns of change in TC as compared to the control group. TC levels remained fairly stable in the jogging group (231±37, 238±35, & 232±36 mg/dL) and weight training group (229±40, 222±47, & 227±44 mg/dL); whereas, TC levels increased in the control group (208±28, 226±38, & 227±40 mg/dL).

Stone and colleagues (1982) divided healthy men (N=31) into three groups consisting of a resistive training group (experimental), a sedentary control group, and an

active control group (runners who were already engaged in a jogging program). Subjects trained three days per week for 12 weeks. The resistive training group performed three sets of 10 repetitions using seven exercises per day. Resistance progressed from light (60-65% of heavy set) to heavy (maximum amount of weight used to complete the required number of sets and repetitions; 10 RM). Twelve-hour fasting blood samples were collected in the morning at zero, six, and 12 weeks to determine TC levels. No significant ($p>0.01$) changes were experienced in any group at six or 12 weeks.

Another study during the same year (Farrell et al., 1982) also compared the TC levels in men who were candidates for the 1980 US Olympic Speed Skating Team (N=11) (17-27 yrs.) (high level of both aerobic and anaerobic training) to a group of well-trained weightlifters (N=11) (20-32 yrs.) (heavy resistance, short-duration exercises), and a sedentary group (N=11) (19-25 yrs.) (no training for six months). Twelve to 16-hour fasting blood samples revealed no significant ($p>0.05$) differences in TC levels for either of the three groups.

The results of the previous resistance trainining studies have revealed inconsistent effects on TC levels due to various extraneous variables. Only one study (Weltman et al., 1988) reported significant decreases in TC levels after the strength training program; on the other hand, several other studies (Manning et al., 1991; Kokkinos et al., 1991; Blessing et al., 1987; Stone et al., 1982) found conflicting results. Manning et al. (1992), Kokkinos et al. (1991), and Smutok et al. (1993) found no changes in TC levels following the training program; whereas, several other studies (Kokkinos et al., 1988; Durak et al., 1990; Blessing et al., 1987; Stone et al., 1982) demonstrated insignificant decreases in TC.

Changes in High-density Lipoproteins

Diet Only

Researchers (Bell, Hectorn, Reynolds, & Hunninghake, 1990) evaluated the effectiveness in lowering serum cholesterol in male (N=58) (24-69 yrs.) subjects using two

different soluble fibers (pectin-enriched cereal and psyllium-enriched cereal) when used as part of a prudent diet. All subjects had hypercholesterolemia, triglyceride levels were ≤3.39 mmol/L, and bodyweight was ≤30% of ideal. All participants consumed an AHA Step I Diet during the entire 12 weeks. After the initial six weeks, participants were randomly assigned to groups incorporating either: corn flakes (control group), pectin-enriched, or psyllium-enriched cereal into the diet for an additional six weeks. During the initial diet phase, all subjects demonstrated a significant ($p \leq 0.005$) reduction in HDL-C levels. Decreases are as follows: corn-flake enriched, -5.7%; pectin-enriched, -5.6%; psyllium-enriched, -4.0%. Only during the cereal-plus diet phase did the psyllium-enriched group demonstrate an insignificant, yet additional reduction (-1.6%) in HDL-C levels. The corn-flake enriched and pectin-enriched groups experienced a non-significant +1.6% and +2.5% increase, respectively. No comparisons were measured between the three groups.

Another study also using a supplement in conjunction with a low-fat diet was done on a large group of moderately hypercholesterolemic, non-obese males and females (N=146) (30-50 yrs.). All subjects had serum cholesterol values between 200-300 mg/dL, serum triglyceride values <500 mg/dL, and between 80-120% of ideal body weight. All were free from hypertension, diabetes, and any type of life-shortening diseases. Participants were randomly assigned to either a control group, AHA Phase II Diet group (HF) with ~15g of dietary fiber, or a AHA Phase II Diet with ~50g of dietary fiber for 12 months. Subjects did not experience a significant ($p > 0.05$) increase in HDL-C levels throughout diet period. The control and AHA diet group had a +0.01 mmol/L change; whereas, the HF group had a -0.04 mmol/L decrease in HDL-C levels (Anderson, Garrity, Wood, Whitis, Smith, & Oeltgen, 1992).

Wood et al. (1988) studied the influence of weight loss on the levels of plasma lipid and lipoprotein in overweight sedentary men (30-59 yrs.). All subjects were nonsmokers, had a resting blood pressure below 160-100 mm Hg, were 120-160% of ideal bodyweight,

had a TC level below 8.28 mmol/L, and TG level below 5.65 mmol/L. Subjects were randomly assigned to a control group (N=52) (CG), or a diet group (N=51) (DG) for one year. Twelve to 16-hour fasting blood samples were taken at seven and 12 months. Results after seven months showed a significant ($P \leq 0.01$) increase in HDL-C levels for the DG (+0.06±0.14 mmol/L) relative to the CG (0.00±0.10). Further significant ($p \leq 0.001$) increases in HDL-C levels at one year were +0.12±0.16 mmol/L for the DG; whereas, the HDL-C levels of the CG slightly decreased (-0.02±0.11).

Williams, Stefanick, Vranizan, and Wood (1994) studied the effects of caloric restriction on HDL cholesterol levels for men (N=84) (30-59 yrs.) over a one-year period as compared to a control group (CG). All participants were nonsmokers, normotensive, normolipidemic, and 20-60% over metropolitan ideal weight. Two separate fasting blood samples were obtained at baseline and at one year. Both the caloric restriction group (CRG) and CG were further divided into three sub-groups, and are as follows: low HDL cholesterol ($\leq$ 37 mg/dL), intermediate HDL cholesterol (38-47 mg/dL), and normal to high HDL cholesterol ($\geq$ 48 mg/dL). All subjects in the CRG (low HDL-C, intermediate HDL-C, and normal to high HDL-C, included) experienced significant ($p \leq 0.05$) HDL-C increases as compared to the CG. The low CRG (+6.6±8.3 mg/dL) experienced significant ($p \leq 0.05$) increases in HDL-C levels as compared to the low CG (+0.6±3.8 mg/dL). The intermediate, and normal to high CRG (+3.6±5.8 mg/dL; +4.8±5.5 mg/dL) also experienced significant ($p \leq 0.05$) increases in HDL-C levels as compared to the intermediate, and normal to high CG (-1.7 ±4.7 mg/dL; +0.1±3.9 mg/dL), respectively.

Dattilo and Kris-Etherton (1992) analyzed the results of 70 studies using the meta-analysis method. Researchers examined the effects of weight reduction by dieting on HDL-C levels. For every Kg decrease in body weight, a +0.009 mmol/L increase ($p \leq 0.01$) occurred in HDL-C levels for subjects at a stabilized, reduced weight, but a -0.007 mmol/L decrease ($p \leq 0.05$) for subjects actively losing weight. Results also indicated that for each week the diet/study period was increased in duration, HDL-C

increased by +0.004 mmol/L ($p \leq 0.01$). Studies lasting close to one year (mostly stabilized weight-loss periods) were associated with a +0.18 mmol/L increase in HDL-C. Studies lasting ≤ six weeks (mostly active-weight loss periods) were associated with a -0.09 mmol/L decrease in HDL-C.

Several of the previous dietary intervention studies (Williams et al., 1994; Wood et al., 1988;) concluded that one year dietary intervention produces favorable changes in HDL-C levels. On the other hand, Bell et al. (1990) demonstrated a significant reduction in HDL-C levels following a 12-week study. Anderson et al. (1992) also found that subjects experienced significant changes in HDL-C after the experimental period.

Resistance Training Only

Fripp and Hodgson (1987) examined 14 healthy male adolescents before and after a nine week resistive exercise program, and compared the changes in plasma lipid and lipoprotein levels with those in 14 non-exercisers. The subjects exercised three days per week for approximately 60 to 80 minutes per session. Each session consisted of 11 different exercises (stations). Ten minutes were spent at each station with two to three minutes between each station. Training consisted of as many repetitions that could be performed in the allotted time. No attempt was made to control dietary consumption. Fasting blood samples were taken 12 hours after last exercise session in both groups. HDL-C values significantly ($p \leq 0.01$) increased from, 35±2 to 45±8 mg/dL, in the training group; whereas, the control group experienced an insignificant ($p > 0.05$) decrease in their HDL-C from 40±9 to 36±10 mg/dL.

Research (Goldberg, Elliot, Schutz, & Kloster, 1984) on a group of healthy sedentary subjects who participated in a 16 week program of progressive resistance weight training demonstrated improvements in HDL-C for the males (N=6) and females (N=8). Each training session was 45 to 60 minutes in duration three times per week. Training consisted of 3 sets of repetitions for 8 training exercises. No less than three and no more

than eight repetitions were allowed for each training exercise. A maximum rest period of two minutes was allowed between sets. There were no dietary changes reported among the participants. Fasting blood samples were taken 36 hours after the last training session. Women had an insignificant ($p>0.05$) increase from 77.4 to 81.1 mg/dL, a +4.8% change; while men demonstrated a significant ($p\leq0.052$) increase from 50.6 to 58.6 mg/dL, a +15.8% increase in HDL-C levels.

