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The Effect of Caloric Restriction and Exercise on Plasma Lipid and Lipoprotein Concentration

Jeffrey J. Zachwieja
Eastern Illinois University

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THE EFFECT OF CALORIC RESTRICTION AND EXERCISE ON PLASMA LIPID AND LIPOPROTEIN CONCENTRATION

(TITLE)

BY

JEFFREY J. ZACHWIEJA

THESIS

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ABSTRACT

THE EFFECT OF CALORIC RESTRICTION AND EXERCISE ON PLASMA LIPID AND LIPOPROTEIN CONCENTRATION

BY JEFFREY J. ZACHWIEJA

The independent and combined effects of exercise training and caloric restriction on the plasma concentration of lipid and lipoprotein was studied. Thirty-two female volunteers were assigned to one of three treatment groups; exercise only, diet only or diet plus exercise. The exercise only group participated in a walk/jog program three days per week. For 30 to 45 minutes these individuals either walked or jogged at a intensity which elicited a heart rate that was 70% of the max heart rate reserve. The diet only group participated in a behavior modification weight loss program which restricted caloric intake to 1200 kcals per day. The combined diet and exercise group participated in both of the above programs. Only 38% of the of the initial 32 subjects fully completed the 12 week study. The diet and diet plus exercise treatments elicited the greatest ($p < 0.05$) amount of weight loss. Weight loss for these two groups averaged 7.3 and 4.4 kgs respectively. The amount of fat weight in these two groups was also significantly reduced ($p < 0.05$), while no change was observed in lean body mass for any of the treatment groups. Maximal oxygen consumption (L/min) was

only increased in the exercise only group. This group demonstrated an 11% increase from pretreatment values. However, when oxygen consumption was expressed per kilogram of body weight, significant ($p < 0.05$) increases in maximal oxygen consumption were observe in all three treatment groups. The effects of exercise, caloric restriction, or a combination of these two treatments on the blood lipid profile were minimal. No changes in the plasma concentration of cholesterol, triglyceride, high density lipoprotein, and low density lipoprotein from pretreatment values were observed. In conclusion, exercise (70% max HR reserve) and weight loss (1200 kcal/day) independently and combined produced changes in body weight and maximal aerobic capacity. However, these three treatments were not sufficient enough to produce changes in the blood lipid profiles of 12 female volunteers.

DEDICATION

This paper is dedicated to my parents. Your love and support throughout my educational career has made this all possible. This paper is also dedicated to my grandfather, Walter Zachwieja Sr., who was not fortunate enough to benefit from the knowledge gained in the last 20 years. May he rest in peace.

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CHAPTER 1

INTRODUCTION

Evidence from epidemiological study has revealed that elevated total blood cholesterol levels are positively associated with an increased risk of developing coronary artery disease (CAD) (4,82,126). Of recent interest has been the study of the cholesterol content in the various lipoprotein carriers. Clinical studies have demonstrated that elevated levels of high density lipoprotein cholesterol (HDL-C) are independently and inversely related to the development of CAD (41,91,106). In addition, elevated levels of low density lipoprotein cholesterol (LDL-C) have been strongly related to the development of CAD (68).

Exercise training prescribed for healthy non-symptomatic (for CAD) individuals seems to have the greatest effect on the fractions of cholesterol contained in HDL and LDL. Specific attention is usually devoted to HDL-C because of its inverse relation to CAD. Although cross-sectional studies surveying physically active individuals routinely demonstrate elevated HDL-C levels when compared to inactive controls (1,30,44,46,48,52,93,118), longitudinal studies have yielded conflicting results. Some studies have demonstrated an

increase in HDL-C in response to exercise training (53,71,100,107,111,132,135); whereas, others have indicated no change (15,34,59,125,128) or a decrease (2) in HDL levels.

Experimental designs devoted to studying the response of HDL to exercise training can be disturbed by a number of different factors. Gender and race of the subjects, alcohol intake, and smoking habits, have all been previously identified to have independent effects on HDL-C concentration (123). Weight loss with associated changes in body composition may be one of the most common confounding factors when studying the effects of a particular training program on lipoprotein cholesterol content. Attempts have been made to study the effect of exercise training on the blood lipid profile by administering conventional weight loss treatments (16,134,139), as well as maintaining body weight during a specified exercise training period (95). While the intentions of these particular methods are good, it is hard to make comparisons among these studies because of the single group design. A more accurate understanding of the relative contributions of weight loss and/or exercise to the alterations in the blood lipid profile may be best obtained through multiple group designs. In this manner, separate and combined treatments of exercise and diet can be compared to each other as well as a control group.

Purpose of the Study

The purpose of this study was to use a multiple group design in an attempt to add to the current knowledge concerning the effects of weight loss and exercise on serum lipid values including; plasma cholesterol, triglycerides, HDL cholesterol, and LDL cholesterol.

Null Hypothesis

There is no difference in changes of absolute body weight and blood lipid values including; plasma cholesterol, triglycerides, HDL cholesterol, and LDL cholesterol between three treatment groups in response to various manipulations of diet and exercise.

Limitations

The following limitations have been placed upon this study. Subject adherence to the study was quite low, as only 38% of the original subject pool actually completed all phases of the study. Secondly, blood lipid measurements were performed in a clinical laboratory. Under these conditions, rarely are attempts made to control for intrasubject variation. Lastly, body composition measurements were obtained via the skinfold technique as equipment for hydrostatic weighing was not available to the investigator.

Definitions

Apoprotein

The protein moiety of a molecule or complex, as of a lipoprotein.

Biopsy

The surgical removal and examination of tissue from the body.

Catabolism

Any destructive process by which complex substances are converted into more simple compounds.

Cholesterol

A pearly fatlike steroid alcohol. It is synthesized by the liver and absorbed from the diet. As well as being a precursor for the synthesis of steroid hormones and bile acids it constitutes a large part of the atheroma found in arteries.

Cofactor

An element or principle with which another must unite in order to function.

Electrophoresis

The movement of charged particles suspended in a liquid on various media under the influence of an applied electrical field.

Endogenous

Produced within an organism.

Enzyme

A protein capable of greatly accelerating a chemical reaction of a substance for which it is specified.

Exogenous

Originating outside of an organism.

Esterify

To combine with an alcohol with elimination of a molecule of water, forming an ester.

Heparin

A substance present in many tissues, especially the liver and lungs, which has powerful anticoagulent properties.

High Density Liopoprotein (HDL)

A plasma lipoprotein containing high levels of protein, little triglyceride, moderate levels of phospholipid, and relatively low levels of cholesterol.

Hydrolysis

A cleavage of a compound by the addition of water.

Insulin

A major fuel regulating hormone secreted into the blood in response to a rise in the concentration of blood glucose.

Intermediate Density Lipoprotein (IDL)

A plasma lipoprotein containing a low percentage of triglyceride, high levels of cholesterol, and moderate levels of phospholipid and protein.

Low Density Lipoprotein (LDL)

A plasma lipoprotein containing a low percentage of triglyceride, high levels of cholesterol, and moderate levels of phospholipid and protein.

Moiety

A part of, as in a portion of a molecule.

Phospholipid

Any lipid that contains phosphorus. They are the major lipids in the cell membrane.

Postabsorbtive

Pertaining to a fasted state.

Receptor

A molecule on the surface or within the cell that recognizes and binds with specific molecules.

Triglyceride

A neutral fat that is usually the storage form of lipid in animals.

Ultracentrifugation

Subjection of a material to an exceedingly high centrifugal force which will separate and sediment the molecules of a substance.

Very Low Density Lipoprotein (VLDL)

A plasma lipoprotein containing high concentrations of triglyceride, moderate concentrations of phospholipid and cholesterol, and little protein.

CHAPTER 2

REVIEW OF RELATED LITERATURE

The relationship between cardiovascular disease risk factors and the occurrence of this disease has been well established (104). Therefore, in order to decrease the risk of developing coronary artery disease (CAD), it is important to keep such factors under control. Our increasing knowledge concerning the disease process has led to widespread awareness of the pathophysiological effects of cholesterol contained within certain lipoprotein carrier molecules. Even though information pertaining to these lipoproteins in terms of their synthesis, catabolism, and regulatory mechanisms is in its infancy, the knowledge we have obtained thus far has allowed us to begin effective management of the CAD process.

The major purpose of this chapter is to review the effects of various forms of exercise and weight loss on the plasma concentration of lipids and lipoproteins. Much of the information to be presented has been obtained from studies which have utilized healthy (free from CAD) male and female subjects between the ages of 20 and 60. Specific attention will be directed toward changes in plasma cholesterol, triglyceride, and the cholesterol contained within high and

low density lipoprotein. The mechanisms by which these changes occur and the effects that various enviormental and habitual factors have on these changes will also be discussed.

In order fully understand the effects that chronic exercise training can have on plasma lipid and lipoprotein concentration, a basic understanding of lipoprotein metabolism is needed. Therefore, the first section of this paper has been dedicated to outlining the basic mechanisms behind lipoprotein formation, breakdown, and use. The information presented in this section was obtained from an essay prepared by Howard A. Eder, 1983 (29).

Lipoprotein Metabolism

The major function of a plasma lipoprotein is to transport lipids so that they may be used for cellular metabolism. Plasma lipoproteins have been divided into 5 major categories based on either their electrophoretic motility or their gravitational density in response to ultracentifugation. The categories are as follows; chylomicrons, very low density lipoprotein (VLDL), intermediate density lipoprotein (IDL), low density lipoprotein (LDL), and high density lipoprotein (HDL) each of which contain various proportions of cholesterol (both free

and esterified), triglyceride, phospholipid, and apoprotein.

A number of different apoproteins have been identified and classified in terms of their protein constituents. The apoproteins add to the stability of the lipoprotein molecule as well as possessing physiological value. Physiologically, they are involved in receptor mediated processes and act as cofactors for enzyme mediated lipoprotein metabolism. Table I lists the major apoproteins which have been identified.

A schematic diagram of lipoprotein metabolism is presented in figure I. Triglyceride and cholesterol can either be exogenously induced into the body through dietary intake, or endogenously produced by the body. Dietary triglyceride and cholesterol enter the blood stream in the form of chylomicrons. The major constituents of these chylomicron molecules are, triglyceride, esterified cholesterol, and apoprotein. Since chylomicrons are to large to pass through the capillary endothelium they are broken down partially in circulation. The enzyme lipoprotein lipase (LPL) hydrolyzes the triglyceride contained within the chylomicron molecule resulting in the passage of free fatty acid (FFA) into near by extra-hepatic cells. The extra hepatic cells will use the FFA for either energy production (muscle cells) or energy storage (adipose tissue). After the reduction of lipid content, the remaining chylomicron

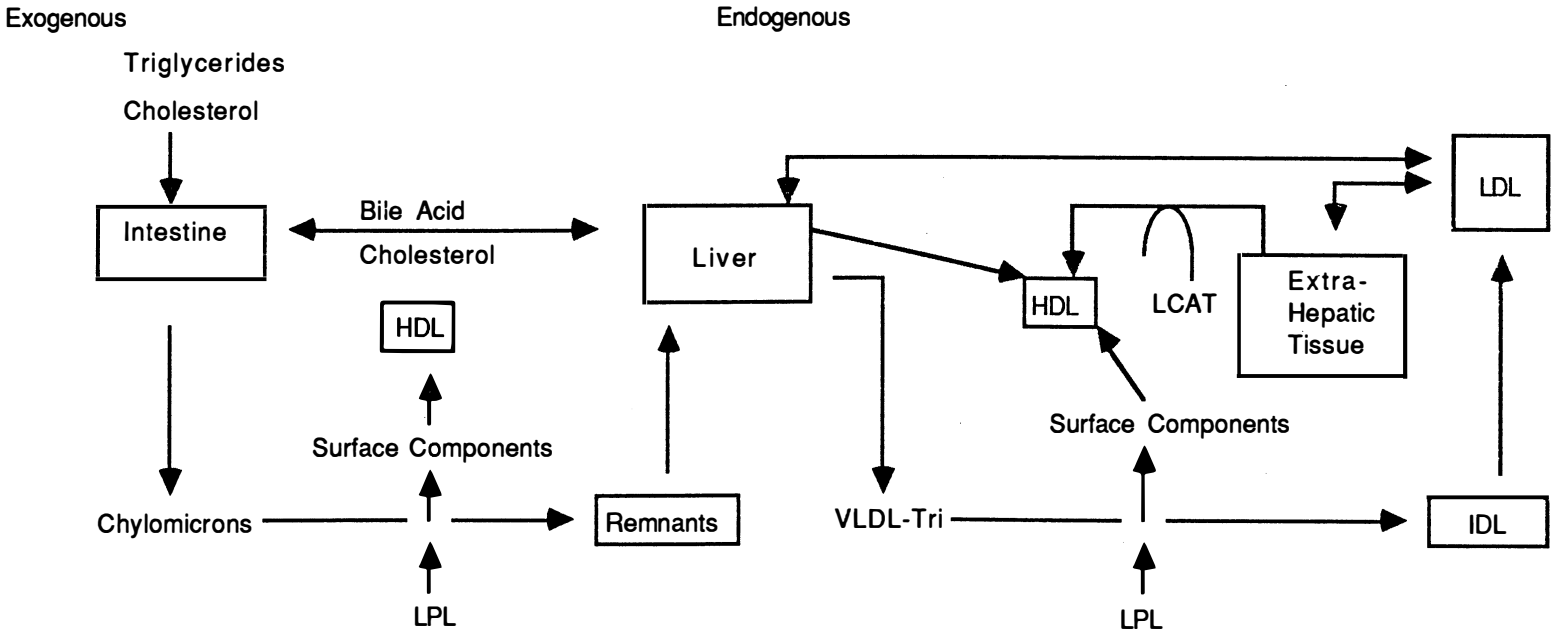
TABLE I

Appoproteins and Their Function.
Adapted from Dufaux et al. (28)

Appoprotein	Lipoprotein Found In	Function
A-I	HDL	Cofactor of LCAT.
A-II	HDL	Binding of phospholipid.
B	Chylomicrons, LDL, VLDL	Binding to cell receptor.
C-I	Chylomicrons	Cofactor of adipose tissue LPL.
C-II	Chylomicrons	Cofactor of adipose tissue LPL.
C-III	Chylomicrons, VLDL	Unknown.
E	VLDL	Binding to cell receptor.

FIGURE I

Pathways of Lipoprotein Metabolism
Adapted From Eder (28)



molecule (referred to as a chylomicron remnant) has decreased in size considerably. This size reduction has left the chylomicron remnant with an excess of surface components (mainly apoprotein). These excess surface components are eventually released and later incorporated into HDL. The chylomicron remnant is then removed from circulation by the liver. In the liver the chylomicron remnants are broken down into amino acid and free cholesterol. Some of the free cholesterol is re-esterified and released as a constituent of VLDL while other portions are converted to bile acids. Free cholesterol and bile acids have two fates. Either they are transported to the intestine where they are eventually reabsorbed or they are lost through the feces.

Hepatic VLDL contains triglyceride, cholesterol, and apoprotein. As with the chylomicrons, VLDL is reduced of its lipid content by LPL, converting it into smaller IDL molecules. IDL is either cleared from the blood stream by the liver or it is rapidly converted to LDL. During each step of this reduction process excess surface components are released and incorporated into HDL.

