AN ABSTRACT OF THE THESIS OF

Young-Soo Lee for the degree of Doctor of Philosophy in Human Performance presented on May 31, 1990.

Title: The Acute Effects of Moderate Intensity Circuit Weight Training on Lipid-Lipoprotein Profiles.

Abstract approved: Redacted for Privacy

Dr. John Patrick O'Shea

Few studies have examined the acute effects of resistive-type exercise on lipid-lipoprotein profiles. This study examined the acute effects of a single session of circuit weight training (CWT) on plasma lipid-lipoprotein profiles: triglycerides (TG), total cholesterol (TC), high density lipoprotein cholesterol (HDL-C), low density lipoprotein cholesterol (LDL-C), and the ratio of TC to HDL-C. The subjects in the study were 17 healthy, nonsmoking male university students, ages of 18-25 years, enrolled in weight lifting classes. Subjects were required to fast overnight (at least 12 hours) before CWT. Subjects repeated a four-station weight training circuit three times, with a resistance determined by their individual 3 repetition maximum (3-RM). The stations were bench press, parallel squat, leg extension, and seated row.

Blood samples were drawn from the antecubital vein at pre-CWT, completion of the 1st and 3rd circuits, and 15 min

post-CWT. All concentrations of plasma lipid and lipoprotein cholesterol were corrected for plasma volume changes. A repeated measures ANOVA was used to determine if significant differences existed among mean values for the dependent variables (i.e., levels of TG, TC, HDL-C, LDL-C, and TC/HDL-C ratio at specified time points).

Results of the study indicated that plasma TC and HDL-C levels were changed significantly during and following CWT. However, the change was not in the anticipated direction: Plasma TC and HDL-C levels were lower at completion of the 1st circuit of CWT ($p<0.05$). The ratios of TC to HDL-C were changed significantly, reflecting a decrease in HDL-C during CWT and a slight increase in HDL-C at 15 min post-CWT. Plasma TG and LDL-C levels were not changed significantly during CWT or 15 min post-CWT. It was concluded that apparent changes in lipoprotein patterns occur during short-term moderate intensity CWT and return to pre-CWT levels in a relatively short time.

The Acute Effects of Moderate Intensity Circuit Weight Training on Lipid-Lipoprotein Profiles

by

Young-Soo Lee

A THESIS

submitted to

Oregon State University

in partial fulfillment of
the requirement for the
degree of

Doctor of Philosophy

Completed May 31, 1990

Commencement June 1991

APPROVED

Redacted for Privacy

Professor of Exercise & Sports Science in charge of major

Redacted for Privacy

Chair of Department of Exercise & Sports Science

Redacted for Privacy

Dean of College of Health and Human Performance

Redacted for Privacy

Dean of Graduate School

Date thesis is presented May 31, 1990

Typed by researcher for Young-Soo Lee

ACKNOWLEDGEMENTS

I would like to express my deepest appreciation to Dr. John P. O'Shea for his professional guidance, encouragement, and patience during the course of this study. I would also like to express my appreciation to the members of my committee, Dr. Richard Irvin, Dr. Dow Poling, Dr. John Ruben, and Dr. Wayne Schultz, for their inspiration and advice. My sincere gratitude is also extended to Dr. Christian Zauner, Dr. Anthony Wilcox, and Dr. Terry Wood for their valuable comments and help and to Chris Quinn for her great effort on the data collection.

I am indebted to Dr. John Dunn and Dr. Christian Zauner, the former and the current Chair of the Department of Exercise and Sports Science, for their continuous financial support and advice during my graduate study at Oregon State University.

I must also acknowledge my parents for their neverending love and support, and my wife Jae-Sook and my children, Hae-Seung and Hae-Soo, for their patience, understanding, and emotional support during my study.

Finally, I thank my Lord Jesus Christ, through whom I have known the meaning of my life, for His everlasting love for me.

TABLE OF CONTENTS

APPENDICES

LIST OF FIGURES

LIST OF TABLES

The Acute Effects of Moderate Intensity Circuit Weight Training on Lipid-Lipoprotein Profiles

CHAPTER 1

INTRODUCTION

In the past decade a significant amount of research has been devoted to clarifying the relationship between lipids and the incidence of coronary artery disease (CAD). Although a direct cause and effect relationship between CAD and lipids has not been established, numerous epidemiological studies have indicated that the lipoprotein cholesterols are important indicators of risk for CAD (Brown & Goldstein, 1984; Kannel, 1983).