A study on healthy trained males (N=10) tried to determine the acute effects of a 90-minute high volume (HV) (8-12 repetition maximum (RM), 70-80% 1RM, 60 second rest interval between sets) versus a 90-minute low volume (LV) (1-5 RM, 87.5-100% 1RM, 3 minute rest interval between sets) resistance exercise session on alterations in lipid and lipoprotein concentrations. Fasting blood samples were drawn immediately before and after exercise, 24 hours, 48 hours, and 72 hours post-exercise. By 24 hour post-exercise, the HV treatment resulted in a 11 % HDL-C increase which was significantly ($p\leq0.01$) greater than the LV treatment and controls, but returned to baseline by 48 and 72 hours (Wallace, Moffatt, Haymes, & Green, 1991). Pronk (1993) also concluded that a strength training program may have the potential to elicit short term improvements in HDL-C levels provided the volume of the exercise bout is sufficient.

Kimura, Kubota, and Yamazaki (1989) compared different types of training modes (bicycling training, circuit weight training, swimming, & control group) and their effects on lipoprotein-lipid levels after ten weeks of training in a group of 45 males. Exercise training was conducted three days per week for approximately 30 minutes per training session. Results revealed that the circuit weight training group had significantly ($p\leq0.05$) higher HDL-C levels than the swimming and control groups; whereas, the difference in HDL-C values between the circuit weight training group and bicycling group were insignificant ($p>0.05$).

Morgan et al. (1986) assessed the HDL-C levels of female weight trainers (N=9) (isotonic and isokinetic training methods, moderate to high resistance, three days/week, 60

to 224 minutes), endurance runners (N=9) (25 to 59 miles/week, three days/week, 30 to 89 minutes), and sedentary controls (N=9) (no consistent exercise training within the year). Twelve to 16 hour fasting blood samples were taken in the morning after last training bout for each subject. Mean HDL-C levels (mg/100 ml) and percent HDL-C were significantly ($p \leq 0.05$) higher in the runners (71.9±11.9 mg/dL; 39.4±5.0%) than in the weight trainers (56.2±5.1 mg/dL; 31.0±4.3%) and controls (57.8±13.5 mg/dL; 31.5±9.0%).

Researchers (Fang, Sherman, Grouse, & Tolson, 1998) measured the HDL-C concentration in a group endurance-trained males (N=19) (ET) (running ~35 miles/week, or intensive cycling or swimming at least six months prior to the study) as compared to a group of strength-trained males (N=19) (ST) (three days/week for six or more months prior to the study) and sedentary males (N=19) (SED) (no regular physical exercise). Two separate 12-hour fasting blood samples were obtained 48 hours after the last bout of exercise. The average HDL-C concentration of the ET group was significantly ($p \leq 0.05$) higher than the SED group. On the other hand, the average HDL-C concentration of the ST group (1.17±0.15 mmol/L) was 13% lower and four percent higher than the respective averages of the ET (1.34±0.20), and SED (1.13±0.10) groups; however, these differences were not statistically significant ($p > 0.05$).

Yki-Jarvinen, Koivisto, Taskinen, & Nikkila (1984) studied and compared HDL-C levels among a group of bodybuilders (N=9) (BB) (engaged in bodybuilding for more than two years at least four to five times per week), weight-matched controls (N=8) (WMC) (matched for relative body weight and maximal aerobic power), and normal-weight controls (N=7) (NWC) (matched for height and maximal aerobic power). The subjects were studied on three separate days, and blood samples were taken after a 12-hour overnight fast. No significant ($p > 0.05$) differences were found in HDL-C levels between the three groups.

Hurley et al. (1984) investigated the HDL-C levels in a group of powerlifters (N=8) (PL) (heavy-resistance, low-repetitions), bodybuilders (N=8) (BB) (moderate-resistance,

high-repetitions), endurance runners (N=8) (ER) (40-55 km/week), and controls (N=8) (C). Twelve- to 14-hour fasting blood samples were taken in the morning from all subjects to determine HDL-C concentrations. All subjects were consuming a regular diet and no androgen use was reported for at least 10 weeks prior to the study. Plasma HDL-C levels were significantly ($p \leq 0.01$) lower in the PL (38±2 mg/dL) than in BB (55±2 mg/dL), ER (47±2 mg/dL), and C (46±1 mg/dL). No further comparisons were measured between the other groups.

Another study done by Hurley and colleagues (1987) investigated the effects of powerlifting strength training on certain CAD risk factors in middle-aged men. Five powerlifters (heavy resistance, low repetition, three to eight RM, four to six days/week, for 11 to 41 years) were compared to 10 distance runners (48±15 miles /week, for three to 15 years), and nine sedentary controls (no regular exercise for several years). All subjects were consuming regular diets and weight stable during the study. Blood samples were taken after a 12-hour overnight fast. HDL-C levels in powerlifters were 37% lower than those of runners, and 29% lower than those of sedentary men. The powerlifters had a significantly ($p \leq 0.01$) lower HDL-C level (34±4 mg/dL) than the runners (54±8 mg/dL) and the sedentary controls (48±12 mg/dL). No comparison was measured between the runners and the controls.

In summary of the previous resistance training study using young men and women who were weight-trained for 16 weeks, Goldberg et al. (1984) found significant increases in HDL-C levels. Similar beneficial alterations in HDL-C have been reported by Fripp and Hodgson (1987) in male adolescents after nine weeks of circuit weight training. However, some of the previous resistance training studies (Hurley et al., 1987; Wallace et al., 1991) have not always shown significant alterations in HDL-C concentration. Wallace et al. (1991) reported little no change in HDL-C concentration; whereas, Hurley et al. (1987) experienced a significant decrease in HDL-C.

Changes in Low-Density Lipoproteins

Diet Only

Sacks, Handysides, Marais, Rosner, and Kass (1986) did a study to determine the effects of a three-month, low-fat, semi-vegetarian diet (LF-SV) and nine to 12 month post-study self-selected diet (PS-SS) on LDL-C levels for normolipidemic, nonvegetarian males and females (N=20) (14-61 yrs.). There was a significant ($p \leq 0.001$) -18% decrease in LDL-C levels after the LF-SV diet, as compared to baseline diet. Pre and post LDL-C values were 125±25 and 103±20 mg/dL, respectively. Mean LDL-C levels significantly ($p \leq 0.001$) increased (+17%) during the PS-SS diet (120±25 mg/dL), as compared to the LF-SV diet (103±20 mg/dL). The LDL-C levels for the PS-SS diet remained -5% ($p=0.13$) lower than during the baseline diet.

Denke, Scott, and Grundy (1994) studied 50 men (31-70 yrs.) with moderate hypercholesterolemia defined as LDL-C levels between 160-220 mg/dL, and fasting TG levels <250 mg/dL. The study was designed to determine whether dietary therapy will produce an adequate reduction in LDL-C levels, and investigate why individuals vary in their response to dietary therapy. Each subject was placed on a one-month high-fat (40%), high cholesterol (450 mg/day), high-saturated fatty acid (16%), and low polyunsaturated fatty acid (5%) diet (Hi-Sat) (largely a continuation of their habitual diet, and resembled the average diet for American men). Each subject was also placed on a four-month low-fat (≤30%), low cholesterol (≤300 mg/day), low-saturated fatty acid (<10%), and high polyunsaturated fatty acid (10%) diet (Lo-Sat) (NCEP Step I Diet). Lipid levels were measured five times during the last two weeks of each dietary period and averaged for each subject. There was a significant ($p \leq 0.00001$) -8% reduction in LDL-C mean values after the Lo-Sat diet (177±25 mg/dL), as compared to the Hi-Sat diet (192±28 mg/dL).

Jenkins and colleagues (1993) studied mild to severely hyperlipidemic males (N=15) and females (N=28) (29-70 yrs.). All subjects were between 95 and 172% of ideal body weight. The subjects were randomly assigned to two metabolically controlled-diets,

each for four months. The metabolic diets were low in saturated fatty acid (<4%), cholesterol (<25 mg/1000 Kcal), high in carbohydrate (≥60%), and very high in fiber (> 224g/1000 kcal). One diet was high in soluble fiber, and the other was high in insoluble fiber. Each four-month dietary period was separated by a two-month NCEP Step II Diet (30% fat, 7% saturated fat, polyunsaturated fat to saturated fat ratio 1:4, < 200 mg dietary cholesterol/day). Fasting blood samples were taken a baseline, two, four, eight, 12, 14, and 16 weeks of each metabolic diet. During both metabolic diets, LDL-C levels fell to their lowest levels by week four and stayed the same for the remaining three months. Men (9.6±1.9%, $p \leq 0.001$) demonstrated a greater percentage reduction than women (2.2±1.4%, $p=0.141$) following the soluble-fiber diet. There were no significant ($p>0.001$) differences for LDL-C levels between men and women following the insoluble-fiber diet. Men and women, combined, demonstrated significant ($p \leq 0.001$) decreases at baseline and after the soluble- (180±7 to 154±5 mg/dL) and insoluble- (177±6 to 161±6 mg/dL) fiber diets.

Research (Clevidence et al., 1992) conducted on 42 males (19-56 yrs.) investigated the influence of a low-fat, high-fiber diet on blood lipid level in men with desirable to moderately elevated cholesterol concentration. Each subject consumed the low-fat diet (19% fat, 4.4% saturated fatty acid, 18 mg cholesterol/MJ, 19.3g fiber/1000 kcal) and a high-fat diet (41% fat, 15% saturated fatty acid, 45 mg cholesterol/MJ, 8.4g fiber/1000 kcal) for ten weeks and were compared to the subject's normal dietary intake (35% fat, 12% saturated fatty acid, 36 mg cholesterol/MJ, 8.2g fiber/1000 kcal). Fasting blood samples were taken at baseline and after the ten-week high-fat and ten-week low-fat dietary period. Mean values for LDL-C were significantly ($p=0.004$) higher when subjects consumed the high-fat rather than the pre-study normal dietary intake: 3.10±0.11 mmol/L to 3.39±0.10 mmol/L, respectively. Mean LDL-C levels significantly ($p \leq 0.001$) decreased from 3.10±0.11 mmol/L to 2.71±0.10 mmol/L when subjects ate the low-fat diet versus the pre-study normal dietary intake. Subjects demonstrated the most significant ($p \leq 0.001$)

decrease in LDL-C values when the low-fat diet was consumed rather than the high-fat diet: 3.39±0.10 mmol/L to 2.71±0.10 mmol/L, respectively. There were no significant ($p>0.001$) differences in LDL-C levels between the pre-study dietary intake (3.10±0.11 mmol/L) and post-study dietary intake (3.10±0.10 mmol/L).