In normal individuals LDL transports a large majority (about 2/3) of the total plasm cholesterol. LDL is most often removed from the circulation by extrahepatic tissue through a receptor mediated process. In particular, cells

undergoing rapid growth or repair are targeted. The cholesterol content of these cells is largely determined by uptake, production, and removal rates. A large percentage of the removal rate is dependent upon HDL mass and activity. HDL activity is regulated by the enzyme Lecinthin-cholesterol acyltransferase (LCAT). LCAT facilitates the transfer of cholesterol from the extrahepatic tissue onto HDL. Cholesterol rich HDL then makes its way back to the liver. The protective action of HDL is based on this process. Increases in LCAT activity as a result of exercise training may be one reason why increased HDL mass is observed in endurance athletes. This and other possible mechanisms will be discussed in greater detail later in this chapter.

Exercise and Blood Lipids

Because of the strong relationship between high levels of plasma cholesterol and CAD (4,82,126), and the observation that more physically active individuals had lower plasma cholesterol levels (56,88), early studies concentrated on changes in plasma cholesterol and triglyceride in response to endurance training. Even though during this time methods for quantification of the lipoprotein transport molecules were available (37), it was not until the mid 1970's that these techniques were incorporated into this type of study. Today, the significance of these lipoprotein transport molecules to

the management of the CAD process is well established. We now know that little or no change in plasma cholesterol concentration as a result of endurance exercise can be accompanied by significant changes in the distribution of this lipid among its lipoprotein carriers (49).

Plasma Cholesterol

Over the years, cross-sectional study has been a popular method for determining the relationship between exercise training and plasma concentrations of cholesterol. When compared to inactive individuals, those who perform endurance exercise have exhibited similar or only slightly lower cholesterol values (1,44,46,62,78,87). However, there are some reports which have demonstrated cholesterol values for endurance trained individuals as being significantly lower than those for age matched controls (11,27,47,137). Individuals who primarily take part in weight training have also been studied. These reports have indicated that weight trained individuals have similar (9,17) or slightly higher (19,30) cholesterol values than inactive controls. This occurrence may be related to similarities in aerobic fitness, body fat percentage, and diet between weight lifters and the controls (9).

To better define a possible relationship between plasma cholesterol values and aerobic fitness, some investigators

have reported on the association between exercise test performance and plasma cholesterol values. Shane (113) studied 877 USAF flying personnel and obtained a significant inverse correlation (from zero correlation) between maximal oxygen consumption and plasma cholesterol values. Similarly, Cooper et al. (20) using fitness classifications (ie. very poor, good, excellent) derived from treadmill performance times reported that of the nearly 3000 men tested, those achieving "excellent" status had significantly lower cholesterol values than those classified as "very poor". Although these results may demonstrate a relationship between exercise capacity and cholesterol values, there is some evidence to suggest that such a relationship does not exist. Through the Tecumseh community health study (Michigan), Montoye et al. (92) performed treadmill tests on 910 males and females and found no relationship between maximal oxygen consumption (L/min) and total cholesterol values when age, weight, and skinfolds were removed by multiple regression. Additional studies controlled in the same manner have reported similar findings (96,116,138).

Because of the very nature of cross-sectional design, it is difficult to infer direct cause and effect relationships between exercise performance and/or participation and plasma cholesterol values. Longitudinal designs which can account

for the methodological problems of cross-section design have been used (12,34,73,58,89,101,105,112). However, information obtained from these studies has also produced conflicting results. In some cases the effects of endurance training (11-24 weeks) has produced a reduction in cholesterol values (73,101), but more often than not reports of no change in response to an exercise training program have been elicited (12,34,58,89,105,112). Differences in length of training program, adherence, intensity of exercise, and mode of activity may have all contributed to the conflicting results. Consideration must also be given to cholesterol assay methodology, initial blood lipid values, weight loss, and the time interval between the last training period and the drawing of blood. These circumstances are also of particular importance when considering changes in lipoprotein fractions. This topic will receive additional attention later in this chapter.

Plasma Triglycerides

Cross-sectional studies investigating the relationship between endurance training and plasma triglyceride values have also been completed. These studies have found endurance training to be consistently associated with lower plasma triglyceride values (44,46,78,87,137). This relationship has been documented for males as well as females, with females

exhibiting a more pronounced decrement (137). In addition, Wood et al. (137) have also reported that endurance training can prevent the age related increase in plasma triglyceride values. In this particular study, cross-sectional data obtained from female runners over a 20 year span demonstrated an age related increase in plasma triglycerides of about 12 mg%. On the other hand, the control group over the same 20 year span demonstrated about a 20 mg% increase in plasma triglycerides. Many believe that the triglyceride values exhibited by endurance athletes are representative of the type of activities performed (44,46,78). However, others believe that these findings may be totally independent of the physical activity performed and are more related to the increased leanness of such individuals (121).

Studies incorporating an exercise training program have been able to consistently lower plasma triglyceride concentration from pre-training values (12,58,71,111). However, throughout the course of these investigations this information has not been produced without reservation. Some interesting questions concerning the acute and chronic effects of exercise on plasma triglyceride concentration have arisen. Bananno and Lies (12) observed the effects of a 12 week exercise program (3 x per week 40-50 minutes) on plasma lipid concentration (cholesterol and triglycerides). After the completion of the exercise program two blood samples were

drawn, one 24 hours and another 96 hours after the last bout of exercise. The 24 hour blood sample revealed a 23% decrease in plasma triglycerides. This value was significantly lower than the pre-exercise value. The 96 hour blood sample revealed a return of the plasma triglyceride content back to the baseline value. A similar relationship was demonstrated by Holloszy et al. (58). Follow-up testing after a 6 month training period revealed a 42% increase from post exercise plasma triglyceride values after 5-6 days of inactivity. Results from both of these studies suggest that the acute effect of the last bout of exercise must be a strong consideration when trying to determine a relationship between plasma triglyceride values and the effects of chronic exercise.

The acute effect of exercise on plasma triglyceride values is highly dependent upon the pre-exercise values for this blood lipid. Initially low levels of plasma triglyceride will not show a significant decrease in response to a moderate bout of exercise (25,26). In order for a change to occur, the exercise session must be substantially longer in duration (> 6 hrs) and high in intensity (> 85 % VO_2 max)(18,70). In contrast to this observation, hypertriglyceridemic individuals demonstrate an immediate lowering in plasma triglyceride concentration in response to

a moderate bout of exercise. This occurs whether or not caloric balance is maintained or increased during the exercise period (43). However, within 48 to 72 hours of the last bout of exercise, the triglyceride levels of these individuals will begin to return to baseline values. This may explain the variation in the data presented by both Bonnonano and Lies (12) and Holloszy et al. (58) as the subjects in both of these studies had baseline triglyceride values in excess of 200 mg%. In conclusion, it seems as though initial levels of plasma triglyceride and the duration and intensity of the exercise play a big role in the acute and/or chronic effects of exercise on plasma triglyceride.

Low Density Lipioprotein Cholesterol

LDL is the major transport particle for cholesterol in circulation. In fact, strong correlations have been obtained between total cholesterol and plasma levels of LDL-C (29). The effects of endurance exercise on LDL-C are quite often of small magnitude, although several investigations have produced evidence in support of significantly decreased levels in endurance trained individuals. Such cross-sectional studies have indicated both male and female endurance athletes, in addition to recreational joggers, have lower LDL-C levels when matched with sedentary controls (47,87,136,137). As is typical for this type of study, equal

or higher values for LDL-C concentrations have also been reported for endurance performers when compared with age matched controls (1,93,97). Cross-sectional studies utilizing weight trained athletes have also been performed. No differences in LDL-C concentrations between these athletes and sedentary controls have been obtained (8,30).

Longitudinal training studies which have lasted for 8 to 15 weeks and have incorporated endurance type exercise 3 to 4 times per week at 60 to 85% maximum heart rate have been able to produce decreases in plasma LDL-C levels (15,63,100). The magnitude of these decreases has been around 8 to 12%, with the amount of exercise performed being negatively correlated with plasma LDL-C levels (135). As was noted with plasma cholesterol values, if LDL-C is elevated beyond what is considered normal, decreases in response to a training program are more likely to occur.

Most of the previous information presented on plasma LDL-C has come from studies which have emphasized changes in other lipid parameters. There are very few, if any studies which have set out to determine the effects of exercise on plasma LDL-C exclusively. It would seem likely that more studies would be concerned with changes in plasma LDL-C in light of its strong relationship with CAD manifestation. However, the increased responsiveness of HDL-C to exercise

makes this lipoprotein fraction a more worthwhile pursuit. Ultimately, it seems that the distribution of cholesterol about LDL-C and HDL-C is what determines CAD potential. Evidence from cross-sectional and longitudinal study has suggested that exercise is more likely to produce significant changes in HDL-C while LDL-C remains constant. The opposite effect rarely occurs. Although an increase in HDL-C with a concomitant decrease in LDL-C would be optimal, the very mechanisms whereby exercise produces its effect decreases the likelihood that a significant combination effect can occur. These statements are based on evidence obtained from individuals who exhibit neither hypertriglyceridemia or hypercholesterolemia.

Plasma High Density Lipoprotein Cholesterol

One of the proposed metabolic functions of plasma HDL-C is to transport cholesterol away from the peripheral tissues and back to the liver where it can be catabolised. In light of this action, the concept that HDL might act as a protective agent against CAD has gained support from epidemiological studies which have determined an inverse relationship between HDL-C and the occurrence of CAD (41,91,106). In addition, the concept that exercise may produce a substantial increase in HDL-C has recieved considerable attention.

Data from cross-sectional study have revealed that

individuals who take part in endurance type activities often possess higher levels of HDL-C than those individuals who do not. For example, male endurance athletes (1,44,46,48,52), female endurance athletes (93), female swimmers (118), and male speed skaters (30) have all been demonstrated to have elevated HDL-C levels. Although some studies comparing endurance trained individuals to sedentary controls have reported no difference between these to groups (19,50), initial low levels of plasma cholesterol may have contributed to these findings. Clarkson et al. (19) reported that the endurance trained individuals in their study had plasma cholesterol levels that were significantly lower than those of the control. As a result, even though the endurance athletes and the controls did not differ in absolute HDL-C levels, a greater percentage of the total plasma cholesterol was carried by HDL in the endurance performers. Cross-sectional data collected on those individuals who primarily take part in weight training have differed little from data collected on sedentary controls (30,94). Biochemical explanations for the similarities in HDL-C levels between these two groups are not readily apparent. Differences in nutrition, muscle adaptation to this specific form of exercise, and circulating levels of hormones may all be possible explanations.

Some investigators have reported a dose response relationship between HDL-C and the distance run per week (40,48,78,107,108,131). Unfortunately, these studies have not been able to come up with consistent data regarding an upper and lower threshold of activity which "turns on" or "turns off" a change in plasma HDL-C. However, all have reported positive correlations (ie. +.26 - +.55) between HDL-C and miles run per week.

A large number of studies have concentrated on the effects of regular endurance exercise on plasma HDL-C. As a result it has been increasingly difficult to deduce the amount of activity necessary to produce significant changes. Evidence concerning the intensity needed to produce changes in plasma HDL-C has suggested that endurance exercise at 75% to 85% max HR is sufficient (15,111). The amount and type of activity needed although a more popular inquiry is not well defined. Combinations of walking, jogging, and calisthenics 3 to 4 times per week for 8 to 17 weeks has produced significant increases in HDL-C concentration (80). Some believe that running or jogging 8 to 12 miles per week is needed if significant strides are to be made toward increasing HDL-C concentrations (108,131,135). However, higher thresholds have also been reported ranging from 35 to 45 miles per week (78,107). Part of the reason why Rotkis et

al. (107) and Lehtonen et al. (78) have reported such high threshold points is the type of subject used. In both of these studies females with initially high levels of HDL-C (ie > 55 mg%) were used. This point is particularly well illustrated in the study of Goodyear et al. (40). In this study female subjects who were already running 20-25 miles per week increased there weekly running mileage by 4 miles per week for 8 weeks. At the end of the eight week treatment period the subjects were averaging 62 miles per week. An increase in HDL of 29% (from 57 mg/dl to 76 mg/dl) was observed from pre-program values. These data suggest that substantial increases in HDL can be obtained in a relatively short time period, however, the type of training which was required to produce this change may be totally unrealistic for the average health conscious individual.

Little information has been obtained on the possibility on a upper limit threshold for "turning off" increases in HDL-C concentrations. Hartung et al. (48) studied two groups of marathon runners who were physiologically similar. Whereas both groups had significantly higher HDL-C values than the average population, one group had a higher HDL-C concentration than the other. When comparing intensity and duration of the typical workout patterns specific to each group, it was observed that the lower HDL-C group ran more miles at a somewhat faster pace. Hartung et al. (48)

concluded that there is for some individuals a threshold of intensity where little change occurs.

Well controlled longitudinal training studies have been able to produce significant changes in HDL-C concentrations from pre-exercise values (53,71,100,107,111,132,135). However, these types of intervention programs have not escaped conflicting results. Some studies have not demonstrated a change in HDL-C levels (15,34,59,125,128), while others have even shown a decrease in HDL-C levels (2). Exercise training studies using a weight lifting regime are beginning to take a different angle. Traditionally, weight lifters who utilize high resistance/low repetition workouts have been studied. Results have indicated that HDL-C levels between these athletes and sedentary controls are not significantly different (8,9,30). Recently, some investigators have been interested in the effects of a weight lifting program utilizing low resistance/high repetition. Results from these studies have indicated that this type of resistance training is capable of producing HDL-C levels comparable to those of endurance athletes (38,60,61,66). Further work in this area is necessary in order to determine whether these results are a chronic effect of the exercise, or whether these changes are attributable to the last acute bout of exercise.

A large number of studies have looked at the effects of exercise on the plasma lipoproteins. Previously mentioned studies (1,15,30,34,44,46,48,52,53,59,71,93,100,107,111,128) only represent a small percentage of the vast amount of work that has been completed. Inconsistencies in the reports of these studies may stem from several factors, some of which have been previously discussed. Initial levels of blood lipids, HDL-C in particular, can greatly effect the exercise response relationship. Baseline levels of low cholesterol and high HDL-C have shown little change in response to exercise of increasing intensity and duration (34,59,93,128). Other factors known to influence HDL-C concentrations are laboratory procedures, seasonal changes, weight loss, and personal habits including cigarette smoking, alcohol consumption, and oral contraceptive use (120,123). Failure to factor out or control these variables when determining the effects of exercise on plasma of HDL-C increases the difficulty of data interpretation.