Early epidemiologic investigations suggested a positive relationship between total serum cholesterol level and the development of CAD (Chapman et al., 1957; Keys et al., 1958; Keys et al., 1963; Paul et al., 1963). Noma et al. (1983) found that patients with CAD have higher levels of cholesterol than subjects without CAD. Attention was focused on the clinical importance of lipid and lipoprotein levels after lipoprotein was identified as the carrier of cholesterol (Gofman, Young, & Tandy, 1966).

Researchers have demonstrated that low density lipoprotein cholesterol (LDL-C) is directly associated with

CAD (Goldstein & Brown, 1983; Rifkind, 1984; Tyroler, 1984; Aro et al., 1986; Blankenhorn et al., 1987). It was found that LDL-C has the greatest correlation to the severity of angiographically defined coronary atherosclerosis (Aro et al., 1986). Evidence suggests that LDL-C may play a key role in the transformation of arterial smooth muscle cells to proliferate excessively and to accumulate lipid, and thus lead to atherosclerotic plaque (Scow et al., 1980).

High density lipoprotein cholesterol (HDL-C) concentration has an inverse relationship with CAD (Castelli, 1986; Kannel et al., 1983), and with coronary mortality (Wilson et al., 1988). An increase in the proportion of HDL-C has been found to facilitate the removal of peripheral cholesterol and transport cholesterol from the plaque to the liver for further metabolism (Goldberg & Elliot, 1987; Gwynne, 1989).

Epidemiological studies in the 1970s suggested an inverse relationship between levels of physical activity and the incidence of CAD (Cassel et al., 1971; Paffenbarger & Hale, 1975). Since then, a number of cross-sectional and longitudinal studies have been undertaken in an attempt to clarify such a relationship. Evidence linking exercise to the prevention of clinical disease has been derived from large-scale surveys of groups having supposed differences in occupational activity, athletic participation, active leisure pursuits or overall lifestyle (Shephard, 1986).

It is generally accepted that endurance-trained athletes possess higher HDL-C concentration than their sedentary counterparts (Hartung et al., 1980; Hicks, MacDougall & Muckle, 1987; Huttunen et al., 1979). Both acute and chronic exposures to long-distance running and other forms of endurance exercise have been associated with increased plasma concentrations of HDL-C and decreased concentrations of LDL-C and very low density lipoprotein cholesterol (VLDL-C) (Griffin, Skinner & Maughan, 1988; Williams et al., 1983; Wood & Haskell, 1979). Endurance events have been related with increased lipoprotein lipase (LPL) activity (Kantor et al., 1984).

The majority of previous investigations examined the effects of aerobic type exercise training. Only a few investigations examined the influence of resistive type exercise, despite evidence suggesting that periodic intense muscular activity of the heavy work occupations reduces the incidence of coronary disease (Paffenbarger & Hale, 1975).

In recent years, resistive exercise in the form of weight training has become very popular throughout the world as a method of conditioning for many types of athletes. Further, the use of weight training in developing the well-being of non-athletes is increasing due to its ability to increase muscle mass, strength, and lower body fat (Goldberg, 1989). However, very little information is available concerning the acute effects of resistive training

on lipid-lipoprotein profiles and its influence on the CAD risk factors.

The major emphasis of previous investigation has been related to effects of chronic training on lipid and lipoprotein levels with epidemiologic or longitudinal designs. Most measurements have been made either before and after a certain period of training, or following completion of a sustained exercise event such as a marathon run or cross-country ski race (Lennon et al., 1983). Few studies have studied the acute or short-term effects of circuit weight training exercise on lipid-lipoprotein profiles.

This study was undertaken to provide more detail of the acute response of lipids and lipoprotein cholesterol to a single period of circuit weight training exercise.

Statement of Problem

The purpose of this study was to determine the acute response of total cholesterol, triglycerides, HDL-C, LDL-C, and the ratio of total cholesterol to HDL-C for the moderate intensity circuit weight training.

Hypotheses

1. There is a significant difference among mean total cholesterol (TC) levels obtained at pre circuit weight training, completion of the first and third circuits, and 15 min post circuit weight training (CWT).