In summary, the previous dietary intervention investigations (Clevidence et al., 1992; Sacks et al., 1986; Jenkins et al., 1993; Denke et al., 1994) reported favorable changes in LDL-C levels as a result of dietary intervention. All studies demonstrated significant reductions in LDL-C for males and females following the experimental period.

Resistance Training Only

A five month resistive exercise program (Boyden, 1993) on healthy, normal weight, premenopausal women (N=46) found a significant ($p\leq0.04$) decrease in LDL-C levels as compared to the control group (N=42). Subjects exercised three days per week for one hour per training session. Training consisted of 12 exercises performed at 70% of RM, using three sets of eight repetitions per exercise. Subjects did not change from their regular dietary habits. Fasting blood samples were drawn 36 to 48 hours after an exercise bout at baseline and after five months of training. Mean LDL-C concentration of the exercise group was significantly ($p\leq0.04$) higher than that of the control group at baseline, 115.8±4.2 and 105±2.7 mg/dL, respectively. A significant ($p\leq0.01$) -8.8% decrease was noted in the mean concentrations of LDL-C in the exercise group (101.7±3.7 mg/dL) after the training period that was significantly ($p\leq0.039$) different from those in the control group (102.6±3.6 mg/dL).

Ullrich, Reid, and Yeater (1987) investigated the changes in LDL-C on a group of healthy untrained men (N=25) who participated in a weight training program for eight weeks. The subjects were randomly assigned to one of four groups: endurance (two sets of 15 repetitions of a 15 to 18 repetition maximum (RM) load), strength I (three sets of six repetitions of a six to eight RM load), Strength II (one set of three repetitions of a three to

five RM load once per week and one set of ten repetitions of a ten to 12 RM load twice per week), and explosive (started at one set of 15 repetitions of a 40% of 1 RM load done as quickly as possible, then progressed to do one set of 15 repetitions of a 15 to 18 RM load). All subjects performed nine exercises during each training session which was conducted three times per week. Diet was not altered throughout training. Fasting blood samples taken 36 hours after the last exercise session demonstrated improved changes in plasma lipids. LDL-C values, combined for all subjects, significantly ($p \leq 0.05$) decreased from 132 mg/dL at baseline to 121 mg/dL after training, an -8% decrease.

Research was done on 6,653 men and women to determine the relation between muscular strength and serum lipoprotein and lipid status. Subjects were tested for maximum upper and lower body strength (1 repetition maximum (1 RM) bench press and leg press) and 12-hour overnight fasting blood samples were taken. After adjustments for age, body composition, and cardiovascular fitness, results showed no significant association between upper and lower muscular strength and LDL-C for either men or women (Kohl, Gordon, Scott, Vaandrager, & Blair, 1992).

Kokkinos et al. (1989) found no significant ($p>0.05$) changes in LDL-C (139±16 to 136±21 mg/dL) in untrained males (N=15) with abnormal lipoprotein-lipid profiles following an 18 week strength training program. Two blood samples were taken from the training and inactive group, before and after the training, to control for day to day variations.

Hurley et al. (1988) determined if a 16-week high-intensity resistance training program (three days/week, 14 exercises, eight to 20 repetitions, one set/exercise) could improve LDL-C profiles in untrained males (N=11) as compared to a control group (N=10). Dietary intake was not altered throughout training. Twelve- to 14-hour overnight fasting blood samples were taken before and after training period. The training group experienced a significant ($p \leq 0.05$) -5% reduction (129±15 to 122±14 mg/dL), as compared to the control group (140±17 to 142±19 mg/dL).

Researchers of the University of Northern Colorado Wellness Letter (Muscling in on Cholesterol, 1994) analyzed 11 studies to determine the effects of weight training on elevated cholesterol levels. Results of the meta-analysis revealed that weight training lowered LDL-C levels by 13%.

Poehlman and colleagues (1992) analyzed the LDL-C levels in a group of aerobically-trained (N=36) (AT) (running an average of 4.8±1.2 years, 42-85 km/week), versus a group of resistance-trained (N=18) (RT) (training an average of 4±1 years, five to six days/week, intensity is 70-85% of 1RM, 10-20 repetitions, using three to five sets), and untrained (N=42) (UT) (no regular participation of any form) males. Fasting blood samples for all subjects were taken 36 to 48 hours after last exercise session. LDL-C levels were significantly ($p \leq 0.05$) higher in the UT group (98±24 mg/dL) as compared to the RT (82±21 mg/dL) group; whereas, there was no significant ($p > 0.05$) difference in LDL-C values between the AT (89±29 mg/dL) and RT groups, or the AT and UT groups. Percent intake of dietary fat was the highest single correlate with LDL-C levels, and after control for this variable, there was no significant ($p > 0.01$) difference noted among the three groups.

Research (Johnson et al., 1982) also tried to determine the effects of a 12 weeks of resistance exercise (N=14) (RE) or aerobic exercise (N=10) (AE), as compared to a control group (N=10) (CG). The RE group trained with weights three days/week for 45-60 minutes, using exercises for the major muscle groups. The AE group was already engaged in regular exercise prior to the study (primarily running approximately 10 km/week. The RE and CG groups had been relatively sedentary for at least six months prior to beginning the training. Fasting blood samples were taken prior to initiation of exercise, and after six and 12 weeks of exercise. The RE group experienced dramatic, significant ($p \leq 0.05$) decreases in LDL-C levels at baseline, six, and 12 weeks (169±16, 160±16, & 122±16 mg/dL). Even though both the AE (119±21, 117±21, & 109±21 mg/dL), and CG

(139±21, 136±21, & 131±21 mg/dL) groups experienced a decrease in LDL-C levels, they were not significant ($p>0.05$). No further comparisons were measured between groups.

In summary of the previous resistance training study using healthy males who were weight-trained for for eight weeks, Ullrich et al. (1987) found significant decreases in LDL-C levels. Of the previous resistance training studies, similar alterations in LDL-C concentrations have been reported by Johnson et al. (1982) and Boyden (1993). Hurley et al. (1988) also found significant reductions in LDL-C in untrained males following a 16-week high-intensity resistance training program; whereas; Kokkinos et al. (1989) deomonstrated no changes in LDL-C in males with abnormal blood lipid profiles after 18 weeks of strength training.

Summary

Investigators agree that dietary modification is the first line of therapy in the treatment of elevated cholesterol (Seim & Holtmeier, 1992). Several studies have consistently shown that diets low in fat and/or high in fiber can reduce serum cholesterol as compared with diets high in fat (Bell et al., 1990; Denke et al., 1994; Jenkins et al., 1993). There was also several studies investigating the effects of strength training on serum lipid levels, but the studies were inconclusive and inconsistent (Goldberg et al., 1984; Ullrich et al., 1987; Johnson et al., 1982; Weltman, 1987).

Several studies (Ginsberg et al., 1990; Wood et al., 1988; Denke et al., 1994) using only dietary intervention demonstrated favorable improvements in lipid-lipoprotein profiles. However, some studies did show evidence of design limitations. Sacks et al. (1986) and Clevidence et al. (1992) used a dietary protocol designed to be similar to a protocol that would be administered in a medical practice. Several studies (Kasim et al., 1993; Bell et al., 1990; Boyd et al., 1990) had subjects self report their dietary intakes using daily food records. Few studies (Sndstrom et al., 1992; Clevidence et al., 1992;

Jenkens et al., 1993) also used a protocol in which all meals for experimental feeding were prepared by dietitians and no participants.

In the literature reviewed, authorities agree that resistance training is increasing in popularity for men and women, but the results of this type of exercise on lipoprotein-lipid levels are less well characterized (Boyden, 1993). No consistent alterations have been noted in serum lipid profiles with resistance training (Boyden, 1993) only, as compared with diet only (Sandstrom et al., 1992; Ginsberg et al., 1990). Table I may be referred to for a complete summary of all research articles mentioned previously.

Many of the studies using only resistance training did show evidence of design flaws or limitations that make results difficult to interpret and conclusions questionable. For example, some studies only used subjects who have low risk profiles (Kokkinos et al., 1988; Fripp et al., 1987; Goldberg et al., 1988; Ullrich et al., 1987), no separate control group (Goldberg et al., 1984; Ullrich et al., 1987), no control for anabolic steroid usage (Hurley et al., 1984; Hurley et al., 1989; Morgan et al., 1986), and lack of control for dietary intake and observation (Fripp et al., 1987; Weltman et al., 1987; Ullrich et al., 1987; Hurley et al., 1989) which can effect the outcome of test results. Several studies (Smutok, et al., 1993; Hurley et al., 1989; Johnson et al., 1982; Fripp et al., 1987; Hurley et al., 1988; Weltman et al., 1987) only took one blood sample at baseline and after training; hence, there was no control for normal day to day variation of lipid levels. Also, some blood samples were taken directly after the end of the last training session (Wallace et al., 1991; Weltman et al., 1987), and it is possible these results represent only short-lived, acute exercise effects (Hurley, 1989; Wallace et al., 1991).