Mechanisms of Exercise Induced HDL-C and Triglyceride Change

Endurance training improves ones capacity to do work. Increased cardiac output, and a more efficient utilization of oxygen by the muscle cells, resulting in an increased ability to utilize fat for energy production, are often cited

as the main factors associated with the adaptation to exercise (57). Along with an increased ability to utilize fatty acid for the production of energy (57), endurance trained athletes have also been characterized as having different lipid profiles when compared to age and weight matched controls (49). Two of the most striking features are decreased levels of plasma triglyceride and elevations in HDL-C (49). Decreased levels of adiposity often accompany a highly trained state. At least in part, this observation has been suggested to account for the decreased triglyceride content and increased HDL-C concentrations in endurance athletes (121). In fact, significant negative correlations have been obtained between percent body fat and HDL-C, as well as positive corelations between triglyceride content and percent body fat (93). However, in light of the acute changes in lipoprotein concentration which can occur during moderate and intense bouts of exercise (54), adaptations in lipoprotein metabolism must also be considered. Although the biochemical changes in lipoprotein metabolism which come about as a result of an exercise training program are not well understood, there is sufficient evidence to suggest that the activities of Lipoprotein lipase (LPL) and Lecithin-cholesterol acyltransferase (LCAT) play an important role (28). Decreased production of hepatic triglyceride in

response to exercise training should also be considered (114), however it will not be discussed in depth during this review.

Enzymatic Regulation of Decreased Triglyceride Content

In his essay, Edner (29) states that LPL is predominately found on the surface of endothilial cells. In particular, LPL is heavily concentrated on the endothilial cells which comprise the walls of capillaries closest in contact with skeletal muscle and adipose tissue. The main metabolic function of LPL is to initiate the catabolism of the triglyceride found in VLDL and chylomicrons. When LPL comes in contact with this triglyceride, FFA and glycerol are released into circulation from these molecules. LPL activity can be measured in humans through adipose and skeletal muscle tissue biopsys (49). LPL activity can also be measured in plasma after injection of heparin (49). Heparin initiates the release of bound LPL from the capillary tissue.

When compared with sedentary controls, male and female endurance athletes have exhibited increased amounts of LPL activity in both skeletal muscle and adipose tissue. Nikkila et al. (97) reported that male distance runners had 1.7 x higher skeletal muscle LPL activity than non-runners. Similar results were also obtained for the female runners. Adipose tissue LPL activity was also higher in both male and female runners. LPL activity in the runners was 2.5 and 1.6

x greater than control LPL activity respectively. It was concluded that endurance training was associated with the adaptive increase of LPL in both adipose and skeletal muscle tissue.

Distribution of skeletal muscle LPL seems to be highly dependent upon the fiber type of a particular muscle. It has been demonstrated that slow twitch muscle fibers possess a greater percentage of LPL than do fast twitch muscle fibers (13,81). However, studies have indicated that exercise training can produce increased LPL activity in both fast twitch and slow twitch muscle fibers (13). Since the early 1970's, there has been an interest in determining the fiber type of elite athletes in order to better understand the mechanisms behind their tremendous success. Generally, endurance athletes have been shown to possess a greater percentage of slow twitch muscle fibers (22), whereas the speed or power athlete possesses a greater percentage of fast twitch muscle fibers (21). This type of information could possibly complicate the results of cross-sectional studies which have commonly shown an increased skeletal muscle LPL activity in endurance trained athletes.

Despite the selection bias which might occur when utilizing a cross-sectional design, exercise training studies have supported the idea that skeletal muscle and adipose

tissue LPL activity can be increased in both men and rats (13,100). Peltonen et al. (100) demonstrated that after a 15 week training program post heparin plasma LPL activity increased by 33%, while adipose tissue LPL increased by 56%. A large majority of the change occurred within the first several weeks of the training period. This finding may suggest that certain tissue LPL possess the ability to adapt quickly to acute bouts of exercise.

If LPL is vulnerable to acute bouts of exercise, then the extent to which lipoprotein bound triglyceride can contribute to the fat oxidation process during this activity should be considered. It has been shown that shorter bouts of submaximal exercise and even marathons have not significantly lowered the concentration of plasma triglyceride in participating athletes (26,54,62). The only exception has been extreme bouts of exercise lasting in excess of 6 hours (18,70). Likewise, LPL activity has only been shown to increase significantly in response to long duration exercise (69,83). Some believe that the stability of triglyceride during the shorter bouts of exercise is do to an increase in the use of plasma triglyceride with a concomitant increase in the production of triglyceride by the liver. Although it takes a substantially longer time for muscle LPL to respond to an acute bout of exercise, adipose

tissue LPL activity may increase after the first hour of exercise (83). This could increase the plasma concentration of FFA and thus an increase in the uptake of FFA by the liver. As a result, there would be an increase in the production of liopoprotein bound triglyceride and no net effect would be observed on the plasma triglyceride concentration (49). However, it seems unlikely that there is actually an increased lipoprotein bound triglyceride production during exercise. It has been shown in rats that the uptake of FFA by the liver does not increase during exercise beyond the clearance which is observed during rest (67). Therefore, it seems fair to assume that lipoprotein bound triglyceride plays a very minor role in the process of energy production during exercise bouts lasting up to 6 hours. Under these conditions most of the energy which is supplied by the oxidation of fats comes in the form of circulating FFA. A small percentage is also derived from the muscle's store of triglyceride (51).

During extended bouts of exercise the muscle not only becomes depleted of its stores glycogen (10), but also experiences decreases in its storage of triglyceride (33). If repeated bouts of exercise are performed the triglyceride content of the muscle can remain low. Although the consequences of this reaction are not as severe as those

observed in glycogen depletion, the need to replenish the triglyceride store of the muscle is necessary. This may be one reason why endurance athletes are observed as having lower resting plasma triglyceride values than matched sedentary controls (44,46,78,87,137). It has been hypothesized that the increase in skeletal muscle LPL activity in the trained state enables the muscle to more readily uptake circulating triglyceride in order to replenish its stores. This increased clearance due to the increased uptake by the muscle has been previously reported (85). Additional support has come from studies which have demonstrated an increased glycerol-3-phosphate synthesis in skeletal muscle and adipose tissue of endurance trained rats (5). The adaptation of adipose tissue LPL also seems beneficial in that it helps replenish the fat stores (which have also been used for oxidation in the muscle) by breaking down circulating plasma triglyceride (5).

The mechanisms whereby exercise training increases the activity of LPL have not been well defined. Although there are several hypotheses, this review will only be concerned with the roles of insulin and caloric balance.

Endurance trained individuals have been known to exhibit a blunted insulin response to a glucose load (77,110,111). This has the net effect of maintaining and in some instances increasing glucose tolerance while also decreasing the

insulin response to similar glucose loads. It is not known whether this is a result of an increased number of insulin receptors in the muscle and adipose tissue, or to an increased responsiveness of the receptors that are already there (74). However, there seems to be a substantial amount of evidence in support of an increased sensitivity due to an increase in the number of insulin receptors. An increased number of insulin binding sites on the cell membranes of erethrocytes and adipose tissue in the human (77) and animal (23) models produce evidence in support of this theory. However, it is not clear whether changes in insulin binding to blood cells and adipose tissue parallel insulin binding to muscle tissue. This change, if present, may be one explanation for the increase observed in LPL activity of endurance trained individuals. It has been previously demonstrated that LPL in the adipose tissue is very sensitive to insulin (35). Endurance training results in tremendous increases in caloric expenditure. Therefore, in order to maintain body weight endurance athletes have to increase their caloric intake substantially. This alone may be enough to increase the activity of LPL. Jacobs et al. (65) have shown that a carbohydrate rich diet increased muscle LPL activity by 82% after these muscles had been significantly depleted of their glycogen stores as a result of an intense

exercise bout.

Enzymatic Regulation of HDL-C Increases

Earlier in this chapter, it was reported that increased levels of HDL-C have been demonstrated in those individuals who were describes as very physically active (1,44,46,48,52,93), and in those who had successfully completed an exercise training program (53,71,100,107,111). Although the precise mechanism(s) whereby HDL-C increases in response to endurance training has/have not been determined, there is sufficient evidence mounting in support of increases in the activity of certain key enzymes involved in the metabolism of lipoproteins.

Previous studies have reported a close relationship between the metabolism of plasma triglycerides and HDL-C (2,41,91). This has been supported through the strong negative correlations which have been obtained between this blood lipid and lipoprotein fraction (41,91). The primary role of LPL is to hydrolyze the triglyceride found in chylomicrons and VLDL. The surface components (apoprotein, phospholipid) which are eventually released as a consequence of this reaction are later incorporated into HDL. It would seem logical that the increases in the activity of LPL which so often accompanies the trained state (13,97,100) would increase the breakdown rate of chylomicron and VLDL molecules

thus leading to an increase in HDL mass. A significant positive correlation between adipose tissue LPL activity and HDL-C concentration has been suggested as evidence in support of this hypothesis (97).

A second enzymatic explanation lies within the activity of LCAT. LCAT is mainly involved in the transfer of unesterified cholesterol onto the HDL molecule. There is both direct and indirect evidence to support increased LCAT activity in the trained state. Endurance trained individuals (86) and those taking part in a controlled endurance training program (84) have been known to exhibit increased LCAT activity. In the study of Marniemi et al. (86) LCAT was correlated highly with increases in HDL-C. A large majority (≈ 90%) of the protein contained within HDL is made up of apoprotein AI and AII, with AI being identified as an essential activator of the LCAT enzyme (75). Since apoprotein AI has also been shown to increase in response to an endurance training program (71,122), it is possible that the activity of LCAT is increased through this occurrence.

Weight Reduction and Lipoproteins

Individuals who demonstrate an increased capacity to perform exercise exhibit rather distinguishable characteristics. Among the most common are; elevated maximal

oxygen consumption, increased cardiac output, a decreased body fat percentage, and a favorable blood lipid profile including increased HDL-C, and decreases in plasma triglycerides, cholesterol, and LDL-C. Much of the information previously presented in this review has concentrated on the blood lipid profile, in particular, how it is effected by an exercising lifestyle, and the mechanisms behind these changes. Although there is a substantial amount of evidence to suggest that a favorable blood lipid profile is common in endurance athletes because of the exercise they perform (1,44,46,48,52,53,71,93,100,107,111,118,132,135), decreased percentages of body fat and body weight have been known to effect the blood lipid profile in a similar fashion (121).

Moderate obesity has been generally associated with decreased levels of HDL-C, and increased levels of triglyceride and LDL-C (43,98), predisposing these individuals to an increased risk of developing CAD. It is commonly accepted that weight reduction will eliminate such unfavorable blood lipid profiles. However, the studies completed on this topic have yielded conflicting results. Although weight reduction has consistently demonstrated decreased levels of triglyceride (98), the effects of weight loss on LDL-C and HDL-C have been to increase (76,119), decrease (6,16,127,129), and to produce no change

(45,134,139).

Effects of Weight Loss on Plasma Triglyceride

Moderate obesity is known to cause an elevation in plasma triglyceride slightly above what would be considered normal. The proposed mechanism behind this reaction is an increase in the production of VLVL triglyceride. Moderately obese individuals tend to exhibit a peripheral tissue resistance to insulin. As a result, increases in insulin secretion are needed to promote similar glucose uptakes. This excess insulin is believed to act on the liver in such a way that the liver begins to increase its production of VLDL triglyceride (98). In some cases of gross obesity, triglyceride values can be extremely high as a result of the above mechanism (79,98). In other situations, plasma triglyceride values can be extremely high as a result of genetic abnormalities leading to an increased production and decreased clearance of plasma triglyceride (29). Upon the implementation of a weight reduction program plasma levels of triglyceride begin to reduce and remain in a reduced state as long as the weight reduction is maintained (98). This may be the result of a decreased peripheral tissue resistance to insulin resulting in a decreased VLDL triglyceride production. An increase in triglyceride clearance due to a greater activity of LPL is also a possibility, however this

occurrence only seems likely when the reduction in body weight has been stabilized (139).

Effects of Weight Loss on LDL-C

Increased levels of LDL-C in the obese state are generally associated with the increased VLDL production. Several previous studies determining the relationship between LDL-C and weight loss have reported conflicting results (16,76,98,127,134,139). For example, Wolf and Grundy. (134) reported that there was no change in LDL-C levels after a weight reduction program consisting of reduced caloric intake in a mixed (males and females) group of subjects. Bronwell et al. (16) reported that both men and women involved in a moderate weight reduction program exhibited significant decreases in LDL-C. Olefesky et al. (98) reported a 21% decrease in total cholesterol in 36 patients after a weight reduction program, however no reports were made on any of the lipoprotein fractions. Finally, Zimmerman et al. (139) reported an initial decrease in plasma LDL-C during periods of weight reduction, however after cessation of weight loss, and during a period of body weight maintenance, LDL-C levels rebounded to initial values.

Why have these conflicting results been obtained? First, Not every study controlled dietary intake in the same manner. Some reduced caloric intake as well as altering the

percent of carbohydrates, fats, protein, and cholesterol ingested (76,134). Others just limited the caloric intake (16,127). In some of the cited studies, blood samples may have been drawn when the subjects were still in a negative caloric balance. This effect of active weight loss on LDL-C levels is best represented in those studies which have presented information on the time course of LDL-C change in response to a caloric restriction program (134,139). In addition, the weight reductions in some of the previously mentioned studies may not have been great enough to produce any rebound effect. It does seem very likely that these two situations are important considerations, especially when dealing with those individuals who are only moderately obese. As Wolf and Grundy (134) explained, weight reduction may not alter LDL-C to any significant degree. Rather, weight reduction may produce a decreased flux (input to and clearance from peripheral tissues) of LDL-C thus lessening the risk for CAD development.

Effects of Weight Loss on HDL-C

Because of HDL-C's negative association with the development of CAD, much emphasis is placed on its alterations through various forms of therapy. Exercise training and weight reduction are two of the most common interventions, with the former being previously discussed. Initially it was reported that weight reduction decreased the

levels of HDL-C (16,127), but more recent evidence tends to indicate increases in HDL-C can be achieved through weight loss (109,119,134,139).

Because HDL-C has been negatively correlated with triglycerides, it is logical to assume that during periods of weight reduction HDL-C will increase as plasma triglyceride decreases. However, several studies have not been able to show this relationship. Thompson et al. (127) measured lipoprotein values in 15 obese women before and after a 10 week weight reduction program. Post-treatment values of HDL-C were determined to be significantly lower than the baseline value by 10%. Similarly, Beneke and Timson (6) observed a 24% decrease in plasma HDL-C values after a 12 week behavior/nutritional weight loss treatment. Both of these studies accounted for the reduction in HDL-C through significant decreases in total plasma cholesterol of 10 and 12% respectively.

Increased levels of HDL-C as a result of weight reduction are not commonly reported during the acute phases of weight loss. Rather, increases in HDL-C are more commonly observed when a stabilized reduced weight has been obtained (109,119,134,139). Zimmerman et al. (139) observed that after 7 weeks of dieting, HDL-C levels did not change significantly in 7 obese women. However, 3 months after the

weight reduction program, a significant increase in HDL-C from 39.2 mg% to 46.1 mg% was observed while these individuals were maintaining a reduced body weight. Similar results have been reported by Wolf and Grundy (134) and Schwartz et al. (109). Information concerning the effect of a stabilized reduced body weight may account for some of the discrepancies of earlier studies. In fact, a closer look at the data of Thompson et al. (127) reveals a rebounding of plasma HDL-C levels slightly beyond the pre-treatment value 8 months after the post-treatment blood sample. Body weight at this time was not significantly different from the post-treatment value indicating a stability in the weight reduction obtained some months ago.