H_0: $\mu_{A1} = \mu_{A2} = \mu_{A3} = \mu_{A4}$

H_1: $\mu_{A1} \neq \mu_{A2} \neq \mu_{A3} \neq \mu_{A4}$

where,

μ_{A1} = mean of TC level at pre-CWT.

μ_{A2} = mean of TC level at completion of the first circuit of CWT.

μ_{A3} = mean of TC level at completion of the third circuit of CWT.

μ_{A4} = mean of TC level at 15 min post-CWT.

2. There is a significant difference among mean triglyceride (TG) levels obtained at pre-CWT, completion of the first and third circuits, and 15 min post-CWT.

H_0: $\mu_{B1} = \mu_{B2} = \mu_{B3} = \mu_{B4}$

H_1: $\mu_{B1} \neq \mu_{B2} \neq \mu_{B3} \neq \mu_{B4}$

where,

μ_{B1} = mean of TG level at pre-CWT.

μ_{B2} = mean of TG level at completion of the first circuit of CWT.

μ_{B3} = mean of TG level at completion of the third circuit of CWT.

μ_{B4} = mean of TG level at 15 min post-CWT.

3. There is a significant difference among mean HDL-C levels obtained at pre-CWT, completion of the first and third circuits, and 15 min post-CWT.

H_0: $\mu_{C1} = \mu_{C2} = \mu_{C3} = \mu_{C4}$

H_1: $\mu_{C1} \neq \mu_{C2} \neq \mu_{C3} \neq \mu_{C4}$

where,

μ_{C1} = mean of HDL-C level at pre-CWT.

μ_{C2} = mean of HDL-C level at completion of the first circuit of CWT.

μ_{C3} = mean of HDL-C level at completion of the third circuit of CWT.

μ_{C4} = mean of HDL-C level at 15 min post-CWT.

4. There is a significant difference among mean LDL-C levels obtained at pre-CWT, completion of the first and third circuits, and 15 min post-CWT.

H_0: $\mu_{D1} = \mu_{D2} = \mu_{D3} = \mu_{D4}$

H_1: $\mu_{D1} \neq \mu_{D2} \neq \mu_{D3} \neq \mu_{D4}$

where,

μ_{D1} = mean of LDL-C level at pre-CWT.

μ_{D2} = mean of LDL-C level at completion of the first circuit of CWT.

μ_{D3} = mean of LDL-C level at completion of the third circuit of CWT.

μ_{D4} = mean of LDL-C level at 15 min post-CWT.

5. There is a significant difference among mean ratio of total cholesterol to HDL-C values obtained at pre-CWT, completion of the first and third circuits, and 15 min post-CWT.

H_0: $\mu_{E1} = \mu_{E2} = \mu_{E3} = \mu_{E4}$

H_1: $\mu_{E1} \neq \mu_{E2} \neq \mu_{E3} \neq \mu_{E4}$

where,

μ_{E1} = mean of ratio (TC/HDL-C) at pre-CWT.

μ_{E2} = mean of ratio (TC/HDL-C) at completion of the first circuit of CWT.

μ_{E3} = mean of ratio (TC/HDL-C) at completion of the third circuit of CWT.

μ_{E4} = mean of ratio (TC/HDL-C) level at 15 min post-CWT.

Delimitations

1. The subjects selected for this study were 17 healthy, nonsmoking male student volunteers, ages 18 to 25 years, enrolled in weight lifting classes at Oregon State University.

2. Subjects had not taken anabolic steroids or other medications that affect the blood lipids and lipoprotein profiles.

3. Blood samples were drawn during the moderate intensity (40-60% of the subject's three repetition maximum, 3-RM) circuit weight training.

Limitations

1. Diet, training, or social habits preceding this study (except for a 12-14 hour overnight fast before the first blood draw and CWT) were not controlled.

Definition of Terms

Atherosclerosis: A disease of the intima of the arteries, especially of the large arteries, that leads to fatty lesions called atheromatous plaques on the inner surfaces of the arteries. The earliest stage in the development of these lesions is believed to be damage to the endothelial cells caused by physical abrasion of the endothelium, by abnormal substances in the blood, or even by the effect of the pulsating arterial pressure on the vessel wall (Guyton, 1986).