Kokkinos and Hurley (1987) also reviewed several cross-sectional and longitudinal studies on the effects of weight training on lipoprotein-lipid profiles. Data that was collected suggested that weight training may reduce LDL cholesterol and raise HDL cholesterol. However, the information is inconclusive and should be interpreted with caution due to various reasons such as lack of control for day to day variations of

lipoprotein during training period, dietary intake, and shifts in plasma volume; most subjects had a normal CAD risk profile prior to training; it was difficult to determine whether reductions in lipoprotein levels were due to training adaptations or to the acute, short-term exercise effects; and lack of information is available concerning training thresholds necessary for eliciting changes in lipoprotein levels.

These design flaws, among others, suggest that a lack of control for some of these limitations are major barriers for consistency in assessing resistance training and its effects on lipoprotein-lipid profiles.

On the other hand, available research consistently indicated that strength training can be used in a direction favorable for improved health benefits and total fitness. In conclusion, because of design flaws and methodological limitations of previous studies, there is inconsistent and inadequate information to determine whether resistance training is effective in improving lipoprotein-lipid profiles.

Further studies in this area that address these design flaws are clearly needed. The present study will investigate the effects of combined dieting and resistance training on lipoprotein-lipid concentration.

TABLE I

COMPLETE SUMMARY OF RESEARCH ARTICLES

TOTAL CHOLESTEROL

Authors	Intervention	Percent Changes	Significance
Anderson 1993	diet	-7.3% † -8.8% ‡	P ≤0.0001 p ≤0.0001
Anderson 1993	diet	-10% § -13% §	p >0.05 p ≤0.004
Kasim et al., 1992	diet	-7% ‡	p >0.05
Sandstrom et al., 1992	diet	-9.8% † -15.6% ‡	p ≤0.001 p ≤0.001
Ginsberg et al., 1990	diet	-14.3 mg/dL † -17.8 mg/dL †	p ≤0.025 p ≤0.025
Boyd et al., 1990	diet	-4.0% ‡	p =0.07
Anderson et al., 1990	diet	-12.1% ‡; -11.3% ‡ -10.2% †; -8.2% † -3.5% †	p ≤0.0001 p ≤0.001 p ≤0.022
Manning et al., 1991	res. tr.	↔; ‡	p >0.0p
Kokkinos et al., 1988	res. tr.	-4% † +6% †	p >0.05 p >0.0p
Kokkinos et al., 1991	res. tr.	↔; †	p >0.05
Durak et al., 1990	res. tr.	-8.1% †	p =0.015
Smutok et al., 1993	res. tr.	↔; †	p >0.05
Weltman et al., 1988	res. tr.	-5.7% †	p ≤0.05
Blessing et al., 1987	res. tr.	-0.8% †	p ≤0.05
Stone et al., 1982	res. tr.	-3.6% †	p >0.01

res. tr.=resistance training
†, Men only
‡, Women only
§, combined groups

(CONT. TABLE I)

HIGH-DENSITY LIPOPROTEIN

Authors	Intervention	Percent Change	Significance
Bell et al., 1990	diet	-8.4% †	$p \leq 0.005$
		-9.7% †	$p \leq 0.005$
Anderson et al., 1992	diet	-3.0% §	$p \leq 0.01$
		+3.2% §	$p \leq 0.01$
Wood et al., 1988	diet	+11% †	$p \leq 0.01$
Williams et al., 1994	diet	+20.5% †	$p \leq 0.05$
		+9.3% †	$p \leq 0.05$
		+9.4% †	$p \leq 0.05$
Fripp et al., 1987	res. tr.	+28.6% †	$p \leq 0.01$
Goldberg et al., 1984	res. tr.	+15.8% †	$p \leq 0.05$
		+4.8% ‡	$p > 0.05$
		+8.5% §	$p > 0.05$
Wallace et al., 1991	res. tr.	↔; †	$p > 0.01$
Hurley et al., 1987	res. tr.	-29% †	$p \leq 0.01$
		-37% †	$p \leq 0.01$

res. tr.=resistance training
†, Men only
‡, Women only
§, combined groups

(CONT. TABLE I)

LOW-DENSITY LIPOPROTEIN

Authors	Intervention	Percent Change	Significance
Sacks et al., 1986	diet	-18% §	$p \leq 0.001$
Denke et al., 1994	diet	-8.0% †	$p \leq 0.00001$
Jenkins et al., 1993	diet	-9.6% † -2.2% ‡	$p \leq 0.001$ $p = 0.141$
Clevidence et al., 1992	diet	-12.5% † -20% †	$p \leq 0.001$ $p \leq 0.001$
Boyden 1993	res. tr.	-8.8% ‡	$p \leq 0.04$
Ullrich et al., 1987	res. tr.	-8.0% †	$p \leq 0.05$
Kokkinos et al., 1989	res. tr.	–2.2% †	$p > 0.0$
Hurley et al., 1988	res. tr.	-5.0% †	$p \leq 0.05$
Johnson et al., 1982	res. tr.	-27.8% †	$p \leq 0.05$

res. tr.=resistance training
†, Men only
‡, Women only
§, combined groups

CHAPTER III

PROCEDURES FOR RESEARCH

This research was an investigation of the effects of a six week resistance training program, combined with a modified-caloric, low-fat, high-fiber diet on lipoprotein-lipid profiles in males and females.

Subject Selection

The subjects for this study consisted of volunteers were drawn from Oklahoma State University campus and the city of Stillwater, Oklahoma. The subjects were eight males and nine females ranging in age from 25 to 60 years. The mean age in years for women, men, and women and men combined were 42±8.6, 36.6±13.7, and 39.5±11.3, respectively. The mean weight in lbs. for women, men, and women and men combined were 164.6±23.4, 239.5±51.5, and 199.5±37.6, respectively. All subjects were non-smokers (no smoking within six months of the study) prior to the study. No pre- and post-measurements were taken for height. Two subjects dropped out during the study (one female, & one male); therefore, only complete data was obtained for 15 of the subjects (seven males, and eight females).

Each participant was required to complete a Biometrics-One-on-One Health Questionnaire, and sign the informed consent statement supplied by Biometrics-One-on-One program. Participants were also required to sign an informed consent permitting blood samples to be taken and a metabolic exercise test to be administered, which was approved and provided by the Oklahoma State University Wellness Center.

Methods and Procedures

All pre- and post-test measurements were taken in the morning after a 12-hour overnight fast. Blood samples were taken 12 to 24 hours after the last exercise session. Measurements included bodyweight (lb.), blood pressure, 11-site circumference test (Biometrics, 1993), Jackson & Pollock seven-site skinfold test (Golding, Myers, & Sinning, 1989), and a six-hour fasting metabolic exercise test. Blood measurements, taken relative to this study, were 12-hour overnight fasting blood tests (SMA-25). Fasting blood samples were taken at the same time in the morning prior to the first training session and 12 to 24 hours after the last training session.

Pre- and post-venous blood samples were drawn from the brachial fossa of either the right or left arm of each subject. A standard aseptic venipuncture technique was employed with the tourniquet being released prior to removal of the #21 gauge needle. All subjects were in the sitting position during venipuncture. Vacutainer red stoppered tubes (serum separation tubes, SST) were used in venous collection. These have a floating gel to aid in separation of the red cells from the serum. One tube, of approximately 10 mL of blood, per subject was collected. All venous samples were allowed to stand at room temperature for a minimum of 30 minutes and a maximum of one hour 30 minutes until a clot formed in the tube. The samples were then centrifuged at a speed of 5,000 revolutions per minute for ten minutes in a table top clinical centrifuge (Roche Biomedical Laboratories VanGuard 6000). The gel separated the red cells from the serum. The Vacutainer tubes were collected by Roche Biomedical Laboratories personnel for measurement according to the Allain-Trinder method (Allain et al., 1974; & Trinder, 1969), at the Kansas City, Missouri regional laboratory. Results are received within 24 hours via the teleprinter which is located at the Oklahoma State University Wellness Center Laboratory.

Diet Program

Dietary intake was controlled throughout the six week training period. Each subject was provided with a customized six week meal plan which incorporated information from current dietary guidelines and the Food Pyramid, which promotes a low-fat, high-fiber (~ 25 grams/day) diet. Participants chose individual menus suited to their individual preferences and nutritional concerns.

All subjects were provided a nutrient distribution plan consisting of 58% carbohydrate, 22% ±2%, fat and 20% protein. Also, two-thirds ounces (oz.) of water per lb. of bodyweight were consumed daily, but not to exceed 160 oz. per day.

Females had a daily caloric intake of 1400 kcal weeks one and two, 1300 kcal weeks three and four, and 1200 kcal weeks five and six. Males had a daily caloric intake of 1800, 1700, 1600 kcal weeks one and two, weeks three and four, and weeks five and six, respectively. Daily caloric adjustments (1600 kcal weeks one and two, 1500 kcal weeks three and four, and 1400 kcal weeks five and six) were made if male subjects were shorter than 5'8", females were taller than 5'10", or if females were shorter than 5'2" and weighed less than or equal to 125 lbs (1200 kcal weeks one and two, 1100 kcal weeks three and four, 1000 kcal weeks five and six). No one in the present study was affected by these changes. Compliance of food and water intake was the responsibility of the personal trainer and were verbally self-reported by the participants during the time of weigh-in before each exercise session.