Although there seems to be strong support for an increase in HDL-C concentration and a decrease in triglyceride concentration after obtaining a reduced stabilized body weight, these results should still be interpreted with caution. Many variables which are not always easy to control can effect plasma levels of lipids and lipoproteins. Strict dietary control in regards to the percent distribution of carbohydrate, fat, and protein is not always feasible. Furthermore, personal habits such as alcoholic intake and activity levels are not always reported accurately by the subject population. Lastly, the initial

values of both body weight and the plasma concentration of lipids and lipoproteins will effect the outcome to some degree. Higher initial values tend to respond more positively, while lower initial values most often remain low. Nonetheless, there seems to be some positive evidence in support of caloric restriction as a practical intervention therapy which could theoretically be beneficial to the prevention of CAD.

Summary

The blood lipid profile is a valuable tool for those individuals involved in the management of the CAD process. Research has indicated that the distribution of cholesterol among the different lipoprotein fractions carries great predictive value. In particular, the cholesterol contained within HDL has been negatively associated with CAD, while the cholesterol contained within LDL has been positively associated with CAD. Attempts have been made through diet and exercise to increase HDL-C and concomitantly decrease LDL-C. Although the results have not always been conclusive, the independent effects of diet and exercise have been to increase HDL-C while either slightly reducing or maintaining LDL-C levels. Future efforts should be directed toward studying a possible summation effect of diet and exercise on

these two plasma lipoproteins.

CHAPTER 3

METHODOLOGY

Descriptions of the subjects, treatment groups, evaluation techniques used, and the methods of data analysis are all contained within this chapter.

Subjects

Thirty-two females ranging in age from 18 to 35 years volunteered to participate in this study. In order to qualify, each had to be at least 15 pounds overweight according to the Fogarty height weight chart (Appendix A) (14). Prior to participation in the program, the subjects were required to complete a health history questionnaire (Appendix B). The information obtained from the questionnaire revealed that these individuals led a sedentary lifestyle and were free from heart disease, diabetes, hypertension, and hyperlipidemia. Although none were currently smoking, two had quit within the last year, and no one reported a consumption of more than 2 alcoholic beverages in one week. To further document the initial condition of these individuals heart rhythm strips and resting blood pressures were obtained and evaluated by qualified personnel. Each person was informed in the nature of the study, the

possible risks involved, and signed an informed consent (Appendix C). The initial physical characteristics of the subjects are presented in table II.

Treatment Groups

Prior to the initial testing, the subjects were randomly assigned to one of three groups; diet only, exercise only, or diet and exercise. The diet treatment consisted of participation in a behavior modification weight loss program which limited caloric intake to 1200 calories per day. Subjects in this group were not encouraged to perform regular physical activity. The exercise group performed 30 to 45 minutes of aerobic activity (walking/jogging) 3 times per week and maintained normal dietary intake. The diet and exercise group was active in both the exercise sessions and the diet program. The effects of these treatment programs were evaluated after 12 weeks of participation.

Methods of Evaluation

Evaluations of weight, height, body composition, aerobic capacity, and blood lipid content (total cholesterol, triglycerides, HDL-C, and LDL-C) were performed prior to, and after the completion of the 12 week treatment period. On the day of each aerobic capacity test, the subjects reported to the Human Performance Lab in the postabsorptive state. Upon

TABLE II

Physical Characteristics of the Subjects (N = 32).

Variable Measured	Mean	SE	Range
Age (yrs)	30.6	± 0.65	18.0 - 35.0
Weight (kgs)	79.7	± 2.8	56.8 - 119.8
Height (cm)	164.9	± 1.2	153.0 - 182.0
BMI ($wt/ht^2 * 1000$)	2.92	± 0.10	2.24 - 4.23
Percent Body Fat	31.8	± 0.93	20.2 - 40.1
Pounds Overweight	47.2	± 5.1	15.0- 136.0

arrival, the subjects were seated in a comfortable position for several minutes. Next, a resting blood pressure and heart rate were obtained. The resting blood pressure was determined by using a standard sphygmomanometer, and resting heart rate was obtained via palpation of the radial artery. Total body weight was measured to the nearest 0.01 kg while standing on a platform balance wearing only shorts and a t-shirt. Height was determined to the nearest 0.1 cm using a standard ruler device.

Maximal aerobic capacity (defined as peak VO_2) was assessed by measuring oxygen consumption during a treadmill (Quinton 65) graded exercise test. The treadmill protocol that was used was specifically designed for the population tested based on the recommendations of Beneke et al. (7). This treadmill protocol is displayed in figure II. Oxygen consumption was measured using an open circuit spirometry system. Expired air was sampled every 15 seconds for fractional components of oxygen (Applied Electrochemistry analyzer S-3A) and carbon dioxide (Applied Electrochemistry analyzer CD-3A). Ventilation was measured by a Rayfield gasmeter (model RAM 9200) which was modified to provide an Apple IIe computer with information for each 0.1 liters of air ventilated. The Apple computer also obtained information of O_2 and CO_2 fractions and by way of a Prowriter Dot Matrix

FIGURE II

Graded Exercise Test Protocol

Stage #	Time (mins)	Speed	% Grade
1	0 - 3	3.5 mph	0
2	3 - 5	4.0 mph	0
3	5 - 7	5.5 mph	0
4	7 - 9	6.0 mph	0
5	9 - 11	6.0 mph	4
6	11 - 13	6.0 mph	6
7	13 - 15	6.0 mph	8

printer displayed values for VE, VO2 (relative and absolute), CO2, and RER. Heart rate was monitored by the use of a single lead system (electrodes placed in a modified V1 and V5 position) connected to a radiotelemetry unit. A numerical value for heart rate and a rhythm strip were displayed by a difibulator unit (Physiocontrol, Lifepack 6).

Body composition was estimated using the skinfold technique. Lange skinfold calipers were used to determine skinfold thickness and the equation of Jackson et al. (64) was used to determine body density. The Siri (115) equation was used to calculate percent body fat, and lean body mass was obtained by subtracting fat weight from total body weight.

On the morning of the blood sampling the subjects reported to the lab in a fasted (12-16 hrs), well rested state. While in a seated position two 5 ml venous blood samples were drawn from the antecubital vein by a registered Phlabotomist. Determinations for total cholesterol, triglycerides, and the fractions of cholesterol contained in HDL and LDL were made by Illinois Medical Laboratories INC., Charleston, Illinois. The procedures of the Lipids Research Clinic were followed. In addition to the specified blood lipid analysis, a Hemogram was also performed. This information allowed for the correction of blood lipid values based on fluctuations in plasma volume.

Aerobic Conditioning Program

The aerobic conditioning program was conducted in conjunction with the Eastern Illinois University Adult Fitness program and involved walking and/or jogging 3 times per week for 30 to 45 minutes per session. The mode of activity was selected on the basis of a predetermined exercise intensity. Each subject was instructed to exercise at a heart rate which was 70% of the heart rate reserve (0.70 x (max HR - rest HR) + rest HR) obtained during the aerobic capacity test. Intensity was monitored by taking occasional 10 second heart rates at either the radial or carotid artery. Typical warm up and cool down procedures were encouraged, and all subjects were required to record each exercise session in terms of duration (minutes), and intensity in a centralized log book (Appendix D). A record of additional activities performed (ie. cycling, aerobic dance) was also kept.

Caloric Restriction Program

Subjects undergoing diet treatment were required to meet one night a week for 45 to 60 minutes. Although the diet group and the diet and exercise group followed the same diet program, these two groups met on separate evenings to promote group unity. Each of the meetings was conducted by a group leader and had a specific focus. The first part of the

meeting was set aside for weigh-in and general discussion. The second part of the meeting was dedicated to evaluating progress and offering encouragement and positive reinforcement. The third and final part of the meeting was set aside for presentation of a behavioral/nutritional lesson. These lessons were obtained from a diet program which has been successfully used in research and as a community based weight loss program. Eating Slim (99), is a behavior modification program which emphasizes eating as a chain of events including planning, purchasing, storing, preparing, serving, eating, and post-meal cleanup. Lesson presentations centered around taking steps to modify this chain of events in order to prevent overeating.

During the week, the subjects kept daily records of the type, amount, and caloric value of the food consumed. Caloric intake was to be restricted to 1200 calories per day. Each subject was provided with a calorie counter booklet and weekly food record sheets (Appendix E) so that accurate information could be kept. The weekly food record sheets were routinely reviewed so that adherence to the dietary program could be evaluated. Records of weekly weight loss were also kept on a weight graph (Appendix F). This allowed the subjects to observe their progress toward a specific goal.

Statistical Analysis

Mean differences pertaining to the measurements made before and after the treatment period were treated for statistical significance with a one way analysis of varience. When a significant F ratio was observed, specific group differences were determined by way of a Fisher's PLSD post hoc test. The alpha level for statistical significance was set at 0.05.

CHAPTER 4

ANALYSIS OF THE DATA

The purpose of this study was to examine the effects of programs of diet and/or exercise on certain health related variables. Aerobic fitness, body composition, and the plasma concentration of cholesterol, triglyceride, HDL-C, and LDL-C were evaluated before and after a 12 week treatment period. Results pertaining to the various treatment effects and a discussion of why certain parameters were changed and others remained unchanged will be presented in this chapter.

Subject Adherence

For various reasons (ie. time conflicts, illness, family responsibilities), several of the initial volunteers had to withdraw from the study prior to the third meeting time of their specified group. Although this situation created an unfavorable reduction in group size, several of the dropouts agreed to become part of a control group. The control group was instructed not to alter dietary intake and to refrain from any sort of regular physical activity. Before the completion of the 12 week treatment period participation had

declined to 38% of the original subject pool, leaving 12 individuals who fully completed their specified treatment and all of the physiological testing. The initial physical characteristics of these subjects plus the control group (N = 4) are presented in table III.

Attendance for those individuals who were involved in the exercise only program was excellent. On the average, these subjects attended 90% of the supervised exercise sessions and trained at a mean intensity of 77% of the max heart rate reserve. In contrast, attendance for the exercise portion of the diet plus exercise treatment was not as good. These individuals attended 68% of the supervised exercise sessions and trained at a mean intensity of 67% of the max heart rate reserve. Official records of attendance for the diet treatment group meetings were not kept. However, if a subjective evaluation was to be given, attendance by the diet and diet plus exercise groups would be classified as "good" and "fair" respectively.

Body Weight and Body Composition Change

Absolute and percentage changes in body weight due to the diet (D) and diet plus exercise (DE) treatments are presented in figures III and IV. In an attempt to maintain a positive morale within the exercise (E) and control (C)

TABLE III

Physical Characteristics of the Subjects (N = 16)

Variable Measured	Mean	SE	Range
Age (yrs)	31.4	± 0.57	27 - 35
Weight (kgs)	77.2	± 4.1	56.8 119.8
Height (cm)	164.1	± 1.5	153.0 - 175.3
BMI (wt/ht^2 * 1000)	2.89	± 0.15	2.24 - 4.23
Percent Body Fat	31.6	± 1.4	20.2 - 40.1
Pounds Overweight	47.0	± 8.4	18.0 - 135.0

FIGURE III

Asolute changes in body weight for the diet and Diet + Exercise treatment groups.

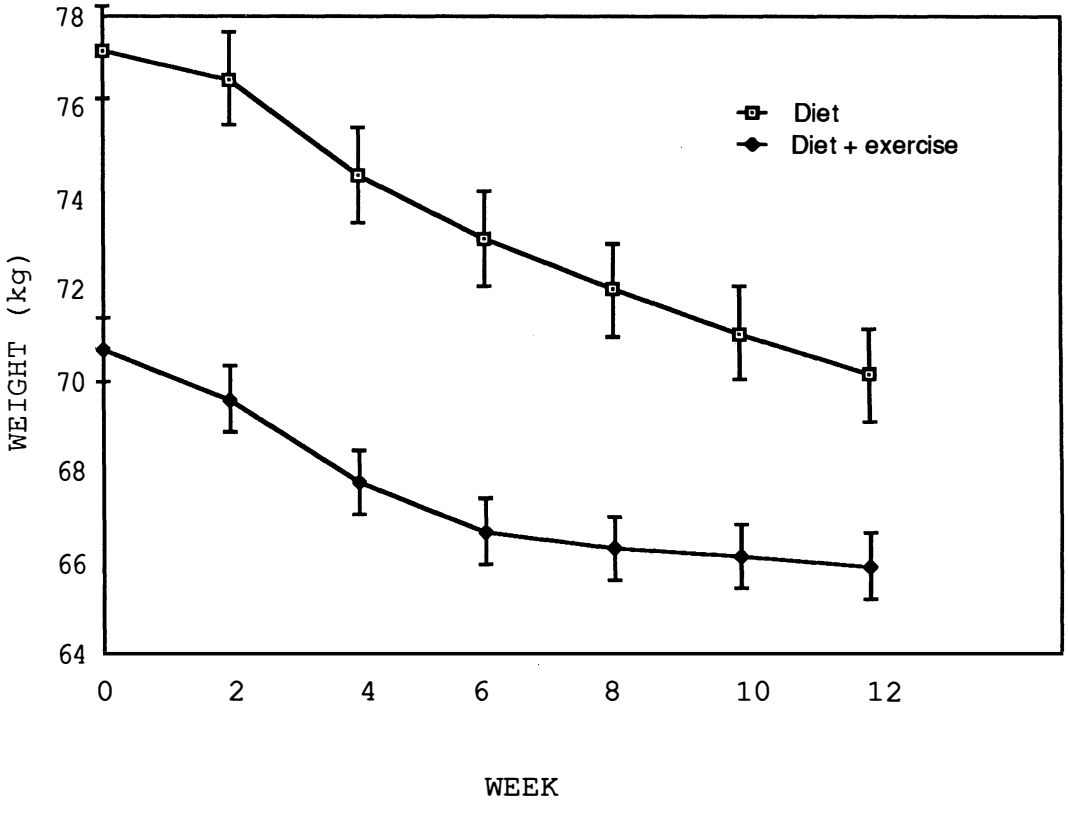
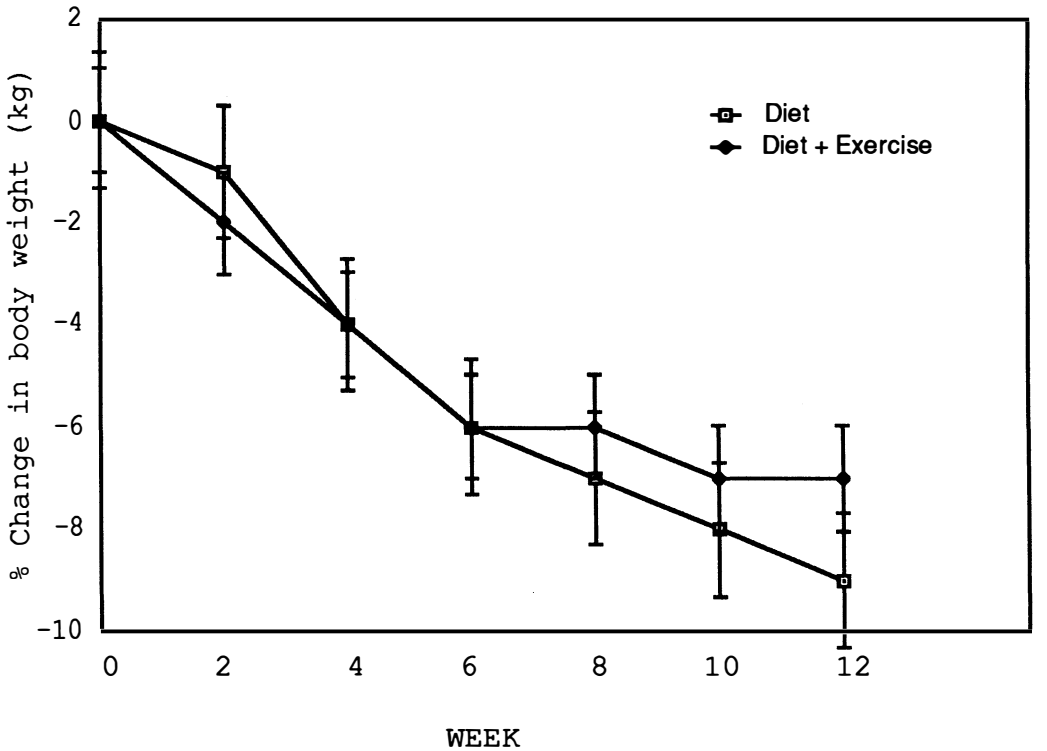