Apolipoprotein: The protein constituents of lipoproteins grouped by function in four classes A, B, C,

and E. The A apoproteins (apo A-I, A-II, A-III, and A-IV) occur primarily in HDL and in lesser amounts chylomicrons; apo A-I is the activator of lecithin cholesterol acyltransferase (LCAT), which forms cholesteryl esters in HDL. The B apoproteins are recognized by specific cell-surface receptors that mediate endocytosis of lipoprotein particles. The C apoproteins (apo C-I, C-II, and C-III) occur in VLDL, HDL, and chylomicrons; apo C-II activates lipoprotein lipase, which hydrolyses triglyceride for transfer from VLDL and chylomicrons to tissue. Apo E occurs in all classes of lipoproteins; it may be involved in the conversion of VLDL to IDL and its clearance from the circulation (Taylor, 1988).

Coronary artery disease (CAD): Narrowing of the coronary arteries sufficiently to prevent adequate blood supply to the myocardium. The narrowing is usually caused by arteriosclerosis (Thomas, 1985).

Chylomicron: A particle of the class of lipoproteins responsible for the transport of exogenous cholesterol and triglycerides from the small intestine to tissues after meals. Chylomicrons are spherical particles with a core of triglycerides surrounded by a monolayer of phospholipids, cholesterol, and apolipoproteins (Taylor, 1988).

Lipids: A heterogeneous group of fats and fat-like substances characterized by being water-insoluble and being extractable by nonpolar solvents. The lipids, which are

easily stored in the body, serve as a source of fuel, are an important constituent of cell structure, and serve other biological functions. Lipids may be considered to include fatty acids, triglycerides, and waxes. Compound lipids comprise the glycolipids, lipoproteins, and phospholipids (Taylor, 1988).

Lipoprotein: The lipid-protein complexes in which lipids are transported in the blood; lipoprotein particles consist of a spherical hydrophobic core of triglycerides or cholesterol esters surrounded by an amphipathic monolayer of phospholipids, cholesterol, and apolipoproteins (Taylor, 1988).

High density lipoprotein (HDL): lipoprotein particles having density of 1.063-1.21 g/ml and diameters of 10-15 nm and divided into two subclasses, HDL_2 and HDL_3. HDL is thought to be the lipoprotein responsible for transport of cholesterol from extrahepatic tissue to the liver for excretion. It is synthesized by the liver as discoid nascent HDL particles lacking a lipid core. A core of cholesterol esters accumulates as cholesterol is transferred from cell membranes to the HDL particle and then esterified by lecithin-cholesterol acyltransferase (LCAT) (Taylor, 1988).

Intermediate density lipoprotein (IDL): lipoprotein having density of 1.006-1.019 g/ml. It is a transitional stage in the conversion of VLDL to LDL (Taylor, 1988).

Low density lipoprotein (LDL): lipoprotein particles having density of 1.019-1.063 g/ml and diameters of 17-26 nm. LDL is responsible for transport of cholesterol to extrahepatic tissues. It is formed in the circulation as VLDL (and possibly HDL) passes through the IDL stage, becoming LDL by gaining and losing specific apolipoproteins. It is taken up and catabolized by both the liver and extrahepatic tissues, by specific receptor-mediated endocytosis (Taylor, 1988).

Very low density lipoprotein (VLDL): lipoprotein particles having density of 0.95-1.006 g/ml and diameters of 28-75 nm. VLDL particles are synthesized by the liver, and their lipid core consists primarily of triglycerides with some cholesteryl esters. The triglycerides are transported to muscle and adipose tissue by the action of endothelial lipoprotein lipase. As the triglycerides are removed the particles lose most of their apolipoprotein C and become IDL, which either is taken up by the liver or becomes LDL (Taylor, 1988).

Total cholesterol (TC): The sum of the cholesterol carried by VLDL, LDL, and HDL (Sherwin, 1988).

Triglyceride (TG): A compound consisting of three molecules of fatty acid esterified to glycerol; it is a neutral fat synthesized in the liver from carbohydrates (Thomas, 1985).

Lipoprotein lipase (LPL): an enzyme of the hydrolase class that hydrolyses triglyceride in chylomicrons that stick to the endothelial wall, releasing fatty acids and glycerol. It causes hydrolysis of phospholipids, thus essentially removing the mass of chylomicrons from the circulating blood (Guyton, 1986).