Exercise Program

Each training session was conducted, one on one with personal trainers, during the entire six weeks. Training sessions were conducted three days per week on non-consecutive days, for approximately 30 minutes per training session. All training sessions were preceded with a weigh-in and a five minute warm-up, and were concluded with full-

body assisted stretching exercises. The equipment used for training was Universal Machines™ and free weights.

Initial light-weight resistance of 60-70% of the subjects ten repetition maximum (10RM) was used. The resistance training exercises used during weeks one and two were knee extension, knee flexion, chest press, lateral pull-down, lateral shoulder raise, and abdominal crunches. Bicep curls and tricep press downs were incorporated during weeks three through six. Training consisted of one set of six repetitions with a ten second count during each concentric (positive) contraction and a two second count during each eccentric contraction.

The first training session consisted of instruction emphasizing proper form and lifting techniques using light weight resistance. After first training period and throughout remaining training period, resistance was increased, accordingly, when the participant could perform six unassisted repetitions with proper form. Personal trainers were responsible for encouragement, support, and verification that each participant completed the exercises thoroughly and correctly.

Statistical Analysis

Data were collected before and after the six-week training/diet period for statistical analysis. Two way analysis of variance (ANOVA) with repeated measures was used to determine possible differences by treatment, gender, and gender by treatment. Any significant gender by treatment differences ($p \leq 0.05$) were further delineated using a post-hoc test.

CHAPTER IV

ANALYSIS OF DATA AND DISCUSSION

The purpose of this research study was to determine if a six-week resistive training program combined with a low-fat, high-fiber diet would significantly improve lipoprotein-lipid profiles. The possible differences based on time, gender , and gender by time were explored.

The null hypotheses of this study stated there would be no significant changes in lipoprotein-lipid levels, based on pre- and post-treatment values (time), no significant gender differences in lipoprotein-lipid concentration, and no significant interaction differences based on gender and time. Significance for this study was set at the $p \leq 0.05$ level. A two-way analysis of variance with repeated measures was used to statistically analyze the data.

Results

Pre-training and post-training mean values for TC, HDL-C, LDL-C, TC/HDL-C, and TG for men, women, and combined groups are presented in Table II. Table III presents the difference based on the pre- and post-treatment values for men and women combined (time), gender difference, and gender by time interaction.

Total cholesterol significantly ($p \leq 0.05$) decreased from 203.5±33.08 to 166.7±30.10 mg/dL based on pre- and post-treatment values for combined groups (time). There was no difference based on gender or gender by time interaction.

Pre- and post-treatment values for HDL-C levels significantly ($p \leq 0.05$) decreased from 44.1±13.61 to 39.8±9.77 mg/dL. Values for males (49.2±11.06 mg/dL)

TABLE II

PRETREATMENT & POSTTREATMENT MEAN VALUES AND PERCENT CHANGES FOR MALES AND FEMALES*

TOTAL CHOLESTEROL

	Pre	Post	% Change
Males	209.3±45.44	171.0±38.30	-18.3%
Females	198.5±18.90	162.9±22.80	-18.0%
Combined	203.5±33.08	166.7±30.10	-18.1%

HIGH-DENSITY LIPOPROTEIN

	Pre	Post	% Change
Males	35.14±5.73	32.0±3.30	-9.0%
Females	51.9±13.90	46.6±8.23	-10.1%
Combined	44.1±13.61	39.8±9.77	-9.8%

LOW-DENSITY LIPOPROTEIN

	Pre	Post	% Change
Males	139.4±42.23	111.9±33.96	-19.7%
Females	124.4±23.30	99.8±25.34	-19.8%
Combined	131.4±33.10	105.4±29.23	-19.8%

* $\bar{x}$ ±SD.

(CONT. TABLE II)*

TOTAL CHOLESTEOL/HIGH-DENSITY LIPOPROTEIN RATIO

	Pre	Post	% Change
Males	6.07±1.72	4.20±2.67	-11.9%
Females	4.04±1.24	3.54±0.91	-12.4%
Combined	4.98±1.77	4.39±1.41	-11.8%

TRIGLYCERIDES

	Pre	Post	% Change
Males	171.1±60.71	133.0±47.74	-22.3%
Females	111.4±52.83	80.0±24.39	-28.2%
Combined	139.3±62.67	104.7±44.98	-24.8%

* $\bar{x}$±SD.

TABLE III

VALUES FOR TIME, GENDER,
& TIME BY GENDER INTERACTION

TOTAL CHOLESTEROL

Source	SS	df	MS	F
Gender	667.55	1	667.55	0.34
Error	25568.65	13	1966.82	
Time	10197.21	1	10197.21	75.85†
Gender X Time	13.21	1	13.21	0.10
Error	1747.65	13	134.43	0.10

HIGH-DENSITY LIPOPROTEIN

Source	SS	df	MS	F
Gender	1835.44	1	1835.44	12.82†
Error	1861.43	13	143.19	
Time	131.49	1	131.49	7.62†
Gender X Time	8.29	1	8.29	0.48
Error	224.18	13	17.24	

†; $p \leq 0.05$

(CONT. TABLE III)

LOW-DENSITY LIPOPROTEIN

Source	SS	df	MS	F
Gender	1377.05	1	1377.05	0.72
Error	25003.15	13	1923.32	
Time	5085.67	1	5085.67	73.07†
Gender X Time	16.21	1	16.21	0.23
Error	904.79	13	69.60	

TOTAL CHOLESTEROL/HIGH-DENSITY LIPOPROTEIN RATIO

Source	SS	df	MS	F
Gender	27.72	1	27.72	9.09†
Error	39.63	13	3.05	
Time	2.75	1	2.75	8.87†
Gender X Time	0.09	1	0.09	0.28
Error	4.03	13	0.31	

†; $p \leq 0.05$

(CONT. TABLE III)

TRIGLYCERIDES

Source	SS	df	MS	F
Gender	23737.63	1	23737.63	6.91†
Error	44636.37	13	3433.57	
Time	9021.10	1	9021.10	7.90†
Gender X Time	85.50	1	85.50	0.07
Error	14848.37	13	1142.18	

†; $p \leq 0.05$

were significantly greater than values for females (33.6±4.50 mg/dL) (gender), but there was no significant ($p \leq 0.05$) difference noted in gender by time interaction.

Pre- and post-treatment values for LDL-C levels also revealed a significant ($p \leq 0.05$) decrease (131.4±33.10 to 105.4±29.23 mg/dL) (time). No significant difference was shown for gender or for gender by time interaction.

TC/HDL-C ratio decreased significantly ($p \leq 0.05$) from 4.98±1.769 to 4.39±1.406 for pre- and post-treatment values (time). There was also a significant ($p \leq 0.05$) difference based on gender. Values for males (5.36±1.259) were significantly greater than for females (3.54±0.909). No significant interaction based on gender by time was noted.

Pre- and post-treatment values for TG demonstrated a significant ($p \leq 0.05$) decrease (139.3±62.67 to 104.7±44.98 mg/dL) (time), as well as, a significant ($p \leq 0.05$) gender difference. Values for males (95.7±38.61 mg/dL) were significantly lower than for females (152.1±54.22). Again, no significant difference was demonstrated in the gender by time interaction.

Discussion

The research design of this study allowed for some unique observations to be made since no previous project has investigated the combined effects of resistive training and dietary intervention of lipid-lipoprotein concentration; therefore, no direct statistical comparison can be made with any other study. The data compiled from the study indicates that both males and females experienced lipid-lipoprotein changes from the resistance training/dietary intervention program.

Compared to the present study, Anderson (1993) also found that women and men had similar TC decreases following a short-term dietary intervention program. Other dietary intervention investigations (Sandstrom et al., 1992; Ginsberg et al., 1990) also reported findings similar to those of the present study due to complex carbohydrates and

fiber replacing fatty acids. The present study, as well as, the reports of Goldberg et al. (1984) and Ullrich et al. (1987) concluded that resistive training increased muscle mass and decreased percent body fat which favorably affected TC levels.

Due to the short-term length of the present study, it was considered a continuous period of weight-loss and no weight stabilization period occurred; therefore, HDL-C levels decreased. Seim and Holtmeier (1992) suggested a possible mechanism for the reduction in HDL-C levels following the experimental period. Early in weight loss, HDL-C will drop due to the restriction of dietary fat and total calories which is the case in the present study. Kasim et al. (1993) futher concludes that additional intakes of complex carbohydrates, fiber, and polyunsaturated fats which replace saturated fats may reduce HDL-C levels. Other resistance training (Hurley et al., 1987; Kokkinos et al., 1991) and dietary intervention studies (Kasim et al., 1993; Nieman et al. 1990; Sandstrom et al., 1992) also concluded that during active weight-loss periods, HDL-C levels were found to slightly decrease. It was also suggested that physical activity tends to elevate HDL-C levels; therefore, the present study may have lacked the adequate volume of exercise to elicit these benefits.

Study duration also indicated an effect on HDL-C levels. Dattilo and Kris-Etherton (1992) reported that studies lasting less than or equal to six weeks (mostly active weight-loss) were associated with a decrease in HDL-C levels. As study duration increases, subjects begin to stabilize in weight (mostly stabilized weight-loss), there was a strong association with HDL-C increases.

LDL-C levels fell sharply in the subjects following the resistance training/dietary intervention program. This finding from the present study compares favorably with the results from the previous resistance training studies (Hurley et al., 1988; Fripp & Hodgson, 1987); however, the physiological mechanism by which resistance training might lower LDL-C levels is unknown. Other dietary intervention investigations (Sacks et al., 1986; Ginsberg wt al., 1990; Denke & Grundy, 1994) also found that decreases in

LDL-C levels were directly related to decreases in animal fat and saturated fat following a long-term dietary period.