FIGURE IV

Percentage change in body weight for the Diet and Diet + Exercise treatment groups.



groups, weekly weight loss records of this kind were not kept. Body weight and body composition values prior to, and after the 12 weeks of treatment are presented for all groups in table IV. Initially, there was little difference in body weight and body composition between the four groups. However, after the 12 week treatment period the D and DE group showed a significantly greater ($p < 0.05$) weight reduction when compared to the E and C groups. Significant reductions in fat weight ($p < 0.05$) accounted for a portion of the total weight lost in the D and DE groups when compared to the controls. This difference for the DE group did not approach significance when compared to the exercise group. However, the reduction in fat weight displayed by the D group was significantly greater ($p < 0.05$) than any reduction in fat weight exhibited by the exercise group. At posttreatment, the control group displayed a 13% increase in fat weight from the pretreatment value. This increase may have accounted for the significant difference in fat weight between the C group and the E group posttreatment. There were no significant changes in lean body mass.

Maximal Aerobic Capacity Change

Changes in maximal aerobic capacity are presented in table V. When compared to the D and C groups, the E group

TABLE IV

Mean ± SE Changes in Body Weight (BW), Fat Weight (FW), and Lean Body Mass (LBM) According to Treatment Group Before and After 12 Weeks of Treatment.

Variable	Group	Before	After 12 wks	P <.05
BW (kgs)	E	78.1 ± 14.2	77.7 ± 14.6	
	D	77.3 ± 7.1	70.0 ± 6.2	D > E,C
	DE	69.5 ± 4.0	65.1 ± 2.2	DE > E,C
	C	83.7 ± 6.6	84.7 ± 7.1	
FW (kgs)	E	23.6 ± 8.2	23.0 ± 7.7	E > C
	D	27.8 ± 3.7	22.4 ± 3.8	D > E,C
	DE	20.6 ± 2.3	18.4 ± 1.4	DE > C
	C	28.3 ± 3.0	31.9 ± 4.4	
LBM (kgs)	E	54.5 ± 6.4	54.7 ± 7.6	NS
	D	49.5 ± 3.6	47.6 ± 2.3	
	DE	49.0 ± 2.4	46.2 ± 1.8	
	C	55.4 ± 4.1	52.9 ± 3.3	

TABLE V

Mean ± SE For VO_2 max (l/min and ml/kg/min) According to Treatment Group Before and After 12 Weeks of Treatment.

Variable	Group	Before	After 12 wks	P <.05
VO_2 max (l/min)				
	E	2.46 ± 0.17	2.73 ± 0.21	E > D,C
	D	2.46 ± 0.16	2.46 ± 0.17	
	DE	2.45 ± 0.11	2.51 ± 0.10	
	C	2.66 ± 0.09	2.56 ± 0.13	
$V0_2$ max (ml/kg/min)				
	E	33.15 ± 3.7	37.75 ± 4.8	E > C
	D	32.10 ± 1.3	35.38 ± 1.7	D > C
	DE	35.19 ± 1.1	38.55 ± 1.8	DE > C
	C	32.18 ± 1.6	30.66 ± 1.9	

significantly increased ($p < 0.05$) their absolute (L/min) aerobic capacity by 11%. This difference was not significant when compared to the change in absolute aerobic capacity of the DE group. When expressed in relative terms, significant increases ($p < 0.05$) in VO2 max (ml/kg/min) can be noted for the E, D, and DE groups when compared to the control. However, any differences observed between the E, D, and DE groups were not significant.

Plasma Lipid and Lipoprotein Change

Mean values for cholesterol, triglycerides, HDL-C, and LDL-C are presented in tables VI and VII respectively. It can be seen in table VI that both plasma cholesterol and triglyceride in all groups remained unchanged as a result of the 12 week treatment period. Table VII indicates similar findings as both HDL-C and LDL-C remained constant. Likewise the percent of cholesterol carried by HDL remained unchanged.

Discussion

There are three ways in which to lose weight; decrease caloric intake (diet), increase caloric expenditure (exercise), or a combination of the two. In the present study, the effectiveness of these three treatments was evaluated. The results of this study are generally in

TABLE VI

Mean ± SE Values For Total Cholesterol (mg/dl) and Triglycerides According to Group Before and After 12 Weeks of Treatment.

Variable	Group	Before	After 12 wks	P <.05
Cholesterol	E	189 ± 10.0	185 ± 6.4	NS
(mg/dl)	D	200 ± 19.9	201 ± 13.7	
	DE	204 ± 14.2	183 ± 19.6	
	C	209 ± 19.2	208 ± 15.4	
Triglyceride	E	74 ± 10.4	67 ± 0.75	NS
(mg/dl)	D	88 ± 18.7	88 ± 13.3	
	DE	79 ± 5.7	61 ± 5.9	
	C	92 ± 20.0	91 ± 5.5	

TABLE VII

Mean ± SE Values for the Major Lipoprotein Fractions (HDL and LDL) and the Percent of Cholesterol Carried by HDL According to Group Before and After 12 Weeks of Treatment.

Variable	Group	Before	After 12 wks	P <.05
HDL (mg/dl)				
	E	53 ± 3.8	60 ± 4.2	NS
	D	49 ± 5.1	49 ± 3.7	
	DE	49 ± 2.1	48 ± 5.8	
	C	49 ± 2.8	59 ± 4.0	
LDL (mg/dl)				
	E	121 ± 9.6	112 ± 2.8	NS
	D	133 ± 14.8	134 ± 10.1	
	DE	138 ± 12.7	124 ± 16.3	
	C	141 ± 13.2	129 ± 10.0	
% HDL				
	E	0.28 ± .03	0.32 ± .01	NS
	D	0.24 ± .02	0.25 ± .02	
	DE	0.24 ± .02	0.27 ± .02	
	C	0.24 ± .02	0.29 ± .02	

agreement with those of Weltman et al. (129) demonstrating that diet alone, and a combination of diet and exercise are the most beneficial in terms of absolute weight loss. The only difference between the former study and this study, is that this study was not able to demonstrate a difference in weight reduction between the D and DE groups. In contrast to these findings, Zuti and Golding (140) reported that even though significant weight reductions were achieved, there was no difference in the magnitude of weight loss between a diet, exercise, and a combination group. However, in this study equal caloric deficits of 500 calories per day were created for each group by adjusting the caloric expenditure and intake which was necessary to maintain a particular body weight over a 24 day period.

It has been previously reported that exercise alone is capable of producing significant weight reductions (42,79,90,103,133). These studies have indicated that exercise requiring a 300 kcal energy expenditure at least 3 days per week is the threshold for such weight reductions. The results from this study differ from this concept and support the views of others which have indicated that exercise alone is not a sufficient weight loss method (36,45,102,129). Although differences in type, intensity, and frequency of training utilized by these studies may

account for the different results obtained, consideration must also be given to an increase or lack of increase in caloric intake which may or may not occur in response to an exercise training program. In the present study it is difficult determine which, if any, of these conditions influenced the lack of weight reduction in the exercise group. No attempts were made to document caloric intake, nor was there sufficient exercise data recorded to determine caloric expenditure per exercise session.

The addition of exercise to a program of mild caloric restriction is thought to produced the best results in body composition changes which are associated with weight loss (4). This combination promotes the loss of fat weight while lean body mass remains constant or in some cases increases. In contrast, mild caloric restriction alone can result in an unfavorable reduction of fat free tissue in addition to decreases in adipose tissue (39,45,140). In a study by Hagan et al. (45), females who had an initial energy intake of 1723 kcals/day reduced their caloric intake to 1200 kcals/day. After a 12 week treatment period these subjects were able to obtain an 8% reduction in total body weight. Eight-nine percent of the total weight reduction was accounted for through losses of fat weight, while the other 11% was due to a loss in lean body mass. Similarly, Zuti and Golding (140) reported a 21% decrease in lean body weight as a result of

500 kcals/day dietary restriction. However, in this same study when regular exercise was performed along with caloric restriction a favorable reduction of body fat was achieved along with a slight increases in lean body mass. In the present study significant decreases in fat weight were observed in both the D and DE group. Along with these decreases no significant difference in lean body mass between the groups was observed. Although, the tendency was for lean body mass to decrease in the D and DE groups while remaining constant in the exercise group. This would seem to suggest that the amount and intensity of exercise performed by the combination group was not sufficient to maintain lean body mass.

It is well documented in males (36,90,102,103) and in females (31,72,117) that exercise training increases the capacity of the cardiorespiratory system to respond to maximal work. Much of this change is dependent upon the frequency, intensity, and duration of the exercise performed. Those studies which have implemented an aerobic training program consisting of 3 to 5 days/wk (24,36,103), 30 to 45 minutes in duration (31,90,103), and at an intensity ranging from 80 to 90% of maximum heart rate (90,102) have demonstrated the greatest changes in maximum aerobic power.

In the present study, the E group trained at a mean

intensity which was 77% of the max heart rate reserve, while the DE group trained at a mean intensity of 67% of the max heart rate reserve. Significant increases in absolute VO2 max were only observed in the E group when compared to D and C. When the gains in absolute aerobic capacity were compared for the E and DE groups, the difference was not significant. Likewise there were no differences between DE and D and C. This outcome can be mainly attributed to the intensity of exercise performed. Previous reports on the improvement in VO2 max associated with moderately intense training in women have reported increases of around 12% (31,72). This is consistent with the 11% increase exhibited by the E group in the present study. In opposition to these findings, Smith et al. (117) reported only a 6% increase in VO2 max in 10 female subjects who trained at a mean intensity of 73% of their max heart rate. The magnitude of this change was not significant. However, the authors attributed this lack of increase to initially high values in VO2 max. Their pretraining mean was 42.3 ml/kg/min, where fairly active women had been previously reported as having oxygen uptakes which range between 37 and 39 ml/kg/mim (55). The pretraining mean in the present was well below (33.15 ml/kg/min) this range, thus much of the lack of increase, especially in the DE group, can be attributed to the low intensity of exercise.

Some have reported that weight loss can decrease absolute (L/min) VO2 max while relative (ml/kg/min) VO2 max remains constant (130). Others have indicated the weight loss will have the effect of keeping absolute VO2 max constant, while increasing relative VO2 max (45). In the present study, the D and DE groups did not show a significant increases in absolute VO2 max in comparison to any other group. However, when oxygen consumption was expressed in relative terms, both the D and DE groups had significantly increased their oxygen consumptions above that of the control. In the case of the DE group, some of this increase may have been the result of an improved cardiorespiratory function. However, it is unlikely that any cardiorespiratory improvement was attained by the D group, as they did not participate in any regular physical activity. The observed increase in relative oxygen consumption for the D group is a result of the amount of weight that was lost over the 12 week treatment period. Therefore, when significant weight changes have occurred over a period of time, expressing oxygen consumption in absolute terms will give a better indication of the actual improvement in cardiorespiratory function.

Previous reports concerning the effects of diet and or exercise on the plasma concentration of lipid and lipoprotein have yielded conflicting results. Several authors have

suggested that exercise training can independently decrease triglycerides and LDL-C and increase HDL-C (28). Others believe that the associated weight loss which often accompanies the trained state significantly alters the blood lipid profile (121). Weight loss through dieting alone has produced reductions in triglyceride (98) and LDL-C (16), while both an increase (134,139) and a reduction (126,128) in HDL-C has been observed. The present study and ones like it (45,119,129) are unique in that they are particularly well suited for determining the singular and additive effects of diet and exercise on the blood lipid profile. Although in each of these studies all three intervention modalities were controlled in a similar manner, conclusive evidence concerning the singular and synergistic effects of diet and exercise still remain insurmountable.

Similar to the results reported by Hagan et al. (45), this study was not able to provide evidence in support of an increase or a decrease in any of the blood lipid parameters as a result of a particular treatment. In contrast to these findings, Weltman et al. (129) reported significant reductions in plasma cholesterol for all groups, while LDL-C was decreased in the DE and E groups and HDL-C was decreased for the D group. Sopoko et al. (119) has provided some of the most encouraging results reporting that triglycerides and plasma cholesterol were significantly reduced in the DE

group. In addition to this finding, HDL-C significantly increased in all three treatment groups with the change in the DE group representing a summation of the singular effects of both diet and exercise. A summary of all four studies is presented in table VIII.

A major limitation in this study was the small number of subjects in each group. Combined with the variation of the individual test results, these two factors made it difficult to obtain statistical significance between treatment groups. However, this factor should not discredit the results of this study completely, as previous studies utilizing a much larger number of subjects per group have provided similar findings (45).