Lecithin-cholesterol acyltransferase (LCAT): an enzyme of the transferase calls that catalyzes the formation of cholesteryl esters in HDL by transferring long-chain fatty acid residues from phosphatidyl choline to a sterol. It is secreted by the liver (Taylor, 1988).

Intensity: The magnitude of force or energy per unit (Webster's New Collegiate). The intensity of exercise reflects both the caloric requirements of the activity and the specific energy sources required. It can be expressed in several ways: (a) as calories consumed; (b) as a percentage of maximal oxygen consumption, and (c) as a particular heart rate or some percentage of maximum heart rate; or (d) in terms of multiples of the resting metabolic rate required to perform the work (Katch & McArdle, 1988). In aerobic exercise, the intensity is usually expressed as a percentage of maximal oxygen uptake, or a percentage of maximal heart rate. In resistive exercise, it refers to the actual amount of resistance lifted per repetition during a training session and is commonly expressed as a percentage of one repetition maximum (1-RM).

Three repetition maximum (3-RM): the amount of weight a subject can lift three repetitions completely.

Repetition: The number of times a dynamic or static contraction is repeated in a given exercise set.

Set: A number of repetitions performed without resting (O'Shea, Simmons, & O'Connor, 1989).

Circuit weight training: A method of resistive training which consists of a series of exercise performed in a continuous fashion with short rest periods between exercises. Each exercise normally involves 12 to 15 repetitions with a resistance of 40-60% of one repetition maximum (Fleck & Kraemer, 1988).

Ethylenediaminetetraacetic acid (EDTA): A chelating agent that binds calcium and heavy metal ions; used as an anticoagulant for blood specimens (Taylor, 1988).

Body mass index (BMI): The ratio of weight to height, expressed as $kg/(m)^2$ (Williams, 1990).

CHAPTER 2

REVIEW OF LITERATURE

The aim of this chapter is to establish a theoretical basis for lipids and lipoproteins as important factors related to the CAD and to provide a review of the most recent research concerning changes in lipid and lipoprotein levels during acute exercise. The review of related literature is presented under the following headings:

1. Lipoprotein Classification and Metabolism.
2. Lipoprotein and Atherosclerosis.
3. Mechanisms Involved in Exercise-Related Blood Lipid and Lipoprotein Changes.
4. Effects of Aerobic Exercise on Lipid-Lipoprotein Profiles.
5. Circuit Weight Training as a Method of Exercise in High risk Individuals.
6. Effects of Resistive Exercise on Lipid-Lipoprotein Profiles.

Lipoprotein Classification and Metabolism

Both endogenously synthesized lipids and dietary lipids are transported to various sites in the body by multimolecular complexes known as lipoproteins (Miller &

Gotto, 1982). Lipoproteins are composed of various percentages of apoproteins, triglyceride (TG), cholesterol, and phospholipids. The lipoproteins consist of five major classes and several subclasses based on their density and ultracentrification (Gotto, Pownall, & Havel, 1986):

1. Chylomicrons, which are synthesized in the gut and carry dietary TG and cholesterol, contain TG as their major lipid constituent.
2. Very low density lipoproteins (VLDL), which are synthesized in the liver, carry endogenously synthesized TG as well as cholesterol.
3. Intermediate density lipoproteins (IDL), which represent an intermediate in the conversion of VLDL to LDL by lipoprotein lipase, contain relatively less TG and cholesterol compared to VLDL.
4. Low density lipoproteins (LDL), which are the major carriers of cholesterol, contain a relatively low percentage of TG, but a very high percentage of cholesterol.
5. High density lipoproteins (HDL), which are much richer in protein and contain approximately one-half protein and one-half lipid by weight, are usually subdivided into at least two subclasses, HDL_2 and HDL_3. The HDL_2 subfraction is considered to be protective against CAD and progression of atherosclerosis (Wood & Stefanick, 1990).

The apoprotein portion of the lipoprotein molecule contains lipid-binding areas which have a specific role in the regulation of synthesis and catabolism of the lipoprotein complex (Goldberg & Elliot, 1987). The function of apoproteins in lipoproteins is not only structural but metabolic as well. Specifically, they function as enzyme activators or inhibitors and sometimes they may be as specific signals for cell receptors participating in the metabolic regulation (Voutilainen & Hietanen, 1982b).