The present study and other resistance training investigations (Weltman et al., 1987; Johnson et al., 1982) found that initial elevated levels of TC may explain the favorable reduction in TC/HDL-C ratio levels following the experimental period. The current data support the findings of Hurley et al. (1988) who reported that resistive training significantly lowered TC/HDL-C ratio levels due to a large reduction in TC and slight increase in HDL-C levels after the training period. Other investigators (Wood et al., 1988; Poehlman et al., 1992) using dietary intervention also produced favorable changes in TC/HDL-C ratio levels. It was concluded that the improvement in TC/HDL-C ratio levels was directly related to reduction in weight loss and percent body fat.

The possibility that TG improvements found in the present investigation and other dietary intervention program (Barnard, 1991; Bell et al., 1990; Nieman et al., 1990) could be the result of reduced consumption of refined sugar (sucrose and fructose) and increased intakes of complex carbohydrates. Kasim et al. (1993) also reported that improvements in TG was significantly related to reduction in weight loss following diet. Dattilo and Kris-Etherton (1992) reported that for every Kg reduction in body weight would result in a 0.015 mmol/L dercease in TG. Similar TG reduction were also experienced following a high-internsity weight training program (Goldberg et al., 1984). In constrast to the present study, Boyden (1993) reported no significant decreases in TG, as well as, weight loss following a resistance training program.

While two primary life-style modifications, diet and exercise (resistance training), were contained in the program, it is concluded that diet played the most significant role in lowering lipid-lipoprotein profiles during the six-week period. Even though there was no attempt to separate the possible effects of resistance training and diet, it has been consistently demonstrated, as shown previously, that dietary intervention alone has a major effect on plasma lipid and lipoproteins in subjects; whereas, resistance training alone has

found more inconsistent results. Dietary intervention controls the calorie and fat intake which results in weight loss (fat); whereas, the resistance training preserves and even increases lean muscle mass tissue (Biometrics, 1993).

CHAPTER V

SUMMARY, CONCLUSIONS, RECOMMENDATIONS

Summary

Biometrics-One-on-One® is a six-week personalized resistance training and dietary intervention program. The purpose of the study was to investigate the effects of the six-week Biometrics One-on-One® program on a group of males and females (Boimetrics, 1993). Participants completed all aspects of the program relative to the current study.

Pre- and post-training data, relative to this study, was collected for all subjects and was analyzed by using an two-way analysis of variance with repeated measures to compare possible differences by treatment, gender, and treatment by gender interaction. Significance for this study was set at $p>0.05$.

A significant ($p<0.05$) decrease was noted for all lipoprotein-lipid parameters for treatment (time). There were significant ($p\leq0.05$) gender differences in HDL-C, TC/HDL-C ratio, and TG. Values for HDL-C and TC/HDL-C in males (49.2±11.06 mg/dL; & 5.36±1.26) were significantly ($p\leq0.05$) higher than for females (33.6±4.50 mg/dL; & 3.54±0.91), respectively. Values for TG in males (95.7±38.61 mg/dL) was significantly ($p\leq0.05$) lower than for females (152.1±54.22 mg/dL). No significant interaction based on time and gender was noted for any lipoprotein-lipid parameters.

Conclusions

The purpose of this study was to determine if a six-week resistance training program combined with a modified-caloric, low-fat, high-fiber diet would significantly improve lipoprotein-lipid profiles. Any significant differences between males and females in lipoprotein-lipid profiles were also determined.

The null hypothesis stated that no changes in lipoprotein-lipid concentration for either males or females after resistance trianing and diet was rejected. Values significantly ($p \leq 0.05$) decreased for TC (203.5±33.08 to 166.7±30.10 mg/dL), HDL-C (44.1±13.61 to 39.8±9.77 mg/dL), LDL-C (131.4±33.10 to 105.4±29.23 mg/dL), TC/HDL-C (4.98±1.77 to 4.39±1.41), and TG (139.3±62.67 to 104.7±44.98 mg/dL). The second hypothesis stating no differences in lipoprotein-lipid concentrations after resistance training and diet based on gender was rejected. Values for males were significantly ($p \leq 0.05$) greater than for females in HDL-C (49.2±11.06 mg/dL; 33.6±4.50 mg/dL) and TC/HDL-C ratio (5.36±1.26; 3.54±0.91), respectively. Values for males were significantly ($p < 0.05$) lower than for females (95.7±38.61 mg/dL; 152.1±54.22 mg/dL) in TG, respectively. The third hypothesis stating that no significant lipoprotein-lipid difference after resistance training and diet will be found based on gender by time interaction was accepted. The conclusions of this study indicates that both males and females benefited from the diet and resistance training program.

Recommendations

Further investigations dealing with resistance training alone, diet alone, and especially the combined effects of resistance training and diet are needed. The issue of whether strength training and diet combined can favorably alter blood lipid profiles has not been adequately researched in the past or addressed in the current literature available.

Future research should be directed towards the combined effects of resistance training and diet as to generate greater support. A more appropriate research design would compare a diet-only group, resistance training-only group, diet and resistance training combined group, and a control group to determine the effects on lipoprotein-lipid profiles. Studies should separate diet from resistance training to better determine and assess the change in lipoprotein-lipid concentration due to the training and due to diet.

The current research, as well as, other previous resistance training (Durak et al., 1990; Ullrich et al., 1987; Goldberg et al., 1984) and dietary modification (Denke & Grundy, 1994; Clevidence et al., 1992) studies failed to use a separate control group which can lead to inaccurate conclusions. Without the separate control group, there is no method of determining what changes occur in the absence of the independent variable (diet or resistance training); therefore, it is important that furture studies implement a separate control group.

Wallace et al. (1991) stated that a dose-response relationship between resistance exercise and lipoprotein modifications may exist; therefore, this would suggest that the volume of exercise (degree of resistance, number of repetitions) which is directly proportional to the total caloric cost of exercise may be a critical factor in affecting lipoprotein modifications. More research information needs to be made available concerning training thresholds (degree of resistance and number of repetitions) necessary for eliciting favorable lipoprotein-lipid changes. The current study is challenging, but incorporates a low-intensity, low-volume of resistance training throughout experimental period. Blessing et al. (1987) also reported the greatest change in lipid-lipoprotein concentration were obtained during the highest volume of resistance training.

The results (Kokkinos et al., 1988) obtained from low risk groups, such as the current study, may not be representative of the population that would benefit most from risk factor intervention. High risk subjects may respond differently to training; therefore, future investigations may focus on subjects who are at risk for CAD.

Strength training exercises have shown to produce reductions in plasma volume which can affect day to day variations. These alterations in plasma volume are short-lived and return to basline values soon after the completion of a training session. No inforamtion is available to further determine the length of time in which it takes plasma volume to return to baseline values after a training program. Wallace et al. (1991) stated it is necessary to adjust for variations in plasma volume in order to distinquish between changes in plasma

volume itself from those due to entirely resistance training. Future research needs to determine a method to be able to verify if any observed changes in lipid and lipoprotein concentration is due to plasma volume itself or due entirely to exercise.

The current investigation required subjects to consume large quantities of water which can also affect day to day variations in plasma volume. Future studies should include continuous monitoring and adjusting of plasma volume shifts throughout entire experimental period, as well as, blood volume calculations which correct for alterations in blood volume.

Future studies need to obtain several blood samples which are representative of a collection period to control for individual daily variables. Blood samples need to be collected at the same time in the morning after a minimum 12-hour overnight fast. It is suggested that several blood collections should be taken, at least, 48 to 72-hours after last exercise bout as to distinguish between an acute exercise effect or chronic training adaptations. More information is needed to determine the length of time that acute effects persist following exercise.

Another suggested area for further research is investigating the mechanism causing a gender difference in the response to diet and exercise. Results from the current study have shown that men and women demonstrated similar responses in blood lipid profiles after the experimental phase; however, Dattilo and Kris-Etherton (1992) found conflicting results. Anderson (1993) also states that gender should be considered when designing a clinical trial or intervention program. Future studies should attempt to separate the results of males and females to better determine the response patterns to the intervention program.

When body composition is not assessed, it is difficult to make valid conclusions concerning the independent effect of the training/diet program. More controlled studies need to be able to determine the independent effects of the training /diet program on plasma lipid-lipoprotein. Continuous monitoring and adjusting of caloric intake throughout the entire training/diet program to adjust for body composition changes is imperative.

In summary, the present research allowed for several unique observations to be made since no other research has investigated the combined effects of resistance training/diet on lipid-lipoprotein levels. The current study also made several unique findings concerning gender based and gender by time differences in lipid-lipoprotein levels; however, more controlled studies are needed to avoid design limitations and flaws and to control for day to day variables which can weaken the conclusions and support for research. Nevertheless, the current investigation has provided some guidelines for future research in this area.

BIBLIOGRAPHY

Allain, C.C., Poon, L.S., Cicely, S.G.C., Richmond, W., & Paul, C.F. (1974). Enzymatic determination of total serum cholesterol. Clinical Chemistry, 20(4), 470-475.

Anderson, J.W. (1993). Diets, lipids and cardiovascular disease in women. Journal of the American College of Nutrition, 12(4), 433-437.

Anderson, J.W., Floore, T.L., Geil, P.B., & Spencer, D.B. (1990). Initial serum cholesterol as a predictor of response to an AHA diet. Clinical Research, 38(2), 744A.