Overweight individuals generally exhibit increased levels of triglyceride and decreased levels of HDL-C. Some believe that this is a result of a decreased peripheral insulin sensitivity which eventually leads to an increased hepatic production of VLDL triglyceride (98). A preliminary indication of decreased insulin sensitivity is an elevatedblood glucose level. The subjects who took part in this study exhibited neither elevated blood glucose (E: 86.3 ± 3.3 mg%, D: 85.3 ± 1.3 mg%, DE: 87.0 ± 3.9 mg%, C: 87.0 ± 2.5 mg%) or plasma triglyceride levels. This finding is consistent with previous observations that have been made on

TABLE VIII

The Effects of Caloric Restriction and Exercise on Plasma Lipids

Author	(ref)	No. of Subjects	Duration	Body Weight (kgs)	Tri	Chol	LDL	HDL	Group
Hagan	(45)	48 F	12 wks	-0.6	-27	+5	+12	-3	E
				-5.5*	-6	-12	-6	-4	D
				-7.5*	-37	-1	+11	-5	DE
				-0.8	+6	+9	+10	-3	C
Sopoko	(119)	21 M	15 wks	-0.5	+5	+16	+9	+4*	E
				-6.2*	+20	+13	+8	+3*	D
				-6.1*	-71	-7*	-12	+11*	DE
				-0.5	-1	-1	+5	-1	C
Weltman	(129)	58 M	10 wks	-0.9	-	-18*	-14	+5	E
				-5.6*	-	-20*	-2	-9*	D
				-5.4*	-	-30*	-19*	nc	DE
				-0.3	-	+5	-1	-1	C
Zachwieja		16 F	12 wks	-0.4	-7	-4	-9	+5	E
				-7.3*	nc	+1	+1	nc	D
				-4.4*	-18	-21	-14	-1	DE
				+1.0	+3	-1	-12	+10	C

$p < 0.05$ = *

females. Females tend to have lower plasma triglyceride and higher HDL-C levels when compared to age matched males (49). It has been suggested that this is a result of the higher LPL activity which is often found in females (97). Initially high HDL-C levels, and initially low triglyceride values, have generally yielded a small insignificant response to exercise (34,59,93,128). It is interesting to note that both Sopoko and Weltman used healthy non-active overweight males in their studies. This factor may have accounted for a number of the observed changes. Initial values for HDL-C and triglyceride in the study of Spoko et al. (119) ranged between 37 mg% and 42 mg% and 112 mg% and 130 mg% respectively. Hagan et al. (45) reported values for HDL-C and triglyceride ranging from 49 mg% to 53 mg% and 83 mg% to 123 mg% respectively. Similarly, we reported much higher initial HDL-C levels (49 mg% to 53 mg%) and lower triglyceride values (74 mg% to 92 mg%) than did Sopoko et al. (119). Both this study and that of Hagan et al. (45) used females for subjects. Although in all three studies similar intensities of exercise were used, it seems likely that because of the initial difference in blood lipid values between men and women, in women higher intensities of exercise may be required to produce changes in the blood lipid profile. In fact, there is evidence to suggest that

jogging 30 to 60 miles per week is the threshold of exercise needed to produce significant changes in the blood lipid profile (40,78,108).

In this study, weight loss produced through dieting alone had no significant effect on the levels of either plasma triglyceride or HDL-C. This is in agreement with the results of Hagan et al. (45), but in opposition to those of Weltman et al. (129) who indicated a decrease in HDL-C levels and Sopoko et al. (119) who indicated an increase in HDL-C levels. Previous studies which have demonstrated an increase in HDL-C in response to weight loss have done so only during a period body weight stabilization (119,134,139). Zimmerman et al. (139) followed the time course of HDL-C and triglyceride response during a 7 week period of active weight loss. During this time, tiglycerides decreased steadily and remained reduced several weeks after the termination of active weight loss. On the other hand, HDL-C concentrations did not differ significantly throughout active weight loss. After active weight loss had stabilized, there was an increase in HDL-C above the pretreatment values. In this study, the negative association which is common between plasma triglyceride and HDL-C was not evident during periods of active weight loss. Thus, the observed reduction in triglyceride during active weight reduction must have been a result of a decreased hepatic production of VLDL triglyceride

and not a consequence of increased LPL activity. A previous report has indicated that postheparin and adipose tissue LPL activity is reduced during periods of caloric restriction (124). This supports the idea of a decreased hepatic production of triglyceride, as well as justifying the static response of HDL-C observed by Zimmerman et al. (139). Presumably, when body weight stabilizes, a return of LPL activity back to or beyond pretreatment levels will result in the increase in HDL-C levels which have been associated with a reduction in body weight (119,134,139). In the study of Sopoko et al. (119) a period of weight stabilization was established before the posttreatment analysis of plasma lipid and lipoprotein. This may explain why they observed increases in HDL-C and the present study did not. Furthermore, Weltman et al. (129) and Hagan et al. (45) also neglected to define periods of active weight loss and weight stabilization.

Evidence for an increase in HDL-C levels resulting from independent programs of diet and exercise has been provided (53,71,100,107,111,119,134,139). In particular, Sopoko et al. (119) has illustrated this phenomenon rather well. In addition to these independent effects, Sopoko et al. (119) demonstrated that a combination of weight loss and exercise resulted in the greatest change in HDL-C levels. Furthermore,

this change represented a summation of the independent increases observed for both the weight loss and exercise interventions. Future studies implementing a similar design are needed in order to substantiate such an effect.

CHAPTER 5

SUMMARY

Thirty-two women volunteered to participate in a 12 week weight loss study. Each was assigned to one of 3 groups; exercise only, diet only, and diet plus exercise. After background information had been obtained on these individuals via a health history questionnaire, initial tests of maximum aerobic capacity, body composition, and blood lipid analysis were given. Those individuals who were assigned to the exercise group met 3 times per week to participate in a progressive walk/jog program. They were instructed to exercise for 30 to 45 minutes at a heart rate which was 70% of the heart rate reserve obtained during the aerobic capacity test. Those individuals assigned to the diet only group participated in a behavioral/nutritional weight loss program. These subjects were required to limit caloric intake to 1200 kcals/day, maintain daily dietary records of the amount, type, and caloric value of the food consumed, and to attend a weekly meeting. Subjects assigned to the diet plus exercise group participated in both of the above programs. For various reasons several of the subjects had to withdraw from the study before the completion of the third week. Some of these individuals agreed to become part of a

control group, while others remained completely withdrawn from the study. By the end of the 12th week, participation in the study declined to 38% of the original subject pool. Those individuals who completed the full 12 weeks of treatment were post tested for aerobic capacity, body composition, and blood lipid content.

Conclusions

Based on the results of this study the following conclusions appear warranted;

1. Programs of diet and diet plus exercise produce the greatest reduction in absolute body weight.
2. Weight loss achieved through diet and diet plus exercise results in a significant reduction of fat weight.
3. Exercise that is performed 3 times per week, 30 to 45 minutes in duration, and at 70% of heart rate reserve, in the absence of weight loss will produce a significant increase in absolute (L/min) VO2 max.
4. During periods of weight loss, it is better to express changes in oxygen consumption in absolute (L/min) terms, as a reduction in body weight can falsely elevate an oxygen consumption expressed in relative (ml/kg/min) terms.

5. Reductions in body weight and/or increases in aerobic fitness achieved through caloric restriction and exercise training (70% heart rate reserve) over a 12 week period does not produce significant changes in the plasma concentrations of cholesterol, triglyceride, HDL-C or LDL-C.

Recommendations

For future studies implementing a similar design, the following recommendations are made based on the results and experiences gained from this study;

1. Weight loss records should be kept for exercise, diet, diet plus exercise, and control groups alike.
2. In addition to pretreatment and posttreatment measurements for plasma lipid and lipoprotein concentration, blood lipid analyses should also be performed every other week for the duration of the study to verify the effect of active weight loss on HDL-C.
3. Posttreatment blood lipid and lipoprotein analysis should only be performed once reductions in body weight have stabilized.
4. Either adipose tissue or heparin released lipoprotein lipase (LPL) activity should be measured prior to the onset of treatment, every other week during the

treatment, and posttreatment to verify the relationship between LPL activity and HDL-C.

5. Follow-up data 4, 6, and 8 months post treatment should be obtained on all parameters previously tested.

6. Attempts should be made to study changes in the distribution of the different apoprotein constituents of HDL.

7. Detailed dietary information via a dietary recall should be obtained on each subject prior to and after the specified treatment period.

8. In addition to exercise time and heart rate, accurate records pertaining to the distance covered during a single exercise session should also be maintained. With this information weekly caloric expenditure can be estimated.

9. Exercise intensities greater than 70% of max heart rate reserve should be prescribed.

10. Both males and females should be used with comparisons between these two groups made.

REFERENCES

1. Adner, M. M., and Castelli, W. W. Elevated High Density Lipoprotein Levels in Marathon Runners. J.A.M.A. 243:534-536, 1980.

2. Allison, T. G., Iammarino, R. M., Metz, K.F., Skrinar, G. S., Kuller, L. H., and Robertson, R. J. Failure of Exercise to Increase High Density Lipoprotein Cholesterol. J. Cardiac.Rehab. 1:257-265, 1981.

3. American College of Sports Medicine Position Stand on Proper and Improper Weight Loss. Med. Sci. Sports. Exercise. 15:9-13, 1983.

4. Anderson, K. M., Castelli, W. P., and Daniel, L. Cholesterol and Mortality: 30 Years of Follow-up From The Framingham Study. J.A.M.A. 257:2176-2180, 1987.

5. Askew, E.W., Huston, R. L., and Dohm, G.L. Effect of Physical Training on Esterification of Glycerol-3-Phosphate by Homogenates of Liver, Skeletal Muscle, Heart, and Adipose Tissue of Rats. Metabolism. 22:473-489, 1973.

6. Beneke, W. M., and Timson, B. F. Some Health-Risk Bebefits of Behavioral Weight-loss Treatments. Psychological Reports. 61:199-206, 1987.

7. Beneke, W. B., Timson, B. F., and Vander Tuig, J. G. Assessment of Health Risk Benefits of Weight Loss Treatments: A Review of Methodological and Pragmatic Considerations. Behavioral Assessment. 9:349-359, 1987.

8. Berg, A., Kuel, J., Ringwald, G., Dues, B., and Wybitul, K. Physical Performance and Serum Cholesterol Fractions in Healthy Young Men. Clinica Chimica Acta. 106:325-330, 1980.

9. Berg, A., Ringwald, G., and Keul, J. Lipoprotein-Cholesterol in Well Trained Atheletes. A Preliminary Communication: Reduced HDL-Cholesterol in Power Atheletes. Int. J.Sports. Medicine. 1:137-138, 1980.

10. Bergstrom, J., Hermansen, L., Hultman, E., and Saltin, B. Diet, Muscle Glycogen, and Physical Performance. Acta.Physiol. Scand. 71:140-150, 1967.

11. Bjorntorp, B. P., Fahlen, M., Grimby, G., Gustafson, A., and Holm, J. Carbohydrate and Lipid Metabolism in Middle-aged, Physically Well-trained Men. Metabolism. 21:1037-1044, 1972.

12. Bonanno, J. A., and Lies, J. E. Effects of Physical Training on Coronary Risk Factors. Am. J. Card. 33:760-764, 1974.

13. Borensztajn, J., Rone, M. S., Babirak, S. P., McGarr, J. A., and Oscai, L. B. Effect of Exercise on Lipoprotein Lipase Activity in Rat Heart and Skeletal Muscle. Am. J. Physiol. 229:394-397, 1975.

14. Bray, G. A., (ed.) Obesity in Perspective. Vol. 2. Washington DC: US Government Printing Office. (NIH Publ. No. 75-708, p.72) 1973.

15. Brownell, K. D., Bachori, K. P. S., and Ayerle, R. S. Changes in Plasma Lipid and Lipoprotein Levels in Men and Women After a Program of Moderate Exercise. Circulation. 65:477-484, 1982.

16. Brownell, K. D., and Stunkard, A. J. Differential Changes in Plasma High-Density Lipoprotein-Cholesterol Levels in Obese Men and Women During Weight Reduction. Arch. Intern. Med. 141:1142-1146, 1981.

17. Campbell, D. E. Influence of Several Physical Activities on Serum Cholesterol Concentrations in Young Men. J. Lipid. Research. 6:478-480, 1965.

18. Carlson, L. A., and Mossfeldt, F. Acute Effects of Prolonged, Heavy Exercise on the Concentration of Plasma Lipids and Lipoproteins in Man. Acta. Physiol. Scand. 62:51-59, 1964.

19. Clarkson, P. M., Hintermister, R., Fillyan, M., and Stylos, L. High-Density Lipoprotein-Cholesterol in Young Adult Weight Lifters, Runners, and Untrained Subjects. Human Biology. 53:251-257, 1981.

20. Cooper, K. H., Pollock, M. L., Martin, A. C., and Jackson, A. Physical Fitness Levels vs. Selected Coronary Risk Factors: A Cross-Sectional Study. J.A.M.A. 236:166-169, 1976.

21. Costill, D. L., Daniels, J., Evans, W., Fink, W., Krahenbuhl, G., and Saltin, B. Skeletal Muscle Enzymes and Fiber Composition in Male and Female Track Athletes. J. Appl. Physiol. 40:149-154, 1976.

22. Costill, D. L., Fink, W. L., and Pollock, M. L. Fiber Composition and Enzyme Activities of Elite Distance Runners. Med. Sci. Sports. Exercise. 8:96-100, 1976.

23. Craig, B. W., Hammonns, G. T., Garthwait, S. M., Jarett, L., and Holloszy, J. O. Adaptation of Fat Cells to Exercise: Response of Glucose Uptake and Oxidation to Insulin. J. Appl. Physiol. 51:1500-1506, 1981.

24. Crews, T. R., and Roberts, J. A. Effect of Interaction of Frequency and Intensity of training. Research Quarterly. 47:48-55,1976.

25. Cullinane, E., Lazarus, B., and Thompson, P. D. Acute Effects of a Single Exercise Session on Serum Lipids in Untrained Men. Clinica. Chimica. Acta. 109:341-344, 1981.

26. Cullinane, E., Siconolfi, S., Saritelli, A., and Thompson, P.D. Acute Decrease in Serum Triglycerides With Exercise: is There a Threshold For an Exercie Effect? Metabolism. 31:844-847, 1982.

27. Dressendorfer, R. H., and Gahagen, H. Serum Lipid Levels in Male Runners. Phys. Sportsmed. 7:119-125, 1979.

28. Dufaux, B., Assmann, G., and Hollmann, W. Plasma Lipoproteins and Physical Activity: A Review. Int. J. Sports. Med. 3:123-136, 1982.

29. Eder, H. A. Lipoproteins and Coronary Artery Disease. Hospital Practice. 18:215-222, 1983.

30. Farrell, P. A., Maksud, M. G., Pollock, M. L., Foster, C., Anholm, J., Hare, J., and Leon, A. S. A Comparison of Plasma Cholesterol, Triglycerides, and High Density Lipoprotein Cholesterol in Speed Skaters, Weight Lifters, and Non-athletes. Eur. J. Appl. Physiol. 48:77-82, 1982.

31. Flint, M. M., Drinkwater, B. L., and Horvath, S. M. Effects of Training on Women's response to Submaximal Exercise. Med. Sci. Sports. Exercise. 6:89-94, 1974.

32. Frey, M. A. B., Doerr, B. M., Laubach, L.L., Mann, B.L., and Glueck, C. J. Exercise Does Not Change High-Density Lipoprotein Cholesterol in Women After 10 weeks of Training. Metabolism. 31:1142-1146, 1982.

33. Froberg, S. O. Effects of Training and of Acute Exercise in Trained Rats. Metabolism. 20:1044-1051, 1971.

34. Gaesser, G. A., and Rich, R. G. Effects of High- and Low-Intensity Exercise training on Aerobic Capacity and Blood Lipids. Med. Sci. Sports. Exercise. 16:269-274, 1984.

35. Garfinkel, A.S., Nilsson-Ehle, P., and Schotz, M. C. Regulation of Lipoprotein Lipase Induction by Insulin. Biochim. Biophysics. Acta. 424:264-273, 1976.

36. Gettman, L. R., Pollock, M. L., Durnstine, J. L., Ward, A., Ayers, J., and Linnerud, A. C. Physiological Response of Men to 1, 3, and 5 Day per Week Training Programs. Research Quarterly. 47:638-646, 1976.