Anderson, J.W., Garrity, T.F., Wood, C.L., Whitis, S.E., Smith, B.M., & Oeltgen, P.R. (1992). Prospective, randomized controlled comparison of the effects of low-fat and low-fat plus high-fiber diets on serum lipid concentrations. American Journal of Clinical Nutrition, 56, 887-894.

Anderson, J.W., Hamilton, C.C., & Brinkman-Kaplan, V. (1992). Benefits and risks of an intensive very-low-calorie diet program for severe obesity. American Journal of Clinical Nutrition, 87, 6-15.

Anderson, J.W., Hamilton, C.C., Crown-Weber, E., Riddlemoser, M., & Gustafson, N.J. (1992). Safety and effectiveness of a multidisciplinary very-low-calorie diet program for selected obese individuals. Journal of the American Dietetic Association, 91, 1582-1584.

Anderson, K.N., Anderson, L.E., & Glanze, W. D. (Eds.). (1994). Mosby's medical, nursing, allied health dictionary (4th ed.). St. Louis: Mosby-Year book, Inc.

Barnard, R.J. (1991, July). Effects of life-style modification on serum lipids. Archives of Internal Medicine, 151, 1389-1394.

Bell, L.P., Hectorn, K.J., Reynolds, H., & Hunninghake, D.B. (1990). Cholesterol-lowering effects of soluble-fiber cereals as part of a prudent diet for patients with mild to moderate hypercholesterolemia. American Journal of Clinical Nutrition, 52, 1020-1026.

Biometrics one-on-one Manual. (1993). Virginia: People Karch International.

Boyd, N.F., Cousins, M., Beaton, M., Kriukov, V., Lockwood, G., & Tritcher, D. (1990). Quantitative changes in dietary fat intake and serum cholesterol in women: Results from a randomized, controlled trial. American Journal of Clinical Nutrition, 52, 470-476.

Boyden, T.W., Pamenter, R.W., Going, S.B., Lohman, T.G., Hall, M.C., Houtkooper, L.B., Bunt, J.C., Ritenbaugh, C., & Aickin, M. (1993, January). Resistance exercise training is associated with decreases in serum low-density lipoprotein cholesterol levels in premenopausal women. Archives of Internal Medicine, 153, 97-100.

Blessing, D., Stone, M., Byrd, R., Wilson, D., Rozenek, R., Pushparani, D., & Lipner, H. (1987). Blood lipid and hormonal changes from jogging and weight training of meddle-aged men. Journal of Applied Sports Science Research, 1(2), 25-29.

Brownell, K.D., & Stunkard, A.J. (1981). Differential changes in plasma high-density lipoprotein-cholesterol levels in obese men and women during weight reduction. Archives of Internal Medicine, 141, 1142-1146.

Clevidence, B.A., Judd, J.T., Schatzkin, A., Muesing, R.A., Campbell, W.S., Brown, C.C., & Taylor, P.R. (1992). Plama lipid and lipoprotein concentrations of men consuming a low-fat, high-fiber diet. American Journal of Clinical Nutrition, 55, 689-694.

Dalen, J.E. (1991). National cholesterol education program report of the expert panel on population strategies for blood cholesterol reduction: Executive Summary. Archives of Internal Medicine, 151, 1071-1084.

Dattilo, A.M. & Kris-Etherton, P.M. (1992). Effects of weight reduction on blood lipids and lipiproteins: A meta-analysis. American Journal of Clinical Nutrition, 56, 320-328.

Dengel, D.R., Hagberg, J.M., Coon, P.J., Drinkwater, D.T., Goldberg, A.P. (1994). Comparable effects of diet and exercise on body composition and lipoproteins in older men. Medicine and Science in Sports and Exercise, 26(11), 1307-1315.

Denke, M.A. & Grundy, S.M. (1994). Individual responses to a cholesterol-lowering diet in 50 men with moderate hypercholesterolimia. Archives of Internal Medicine, 154, 317-325.

Durak, E.P., Jovanovic-Peterson, L., & Peterson, C.M. (1990). Randomized crossover study of effect of resistance training on glycemic control, muscular strength, and cholesterol in type I diabetic men. Diabetes Care, 13, 1039-1043.

Fang, C-L., Sherman, M., Crouse, S.F., & Tolson, H. (1988). Exercise modality and selected coronary risk factors: A multivariate approach. Medicine and Science and Sports and Exercise, 20(5), 455-462.

Farrell, P.A., Maksud, M.G., Pollock, M.L., Foster, C., Anholm, J., Hare, J., & Leon, A.S. (1982). A comparison of plasma cholesterol, triglycerides, and high density lipoprotein-cholesterol in speed skaters, weightlifters,and non-athletes. European Journal of Applied Physiology, 48, 77-82.

Fripp, R.R. & Hodgson, J.L. (1987). Effects of resistive training on plasma lipid and lipoprotein levels in male adolescents. Journal of Pediatrics, 111, 926-931.

Ginsberg, H.N., Barr, S.L., Gilbert, A., Karmally, W., Deckelbaum R., Kaplan, K., Ramakrishnan, R., Holleran, S., & Dell, R.B. (1990). Reduction of plasma cholesterol levels in normal men on an american heart association step 1diet or a step 1 diet with added monounsaturated fat. New England Journal of Medicine, 322(9), 574-579.

Goldberg, A.P. (1989). Aerobic and resistive exercise modify risk factors for coronary heart disease. Medicine and Science in Sports and Exercise, 21(6), 669-674.

Goldberg, L., Elliot, D.L., Schutz, R.W., & Kloster, F.E. (1984). Changes in lipid and lipoprotein levels after weight training. Journal of the American Medical Association, 252(4), 504-506.

Golding, L. A., Myers, C.R., & Sinning, W.E. (Eds.). (1989). Y's way to physical fitness: The complete guide to fitness testing and instruction. Champaign, Illnois: Human Kinetics Publishers, Inc.

Howley, E.T., Franks, B.D. (Eds.). (1986). Health fitness instructor's handbook. Champaign, Illnois: Human Kinetics Publishers, Inc.

Hurley, B.F. (1989). Effects of resistive training on lipoprotein-lipid profiles: A comparison to aerobic exercise training. Medicine and Science in Sports and Exercise, 21(6), 689-693.

Hurley, B.F., Hagberg, J.M., Goldberg, A.P., Seals, D.R., Ehsani, A.A., Brennan, R.E., & Holloszy, J.O. (1988). Resistive training can reduce coronary risk factors without altering VO2max or percent body fat. Medicine and Science in Sports and Exercise, 20(2), 150-154.

Hurley, B.F., Hagberg, J.M., Seals, D.R., Ehsani, A.A., Goldberg, A.P., Holloszy, J.O. (1987). Glucose tolerance and lipid-lipoprotein levels in meddle-aged powerlifters. Clinical Physiology, 7, 11-19.

Hurley, B.F., Kokkinos, P.F. (1987). Effects of weight training on risk factors for coronary artery disease. Sports Medicine, 4, 231-238.

Hurley, B.F., Seals, D.R., Hagberg, J.M., Goldberg, A.C., Ostrove, S.M., Holloszy, J.O., Wiest, W.G., Goldberg, A.P. (1984). High-density-lipoprotein cholesterol in bodybuilders v powerlifters. Journal of the American Medical Association, 252(4), 507-513.

Jenkins, D.J.A., Wolever, M.S., Venketeshwer-Rao, A., Hegele, R.A., Mitchell, S.J., Ransom, T.P.P., Boctor, D.L., Spadafora, P.J., Jenkins, A.L., Mehling, C., Katzman-Relle, L., Connelly, P.W., Story, J.A., Furumoto, E.J., Corey, P., & Wursch, P. (1993). Effect on blood lipids of very high intakes of fiber in diets low in saturated fat and cholesterol. New England Journal of Medicine, 329(1), 21-26.

Johnson, C.C., Stone, M.H., Lopez-S, A., Herbert, J.A., Kilgore, L.T., & Byrd, R.J. (1982). Diet and exercise in middle-aged men. Journal of the American Dietetic Association, 81, 695-701.

Kasim, S.E., Martino, S., Kim, P-N., Khilnani, S., Boomer, A., Depper, J., Reading, B.A., & Heilbrun, L.K. (1993). Dietary and anthropometric determinants of plasma lipoproteins during a long-term low-fat diet in healthy women. American Journal of Clinical Nutrition, 57, 146-153.

Kenney, W.L., Humphrey, R.H., & Bryant, C.X. (Eds.). (1995). American college of sports medicine's guidelines for exercise testing and prescription (5th ed.). Philadelphia: Lea & Febiger.

Kimura, Y., Kubota, M., & Yamazaki, S. (1989). Effects of different modes of conditioning on cardiovascular function, body composition, and serum lipids profile. Medicine and Science in Sports and Medicine, 21(2), S75.

Kohl, H.W., Gordon, N.F., Scott, C.B., Vaandrager, H., & Blair, S.N. (1992). Musculoskeletal strength and serum lipid levels in men and women. Medicine and Science in Sports and Exercise, 24,(10), 1080-1087.

Koivisto, V.A., Nikkila, E.A., Taskinen, M-R., & Yki-Jarvinen, H. (1984). Glucose tolerance, plasma lipoproteins and tissue lipoprotein lipase activities in body builders. European Journal of Applied Physiology, 53,253-259.

Kokkinos, P.F., Hurley, B.F. (1990). Strength training and lipoprotein-lipid profiles: A critical analysis and recommendations for further study. Sports Medicine, 9(5), 266-272.