37. Gofman, J., Lindgren, F., and Elliot, H. Ultracentrifugation Studies of Lipoproteins of Human Serum. J. Biol. Chem. 173:973-981, 1949.

38. Goldberg, L. E., Elliot, D. L., Schutz, R. W., and Kloster, F. E. Changes in Lipid and Lipoprotein Levels After weight Training. J.A.M.A. 252:504-506, 1984.

39. Goldman, R. F., Bullen, B., and Seltzer, C. Changes in Specific Gravity and Body Fat in Overweight Female Adolescents as a Result of Weight Reduction. Ann. NY Acad. Sci. 110:913-917, 1963.

40. Goodyear, L. J., Fronsoe, M. S., Van Houten, D. R., Dover, E. V., and Larry, J. Increased HDL-Cholesterol Following Eight Weeks of Progressive Endurance Training in Female Runners. Annals of Sports Med. 3:33-38, 1986.

41. Gordon, T., Castelli, W. P., Hjortland, M.C., Kannal, W. B., and Dawber, T. R. High Density Lipoprotein as a Protective Factor Against Coronary Heart Disease. Am. J. Med. 62:707-714, 1977.

42. Gwinup, G. Effect of Exercise Alone on the Weight of Obese Women. Arch. Intern. Med. 135:676-680, 1975.

43. Gyntelberg, F., Brennan, R., Holloszsy, J.O., Schonfeld, G., Rennie, M. J., and Weifman, S. W. Plasma Triglyceride Lowering by Exercise Despite Increased Food Intake in Patients With Type IV Hyperlipoproteinemia. Am. J. Clin. Nutr. 30:716-720, 1977.

44. Hagan, R. D., and Gettman, L. R. Maximal Aerobic Power, Body Fat, and Serum Lipoproteins in Male Distance Runners. J. Cardiac. Rehab. 3:331-337, 1983.

45. Hagan, R. D., Upton, S. J., Wong, L., and Whittam, J. The Effects of Aerobic Conditioning and/or Caloric Restriction in Overwight Men and Women. Med. Sci. Sports. Exercise. 18:87-94, 1986.

46. Hartung, G. H., Farge, E. J., and Mitchell, R. E. Effects of Marathon Running, Jogging, and Diet on Coronary Risk Factors in Middle-Aged Men. Prev. Med. 10:316-323, 1981.

47. Hartung, G. H., Foreyt, J. P., Mitchell, R. F., Vlasek, I., and Gotto, A. M. Relation of Diet to High Density Lipoprotein Cholesterol in Middle-aged Marathon Runners, Joggers, and Inactive Men. N. Eng. J. Med. 302:357-361, 1980.

48. Hartung, G. H., and Squires, W. G. Exercise and High Density Lipoprotein Cholesterol in Middle-aged Men. Phys. Sportsmed. 8:74-79, 1980.

49. Haskell, W. L. The Influence of Exercise on the Concentrations of Triglyceride and Cholesterol in Human Plasma. In: Exercise and Sports Science Reviews, R.L., Terjung (ed.). Lexington, MA: Collamore Press, 1984. Vol. 12, pp. 204-244.

50. Haskell, W. L., Taylor, H. L., Wood, P. D., Schrott, H., and Heiss, G. Strenuous Physical Activity, Treadmill Exercise Test Performance and Plasma High Density Lipoprotein Cholesterol. Circulation. 62:(suppl IV)53-61, 1980.

51. Havel, R. J., Pernow, B., and Jones, N. L. Uptake and Release of Free Fatty Acids and Other Metabolites in the Legs of Exercising Men. J. Appl. Physiol. 23:90-96, 1967.

52. Herbert, P. N., Bernier, D. N., Cullinane, E. M., Edelstein, L., Kantor, M. A., and Thompson, P. D. High Density Lipoprotein Metabolism in Runners and Sedentary Men. J.A.M.A. 252:1034-1037, 1984.

53. Hespel, P., Lijnen, P., Fagard, R., Van Hoof, R., Rosseneu, M., and Amery, A. Changes in Plasma and Apoproteins Associated with Physical Training in Middle-aged Sedentary Men. Am. Heart. J. 115:786-792, 1988.

54. Hicks, A. L., MacDougall, J. D., and Muckle, T. J. Acute Changes in High Density Lipoprotein Cholesterol With Exercise of Different Intensities. J. Appl. Physiol. 63:1956-1960, 1987.

55. Higgs, S. L. Maximal Oxygen Intake and Maximal Work Performance of Active College Women. Research Quarterly. 44:125-131, 1973.

56. Hoffman, A. A., Nelson, W., and Goss, F. A. Effects of an Exercise Program on Plasma Lipids of Senior Air Force Officers. Am. J. Card. 20:516-524, 1967.

57. Holloszy, J. O. Adaptation of Muscular Tissue to Training. In, Sonnenblick, E. H., Lesch, M. (eds): Exercise and Heart Disease. New York, Grune and Stratton, 1977.

58. Holloszy, J. O., Skinner, J. S., Toro, G., and Cureton, T. K. Effects of a Six Month Program of Endurance Exercise on the Serum Lipids of Middle-aged Men. Am. J. Card. 14:753-760, 1964.

59. Hunt, H. F., and White, J. R. Effects of 10 Weeks of Vigorous Daily Exercise on Serum Lipids and Lipoproteins in Teenage males. Med. Sci. Sports. Exercise. 12:(abstr)93, 1980.

60. Hurley, B. F., Hagberg, J. M., Goldberg, A. P., and Holloszy, J. O. Resistive Training Can Reduce Coronary Risk Factors Without Altering VO2 max or Percent Body Fat. Med. Sci. Sports. Exercise. 20:150-154, 1988.

61. Hurley, B. F., Seals, D. R., and Hagberg, J. M. High Density Lipoprotein Cholesterol In Bodybuilders vs. Powerlifters: Negative Effects of Androgen Use. J.A.M.A. 252:507-513, 1984.

62. Hurtler, R., Swale, J., Peyman, M. A., and Barnett, H. Some Immediate and Long-term Effects of Exercise on Plasma Lipids. Lancet. 2:671-674, 1972.

63. Huttunen, J. K., Lansimies, E., Voultilainen, E., Ehnholm, C., Hietanen, E., Penttila, I., Siitonen, O., and Rauramaa, R. Effects of Moderate Physical Exercise on Serum Lipoproteins. Circulation. 60:1220-1229, 1979.

64. Jackson, A. S., Pollock, M. L., and Ward, A. Generalized Equations For Predicting Body Density in Women. Med. Sci. Sports. Exercise. 12:175-182, 1980.

65. Jacobs, I., Lithell, H., and Karlsson, J. Dietary Effects on Glycogen and Lipoprotein Lipase Activity in Skeletal Muscle in Man. Acta. Physiol. Scand. 115:85-90, 1982.

66. Johnson, C. C., Stone, M. H., Lopez, S. A., Herbert, J. A., Kilgore, L. T., and Byrd, R. J. Diet and Exercise in Middle-aged Men. J. Am. Dietetic Assoc. 81:695-701, 1982.

67. Jones, N. L., and Havel, R. J. Metabolism of Free Fatty Acids and Chylomicron Triglycerides during Exercise in Rats. Am. J. Physiol. 213:824-828, 1967.

68. Kannel, W. B., Castelli, W. P., and Gordon, T. Cholesterol in the Prediction of Atherosclerotic Disease: New Perspectives Based on the Framingham Study. Ann. Int. Med. 90:85-91, 1979.

69. Kantor, M. A., Cillinane, E. M., Herbert, P. N., and Thompson, P. D. Acute Increase in Lipoprotein Lipase Following Prolonged Exercise. Metabolism. 33:454-457, 1984.

70. Keul, J., Kohler, B., van Glutz, G., Luthi, U., Berg, A., and Howald, H. Biochemical Changes in a 100 km Run: Carbohydrates, Lipids, and Hormones in Serum. Eur. J. Appl. Physiol. 47:181-189, 1981.

71. Kiens, B., Jorgensen, I., Lewis, S., Jesen, G., Lithell, H., Vessby, B., Hoe, S., and Schnohr, P. Increased Plasma HDL-C Cholesterol and A-1 in Sedentary Middle-aged Men After Physical Conditioning. Eur. J. Clin. Invest. 10:203-209, 1980.

72. Kilbom, A. Physical Training in Women. Scand. J. Clin. Lab. Invest. 28:(suppl. 119), 1971.

73. Kilbom, A., Hartley, L. H., Saltin, B., Bjore, J., Grimby, G., and Astran, I. Physical Training in Sedentary Middle-aged and Older Men. J. Clin. Lab. Invest. 24:315-322, 1969.

74. King, D. S., Dalsky, G. P., Clutter, W. E., Young, D. A., Staten, M. A., Cryer, P. E., and Holloszy, J. O. Effects of Exercise and Lack of exercise on Insulin Sensitivity and Responsiveness. J. Appl. Physiol. 64:1942-1946, 1988.

75. Kuusi, T., Nikkila, E. A., Saarinen, P., Varjo, P., and Laitinen, L. A. Plasma High Density Lipoproteins HDL2, HDL3 and Postheparin Plasma Lipases in Relation to Parameters of Physical Fitness. Atherosclerosis. 41:209-219, 1982.

76. Larosa, J. C., Fry, A. G., Muesing, R., and Rosing, D. R. Effects of High-Protein Low-Carbohydrate Dieting on Plasma Lipoproteins and Body Weight. J. Am. Dietetic. Assoc. 77:264-270, 1980.

77. LeBlanc, J., Nadeau, A., Bouloy, M., and Rausseuu-Migneron, S. Effects of Physical Training and Adiposity on Glucose Metabolism, and ^{125}I Binding. J. Appl. Physiol. 46:235-239, 1979.

78. Lehtonen, A., and Viikari, J. Serum Triglycerides and Cholesterol and Serum High Density Lipoprotein in Highly Physicaly Active Men. Acta. Med. Scand. 204:111-114, 1978.

79. Leon, A. S., Conrad, J., Hunninghake, D. B., and Serfass, R. Effects of a Vigorous Walking Program on Body Composition, and Carbohydrate and Lipid Metabolism of Obese Young Men. Am. J. Clin. Nutr. 32:1776-1787, 1979.

80. Lewis, S., Haskell, W. L., Wood, P. D., Manoogian, N., Baily, J. E., and Pereira, M. Effects of Physical Activity on Weight Reduction in Obese Middle-aged Women. Am. J. Clin. Nutr. 29:151-156, 1976.

81. Linder, C., Chernick, S. S., Fleck, T. R., and Scow, R. O. Lipoprotein Lipase and Uptake of Chylomicron Triglyceride by Skeletal Muscle of Rats. Am. J. Physiol. 231:860-864, 1976.

82. The Lipid Research Clinics Coronary Primary Prevention Trials Results II. The Relationship of Reduction Incidence of Coronary Heart Disease to Cholesterol Lowering. J.A.M.A. 251:365-374, 1984.

83. Lithel, H., Orlander, J., Schele, R., Sjodin, B., and Karlsson, J. Changes in Lipoprotein Lipase Activity and Lipid Stores in Human Skeletal Muscle With Prolonged Heavy Exercise. Acta. Physiol. Scand. 107:257-261, 1979.

84. Lopez-S, Vial, R., Balart, L., and Arroyave, G. Effect of Exercise and Physical Fitness on Serum Lipidss and Lipoproteins. Atherosclerosis. 20:1-9, 1974.

85. Mackie, B. G., Dudley, G. A., Kaciuba-Uscilko, H., and Terjung, R. L. Uptake of Chylomicron Triglycerides by Contracting Skeletal Muscle in Rats. J. Appl. Physiol. 49:851-855, 1980.

86. Marniemi, J., Dahlstrom, S., Kuist, M., Seppanen, A., and Hietanen, E. Dependence of Serum Lipid and Lecithin: Cholesterol Acyltransferase levels on Physical Training of Young Men. Eur. J. Physiol. 491:25-35, 1982.

87. Martin, R. R., Haskell, W. P., and Wood P. D. Blood Chemistry and Lipid Profiles of Elite Distance Runners. Ann. NY. Acad. Sci. 301:346-360, 1977.

88. Melichar, F. Plasma Cholesterol and Phospholipids in Various Occupational Groups. J. Atherosclerosis. Res. 5:432-435, 1965.

89. Milesis, C. A., Effects of Metered Physical Training on Serum Lipids of Adult Men. J. Sports. Med. Phys. Ed. 14:8-13, 1974.

90. Milesis, C. A., Pollock, M. L., Bah, M. D., Ayers, J. J., Ward, A., and Linnerud, A. C. Effects of Different Durations of Physical Training on Cardiorespiratory Function, Body Composition, and Serum Lipids. Research Quarterly. 47:716-725, 1976.

91. Miller, G. J., Miller, N. E. Plasma High Density Lipoprotein Concentration and Development of Ischemic Heart Disease. Lancet. 1:16-19, 1975.

92. Montoye, H. J., Block, W. D., and Gayle, R. Maximal Oxygen Uptake and Blood Lipids. J. Chron. Dis. 31:111-118, 1978.

93. Moore, C. E., Hartung, G. H., Mitchel, R. E., Kappus, C. M., and Hinderlitter, J. The Relationship of Exercise and Diet on High-Density Lipoprotein Cholesterol Levels in Women. Metabolism. 32:189-196, 1983.

94. Morgan, D. W., Cruise, R. J., Girardin, B.W., Lutz-Schneider, V., Morgan, D. H., and Wang, M. HDL-C Concentration in Weight- Trained, Endurance Trained and Sedentary Females. Phys. Sportsmed. 14:166-181, 1986.

95. Moss, R. F., and Bonner, H. The Effect of a 5 Week Monitored Training Program on Fasting Levels of Serum Cholesterol and Triglyceride in Males Maintained at Constant Weight. Med. Sci. Sports. Exercise. 11:(abstr)109, 1979.

96. McDonough, J. R., Kusumi, F., and Bruce, R. A. Variation in Maximal Oxygen Intake With Physical Activity in Middle-Aged Men. Circulation. 41:743-751, 1970.

97. Nikkila, E. A., Taskinen, M., Rehunen, S., and Harkonen, M. Lipoprotein Lipase Activity in Adipose Tissue and Skeletal Muscle of Runners: Relation to Serum Lipoproteins. Metabolism. 27:1616-1671, 1978.

98. Olefsky, J., Reaven, G. M., and Farquhar, J. W. Effects of Weight Reduction on Obesity. J. Clin. Invest. 53:64-76, 1974.

99. Paulsen, B. K., Beneke, W. M., Moore, C. Eating Slim: A Treatment Manuel For Effective Weight Control Through Behavior Therapy and Nutritional Education. Columbia MO: Lincoln University and University of Missouri Extension Press. 1976.

100. Peltonen, P., Marniemi, J., Hietonen, E., Vuori, I., and Ehnholm, C. Changes in Serum Lipids, Lipoproteins, and Heparin Releasable Lypolitic Enzymes During Moderate Physical Training in Man: A Longitudinal Study. Metabolism. 30:518-526, 1981.

101. Peltonen, P., Marniemi, J., Vuori, I., and Hietaned, E. The Effect of Controlled Physical Training on Lipid Metabolism in Man. A Longitudinal Study. Acta. Physiol. Scand. 108:(abstr)A26, 1980.

102. Pollock, M. L., Broida, J., Kendrick, Z., Miller, H. S., Janeway, R., and Linnerud, A. C. Effects of Training Two Days a Week at Differenrt Intensities on Middle-Aged Men. Med. Sci. Sports. Exercise. 4:192-197, 1972.

103. Pollock, M. L., Cureton, T. K., and Greninger, L. Effects of Frequency of Training on Working Capacity, Cardiovascular Function, and Body Composition of Adult Men. Med. Sci. Sports. Exercise. 1:70-74, 1969.

104. Pollock, M. L., Wimore, J. H., Fox, S. M. Exercise in Health and Disease. Philadelphia: W.B. Saunders Company. 1984, pp. 3-28.

105. Ratliff, R., Elliot, K., and Rubenstein, C. Plasma Lipid and Lipoprotein Changes With Chronic Training. Med. Sci. Sports. Exercise. 10:(abstr)55, 1978.

106. Rhodes, G. G., Gulbrandsen, C., L., and Kagan, A. Serum Lipoproteins and Coronary Heart Disease in a Population Study of Hawaii Japanese Men. N. Eng. J. Med. 294:293-298, 1976.

107. Rotkis, T., Boyden, T. W., Pamenter, R. W., Stanforth, P., and Wilmore, J. High Density Lipoprotein Cholesterol and Body Composition of Female Runners. Metabolism. 30:994-995, 1981.

108. Rotkis, T. C., Cote, R., Coyle, E., and Wilmore, J. H. Relationship Between High Density Lipoprotein Cholesterol and Weekly Running Milage. J. Cardiac. Rehab. 2:109-112, 1982.

109. Schwartz, R. S., and Brunzell, J. D. Increase in Adipose Tissue Lipoprotein Lipase Activity With Weight Loss. J. Clin. Invest. 67:1425-1430, 1981.

110. Seals, D. R., Hagberger, J. M., Allen, W. K., Hurley, B. F., Dalsky, G. P., Ehsoni, A. A., and Holloszy, J. O. Glucose Tollerance in Young and Older Athletes and Sedentary Men. J. Appl. Physiol. 56:1521-1525, 1984.

111. Seals, D. L., Hagberg, J. M., Hurley, B. F., Ehsani, A. A., and Holloszy, J. O. Effects of Endurance Training on Glucose Tollerance and Plasma Lipid Levels in Older Men and Women. J.A.M.A. 252:645-649, 1984.

112. Sedgwick, A. W., Brotherhood, J. R., Harris-Davidson, A., Taplin, R. E., and Thomas, D. W. Long-Term Effects of Physical Training Programme on Risk Factors For Coronary Heart Disease in Otherwise Sedentary Men. British. Med. J. 281:7-10, 1980.

113. Shane, S. R. Relation Between Serum Lipids and Physical Conditioning. Am. J. Card. 18:540-543, 1966.

114. Simonelli, C., and Eaton, R. P. Reduced Triglyceride Secretion: A Metabolic Consequence of Chronic Exercise. Am. J. Physiol. 234:E221-E227, 1978.

115. Siri, W. E. The Gross Composition of the Body. In: Advances in Biological Medical Physics. Vol. 4. NY: Academic Press, 1956 pp. 239-280.

116. Skinner, J. S., Benson, H., McDonough, J. R., and Hairess, C. G. Social Status, Physical Activity, and Coronary Proneness. J. Chron. Disease. 19:773-783, 1966.

117. Smith, D. P., and Stransky, F. W. The Effect of Training and Detraining on the Body Composition and Cardiovascular Response of Young Women to Exercise. J. Sports. Med. Phys. Ed. 16:112-120, 1976.

118. Smith, M. P., Mendez, J., Dauckenmiller, M. Exercise Intensity, Dietary Intake, and High Density Lipoprotein in Young Female Competitive Swimmers. Am. J. Clin. Nutr. 36:251-255, 1982.

119. Sopko, G., Leon, A. S., Jacobs, D. R., Foster, N., Moy, J., Kuba, K., Anderson, J. Y., Casal, D., McNally, C., and Frantz, I. The Effects of Exercise and Weight Loss on Plasma Lipids in Young Obese Men. Metabolism. 34:227-236, 1985.

120. Stamford, B.A., Matter, S., Fell, R.D., Sady, S., Cresanata, M.K., and Papanek, P. Cigarette Smoking, Physical; Activity, and Alcohol Consumption: Relationship to Blood Lipids and Lipoproteins in Premenopausal Females. Metabolism. 33:585-590, 1984.

121. Stern, M. P., Olefsky, J., Farquhar, J.W., and Reaven, G. M. Relationship Between Fasting Plasma Lipid Levels and Adipose Tissue Morphology. Metabolism. 22:1311-1317, 1973.

122. Stubbe, I., Hansson, P., Gustafson, A., and Nilsson-Ehle, P. Plasma Lipoproteins and Lypolitic Enzyme Activities Durning Endurance Training in Sedentary Men: Changes in High Density Lipoprotein Subfractions and Composition. Metabolism. 32:1120-1128, 1983.

123. Superko, R. H., Bachorik, P.S., Wood, P. D. High Density Lipoprotein Cholesterol Measurements. A Help or Hinderance in Practical Medicine? J.A.M.A. 256:2714-2717, 1986.

124. Taskinen, M., and Nikkila, E. A. Lipoprotein Lipase of Adipose Tissue and Skeletal Muscle in Human Obesity: Response to Glucose and Semi Starvation. Metabolism. 30:810-817, 1981.

125. Thomas, T. R., Adeniran, S.B., and Etherridge, G. L. Effects of Different Running Programs on VO2 max, Percent Fat, and Plasma Lipids. Can. J. Appl. Sports. Sci. 9:55-62, 1984.

126. Thompson, J., Cole, J., Trost, R., Yeshurun, D., and Debakey, M. Relationship Between Plasma Lipid Concentration and Coronary Artery Disease in 496 Patients. Circulation. 56:875-883, 1977.

127. Thompson, P. D., Jeffery, R. W., Wing, R. R., and Wood, P. D. Unexpected Decrease in Plasma High Density Lipoprotein Cholesterol with Weight Loss. Am. J. Clin. Nutr. 32:2016-2021, 1979.

128. Webster, W. A., Smith, D.P., LaRosa, J. C., Muesing, R., and Wilsom, P. K. Effect of 12 Weeks of Jogging on Serum Lipoprotein Levels of Middle-Aged Men. Med. Sci. Sport. Exercsie. 10:(abstr)55, 1978.

129. Weltman, A., Matter, S., and Stamford, B.A. Caloric Restriction and/or Mild Exercise: Effects on Serum Lipids and Body Composition. Am. J. Clin. Nutr. 33:1012-1009, 1980.

130. Widerman, P. M., and Hagan, R. D., Body Weight Loss in a Wrestler Preparing for Competition: A Case Report. Med. Sci. Sports. Exercise. 14:413-418, 1982.

131. Williams, P. T., Wood, P. D., Haskel, W. L., and Vranizan, K. The Effects of Running Milage and Duration on Plasma Lipoprotein Levles. J.A.M.A. 247:2674-2679, 1982.

132. Williams, R.S., Logue, E. E., Lewis, J. L., Barton, T., Stead, N. W., Wallace, A.G., and Pizzo, S.V. Physical Conditioning Auguments the Fibrinolytic Response to Venus Occlusion in Healthy Adults. N. Eng. J. Med. 302: 987-991, 1980.

133. Wilmore, J. H., Royce, J., Girandola, R. N., Katch, F. I., and Katch V. L. Body Composition Changes With a Ten Week Program of Jogging. Med. Sci, Sports. Exercise. 2: 113-117, 1970.

134. Wolf R. N., and Grundy, S. M. Influence of Weight Reduction on PLasma Lipoproteins in Obese Patients. Arteriosclerosis. 3:160-169, 1983.

135. Wood, P. D., Haskell, W. L., Blair, S. N., Williams, P. T., Krauss, R. M., Lindgren, F. t., Albers, J .J., Ho, P. H., and Farquhar, J. W. Increased Exercise Level and Plasma Lipoprotein Concentrations: A One-Year Randomized Controlled Study in Sedentary Middle-Aged Men. Metabolism. 32:31-39, 1983.

136. Wood, P. D, Haskell, W., Klein, H., Lewis, S., Stern, M. P., and Farquhar, J. W. The Distribution of Plasma Lipoproteins in Middle-Aged Male Runners. Metabolism. 25:1249-1257, 1976.

137. Wood, P. D., Haskell, W. L., Stern, M. P., Lewis, S., and Perry, C. Plasma Lipoprotein Distributions in Male and Female Runners. Ann. NY. Acad. Sci. 301:748-763, 1977.

138. Zauner, C. W., and Swenson, E. W. Physical Training and Physical Performance in Relation to Blood Lipid Levels and Pulmonary Function. Am. Corr. Ther. J. 21:159-165, 1967.

139. Zimmerman, J., Kaufmann, N. A., Fainaru, M., Eisenberg, S. Oschry, Y., Friedlander, Y., and Stein, Y. Effects of Weight Loss in Moderate Obesity on Plasma Lipoprotein and Apolipoprotein Levels and on High Density Lipoprotein Composition. Arteriosclerosis. 4:115-123, 1984.

140. Zuti, W. B., and Golding, L. A. Comparing Diet and Exercise as Weight Reduction Tools. Phys. Sportsmed. 4:49-53, 1976.

APPENDIX A

Fogarty International Center Conference on Obesity Recommended Weight in Relation to Height (14).

Height		Men		Women	
Feet	Inches	Average	Range	Average	Range
4	10	.....		102	92-119
4	11	.....		104	94-122
5	0	.....		107	96-125
5	1	.....		110	99-128
5	2	123	112-141	113	102-131
5	3	127	115-144	116	105-134
5	4	130	118-148	120	108-138
5	5	133	121-152	123	111-142
5	6	136	124-156	128	114-146
5	7	140	128-161	132	118-150
5	8	145	132-166	136	122-154
5	9	149	136-170	140	126-158
5	10	153	140-174	144	130-163
5	11	158	144-179	148	134-168
6	0	162	148-184	152	138-173
6	1	166	152-189	.....	
6	2	171	156-194	.....	
6	3	176	160-199	.....	
6	4	181	164-204	.....	

Height without shoes, weight without clothes. Adapted from the table of the Metropolitan Life Insurance Co.

APPENDIX B

Questionnaire

Personal Information Date _____

Name _____ Age _____ S.S. # _____

Address _____ Telephone # (home) _____

_____ (work) _____

_____ Occupation _____

Date of Birth _____ Place of Employment _____

Personal Physician _____ Location _____

Medical History - Have you ever had, or been told you have:

High Blood Pressure ..... Yes ____ No____

High Cholesterol ..... Yes ____ No____

Any Heart Trouble ..... Yes ____ No____

Disease of the Arteries ..... Yes ____ No____

Rheumatic Fever/Heart Murmur ..... Yes ____ No____

Varicose Veins ..... Yes ____ No____

Emphysema ..... Yes ____ No____

Bronchitis ..... Yes ____ No____

Asthma ..... Yes ____ No____

APPENDIX B CONTINUED

Diabetes ..... Yes ____ No____

Kidney Disease ..... Yes ____ No____

Arthritis ..... Yes ____ No____

Current Well-Being - Have you recently experienced:

Chest Pain/Discomfort ..... Yes ____ No____

Shortness of Breath ..... Yes ____ No____

Indigestion ..... Yes ____ No____

Blood in Urine ..... Yes ____ No____

Frequent Headaches ..... Yes ____ No____

Stiff or Painful Joints ..... Yes ____ No____

Orthopedic Problems ..... Yes ____ No____

Lower Back Pain ..... Yes ____ No____

Please list all current medications (include birth control pills)

Medication	Reason for taking	For how long?

APPENDIX B CONTINUED

Lifestyle

Do you currently smoke? Yes____ No____ If so, what____ How Much____

Have you ever quit smoking? Yes ____ No ____ How long ago? _____

How much alcoholic beverage do you consume in one week?

Type: Beer ____(# of cans) Wine ____(# of Glasses)

Hard Liquor ____(# of drinks)

Do you take part in any sort of regular physical activity?

If so what type? _____(walk, jog, bike, swim)

How many times per week? _____

How much time per day? _____

Have you ever experienced chest discomfort with exercise?

Yes ____ No ____

APPENDIX C

EASTERN ILLINOIS UNIVERSITY HUMAN PERFORMANCE LAB STATEMENT REGARDING INVOLVEMENT IN A RESEARCH PROJECT WEIGHT LOSS STUDY: JANUARY 1988 TO APRIL 1988

Effective weight loss has been achieved through several mechanisms Among the most popular have been manipulations of diet and exercise In an attempt to provide a better understanding concerning the contribution of these two interventions, the present research project is is being conducted.

Involvement in this project will require a commitment to a specified diet, exercise, or combined diet and exercise program for approximately 12 weeks. In addition to program adherence, individuals will be asked to participate in tests of cardiovascular fitness, blood lipid analysis, and body composition prior to, and immediately following their prescribed treatment. Cardiovascular fitness will be evaluated via the administration of a graded exercise test given by qualified personnel in the E.I.U. Human Performance Lab. All blood work including drawing and analysis will be done by Illinois Medical Laboratory INC. Finally, body composition by way of the skinfold technique will be assessed in the E.I.U. Human Performance Lab.

Though the possible benefits that accompany this project are quite evident, there are certain risks, particularly those which parallel exercise and exercise testing which should be noted. Those individuals who partake in the afformentioned activities run the risk of sustaining an injury, heart attack, stroke, or other cardiovascular complications which may include death.

At any time the participants are free to withdrawal from this project without questioning. Any data as a result of an individuals participation in this project will remain confidential and be presented in a research report in an

APPENDIX C CONTINUED

anonymous manner. Though questionnaires and personal interviews may become part of this study, each participant reserves the right to not answer to any specific items or questions.

I have carefully read the above statement and have had the opportunity to ask questions pertaining to the parameters of this study. By signing this document I do hereby freely and voluntarily consent to my participation in this research project.

_____ _____

Signature Date

APPENDIX D

WORKOUT LOG

Week of: _____

Name	Mon	Tue	Wed	Thur	Fri	Sat	Sun
JB (23)							
EB (23)							
SC (23)							
KC (24)							
RD (23)							
KP (23)							
LW (22)							
EW (22)							
TF (26)							
GH (22)							
JH (24)							
DM (23)							
KS (24)							
ES (24)							
AW (25)							
KW (25)							
PR (25)							
PG (25)							
JA (23)							

APPENDIX E

Food Record

Day _____

Date _____ Name _____

Breakfast Time: Food Amount	Lunch Time: Food Amount
Dinner Time: Food Amount	Other Time: Food Amount
Other Time: Food Amount	Other Time: Food Amount

APPENDIX F

Weight Graph

NAME PROGRESS TOWARD