Kokkinos, P.F., Hurley, B.F., Smutok, M.A., Farmer, C., Reece, C., Shulman, R., Charabogos, C., Patterson, J., Goldberg, A.P. (1989). Lipoprotein-lipid profiles and post-heparin lipase activities are unaltered from strength training. Medicine and Science in Sports and Exercise, 2(2), S116.

Kokkinos, P.F., Hurley, B.F., Smutok, M.A., Farmer, C., Reece, C., Shulman, R., Charabogos, C., Patterson, J., Will, J., DeVane-Bell, J., & Goldberg, A.P. (1991). Strength training does not improve lipoprotein-lipid profiles in men at risk for CHD. Medicine and Science in Sports and Exercise, 23(10), 1134-1139.

Kokkinos, P.F., Hurley, B.F., Vaccaro, P., Patterson, J.C., Gardner, L.B., Ostrove, S.M., & Goldberg, A.P. (1988). Effects of low- and high-repetition resistive training on lipoprotein-lipid profiles. Medicine and Science in Sports and Exercise, 20(1), 50-54.

Kris-Etherton, P.M. & Krummel, D. (1993, September). Role of nutrition in the prevention and treatment of coronary heart disease in women. Journal of the American Dietetic Association, 93(9), 987-993.

Manning, J.M., Dooly-Manning, C.R., White, K., Kampa, I., Silas, S., Kesselhaut, M., & Ruoff, M. (1991). Effects of a resistive training program on lipoprotein-lipid levels in obese women. Medicine and Science in Sports and Exercise, 23(11), 1222-1226.

Mensink, R.P., Katan, M.B. (1992). Effect of dietary fatty acids on serum lipids and lipoproteins. A meta-analysis of 27 trials. Journal of the American Dietetic Association, 92, 434-440.

Morgan, D.H., Cruise, R.J., Girardin, B.W., Lutz-Schneider, V., Morgan, D.H., & Qi, W.M. (1986). HDL-C concentrations in weight-trained, endurance-trained, and sedentary females. The Physician and Sportsmedicine, 14(3), 166-179.

Muscling in on cholesterol. (1994). University of California at Berkeley Wellness Letter, 11(1), 2-3.

Nieman, D.C., Haig, J.L., Fairchild, K.S., DeGuia, E.D., Dizon, G.P., & Register, U.D. (1990). Reducing-diet and exercise-training effects on serum lipids and lipoproteins in mildly obese women. American Journal of Clinical Nutrition, 52, 640-645.

Pate, P.P. & Burgess, M.L. (1993). Terminology in exercise physiology. In J. L. Durstine, A. C. King, P. L. Painter, J. L. Roitman, L. D. Zwiren, & W.L. Kenney (Eds.), American college of sports medicine resource manual for guidelines for exercise testing and prescription (2nd ed.). Philadelphia: Lea & Febiger.

Poehlman, E.T., Gardner, A.W., Ades, P.A., Katzman-Rooks, S.M., Montgomery, S.M., Atlas, O.K., Ballor, D.L., & Tyzber, R.S. (1992). Resting energy metabolism and cardiovascular disease risk in resistance-trained and aerobically trained males. Metabolism, 41(12), 1351-1360.

Pronk, N.P. (1993). Short term effects of exercise on plasma lipids and lipoproteins in humans. Sports Medicine, 16(6), 431-448.

Ramsay, L.E., Yeo, W.W., & Jackson, P.R. (1991). Dietary reduction of serum cholesterol concentration: Time to think again. British Journal of Medicine, 303, 953-957.

Sacks, F.M., Handysides, G.H., Marais, G.E., Rosner, B., Kass, E.H. (1986). Effects of a low-fat diet on plasma lipoprotein levels. Archives of Internal Medicine, 146, 1573-1577.

Sandstrom, B., Marckmann, P., & Bindslev, N. (1992). An eight-month controlled study of a low-fat high-fiber diet: Effects on blood lipids and blood pressure in healthy young subjects. European Journal of Clinical Nutrition, 46, 95-100.

Seim, H.C., Holtmeier, K.B. (1992). Effects of a six-week, low-fat diet on serum cholesterol, body weight, and body measurements. Family Practice Reasearch Journal, 12(4), 411-419.

Smutok, M.A., Reece, C. Kokkinos, P.F., Farmer, C., Dawson, P., Shulman, R., DeVane-Bell, J., Patterson, J., Charabogos, C., Goldberg, A.P., & Hurley, B.F. (1993). Aerobic versus strength training for risk factor intervention in middle-aged men at high risk for coronary heart disease. Metabolism, 42(2), 177-184.

Snyder, S. (1990). Comparison of cholesterol-lowering regimens. Clinical Pharmacology, 42(3), 761-768.

Stevenson, D.W., Darga, L.L., Spafford, T.R., Ahmad, N., & Lucas, C.P. (1988). Variable effects of weight loss on serum lipids and lipoproteins in obese patients. International Journal of Obesity, 12, 495-402.

Stone, M.H., Blessing, D., Byrd, R., Tew, J., Boatwright, D., Karr, B., Johnson, C., Lopez, A., Johnson, R., & Carter, D. (1982, October/November). Physiological effects of a short term resistive training program on middle-age untrained men. National Strength and Conditioning Association Journal, 16-21.

Stone, M.H., Fleck, S.J., Triplett, N.T., & Kraemer, W.J. (1991). Health- and performance-related potential of resistance training. Sports Medicine, 11(4), 210-231.

Stone, S.H., & Wilson, G.D. (1985, January). Resistive training and selected effects. Medical Clinics of North America, 69(1), 109-122.

Trinder, P. (1969). Determination of glucose in blood using glucose oxidase with an alternative oxygen acceptor. Annuals of Clinical Biochemistry, 6, 24-27.

Tyroler. H.A. (1989). Overview of clinical trials of cholesterol lowering in relationship to epidemiologig studies. American Journal of Medicine, 87, 14-9s.

Ullrich, I.H., Reid, C.M., & Yeater, R.A. (1987). Increased HDL-cholesterol levels with a weight lifting program. Southern Medical Journal, 80(3), 328-331.

Verrill, D., Shoup, E., McElveen, G., Witt, K., & Bergery, D. (1992). Resistive exercise training in cardiac patients: Recommendations. Sports Medicine, 13(3), 171-193.

Wallace, M.B., Moffatt, R.J., Haymes, E.M., & Green, N.R. (1991). Acute effects of resistance exercise on parameters of lipoprotein metabolism. Medicine and Science in Sports and Exercise, 23(2), 199-204.

Weltman, A., Janney, C., Rians, C.B., Strand, K., & Katch, F.I. (1987). The effects of hydraulic-resistance strength training on serum lipid levels in prepubertal boys. American Journal of Diseased Children, 141, 777-780.

Williams, P.T., Stefanick, M.L., Vranizan, K.M., & Wood, P.D. (1994). The effects of weight loss by exercise or by dieting on plasma high-density lipoprotein (HDL) levels in men with low, intermediate, and normal-to-high HDL at baseline. Metabolism, 43(7), 917-924.

Wood, P.D., Stefanick, M.L., Dreon, D.M., Frey-Hewitt, B., Garay, S.C., Williams, P.T., Superko, H.R., Fortmann, S.P., Albers, J.J., Vranizan, K.M., Ellsworth, N.M., Terry, R.B., & Haskell, W.L. (1988, November). Changes in plasma lipids and lipoproteins in overweight men during weight loss through dieting as compared with exercise. The New England Journal of Medicine, 319(18), 1173-1179.

Wood, P.D., Stefanick, M.L., Williams, P.T., & Haskell, W.L. (1991). The effects on plasma lipoproteins of a prudent weight-reducing diet, with or without exercise, in overweight men and women. New England Journal of Medicine, 325(7), 461-466.

Yki-Jarvinen, H., Koivisto, V.A., & Nikkila, E.A. (1984). Glucose tolerance, plasma lipoproteins and tissue lipoprotein lipase activities in body builders. European Jounal of Applied Physiology, 53, 253-259.

VITA

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Candidate for the Degree of

Master of Science

Thesis: EFFECTS OF A COMBINED RESISTANCE TRAINING AND DIET PROGRAM ON LIPOPROTEIN AND LIPID PROFILES: A COMPARISON BETWEEN MALES AND FEMALES

Major Field: Health, Physical Education, and Leisure

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OKLAHOMA STATE UNIVERSITY
INSTITUTIONAL REVIEW BOARD
HUMAN SUBJECTS REVIEW

Date: 03-11-96 **IRB#:** ED-96-086

Proposal Title: EFFECTS OF A COMBINED RESISTANCE TRAINING AND DIET PROGRAM ON LIPOPROTEIN AND LIPID PROFILES: A COMPARISON BETWEEN MALES AND FEMALES

Principal Investigator(s): Frank A. Kulling, Diane Spivey

Reviewed and Processed as: Exempt

Approval Status Recommended by Reviewer(s): Approved

ALL APPROVALS MAY BE SUBJECT TO REVIEW BY FULL INSTITUTIONAL REVIEW BOARD AT NEXT MEETING.
APPROVAL STATUS PERIOD VALID FOR ONE CALENDAR YEAR AFTER WHICH A CONTINUATION OR RENEWAL REQUEST IS REQUIRED TO BE SUBMITTED FOR BOARD APPROVAL.
ANY MODIFICATIONS TO APPROVED PROJECT MUST ALSO BE SUBMITTED FOR APPROVAL.

Comments, Modifications/Conditions for Approval or Reasons for Deferral or Disapproval are as follows:

Signature: John H. Wyatt

Chair of Institutional Review Board

Date: March 19, 1996