Specific apoproteins are associated with certain lipids. Apoprotein A-1 is primarily associated with HDL-C, and is distributed among both HDL_2 and HDL_3 subfractions. Apoprotein A-II, also found with HDL-C, has a greater association with HDL_3, whereas apoprotein B makes up approximately 95% of the apoprotein content of LDL (Natio & Galen, 1983).

Lipids and apoproteins are exchanged continuously between the lipoprotein particles and cells. In this exchange lipoprotein lipase (LPL), hepatic lipase (HL), and lecithin cholesterol acyltransferase (LCAT) play a role in the metabolism of HDL (Dufaux et al., 1982; Goldberg & Elliot, 1987).

Chylomicrons are synthesized by the small intestine in response to the absorption of dietary fat (Illingworth & Connor, 1985). The delipidation of chylomicrons and VLDL results in the liberation of TG, cholesterol, phospholipids,

and apoproteins (Eisenberg, Chajeck & Deckelbaum, 1982). Subsequently, chylomicrons and VLDL remnants are formed. The chylomicron remnants are recognized by specific hepatic receptors that facilitate their rapid removal from plasma (Illingworth & Connor, 1985). The VLDL remnant known as IDL undergoes further delipidation and forms LDL (Eisenberg, Chajeck & Deckelbaum, 1982).

The enzyme, LPL, can enzymatically act on VLDL in an intermediary step of IDL formation to form LDL. LPL may promote transfer of lipids from chylomicrons and VLDL to HDL for potential catabolism (Goldberg & Elliot, 1987). The LDL particle is removed from the plasma by binding to high affinity LDL receptors located in the liver and extrahepatic tissue cells. (Brown & Goldstein, 1983). In functional terms, LDL subserves a major role in the transport of cholesterol from the liver to extrahepatic tissues (Illingworth & Connor, 1985).

HDL particles appear to be synthesized in the liver and the intestinal epithelial cells, and as a product of TG-rich lipoprotein hydrolysis (Haskell, 1984). The composition of HDL lipids undergoes constant change in the circulation with the generation of cholesterol esters from free cholesterol and lecithin via lecithin cholesterol acyltransferase (LCAT). LCAT, a key enzyme in lipoprotein metabolism, is believed to play a central role in a reverse cholesterol transport function of HDL facilitating the transfer of

cholesterol from the peripheral cells onto HDL, which transports it to the liver for ultimate degradation and elimination (Tsopanakis et al., 1988). This enzyme transfers the liberated cholesterol, phospholipids, and apo-A from the surface of the TG-rich chylomicrons and VLDL, to the circulating nascent HDL (Eisenberg, Chajek, & Decklbaum, 1982; Haskell, 1984). LCAT converts free cholesterol to cholesterol esters, which enter core compartments of the nascent HDL, converting the disk-like structures to spheres (Haskell, 1984). This HDL particle is capable of gathering more free cholesterol from peripheral cells to further decrease its density and became HDL_2. Hepatic lipase located in the liver cells eventually picks up the HDL_2 particle and either catabolizes it completely or removes part of its cholesterol and the lipoprotein enters the circulation as HDL_3 (Nikkilä et al., 1982).

The major site of degradation of HDL probably is the liver, achieved by action of the hepatic lipase enzyme, but it is possible that some HDL particles are catabolized in other tissues, especially in smooth muscle cells or the kidney (Haskell, 1984).

Lipoproteins and Atherosclerosis

Schwartz et al. (1989) described the atherosclerosis as the result of a multiplicity of interactive cascades among

injury stimuli and the healing responses of the arterial wall, occurring concurrently within a hyperlipidemic environment.

The basic mechanisms in the development of atherosclerosis are far from clear. One of the major theories, the lipid hypothesis, presumes that atherosclerosis is connected with the accumulation of cholesterol in the vascular walls. This theory is based on experimental studies showing that dietary manipulation may produce atheromatous lesions in animal, and on human epidemiological studies showing the relationship between the incidence of atheromatous diseases such as CAD and elevated blood lipids (Voutilainen & Hietanen, 1982a).

During the past decade, results from therapeutic trials have indicated that control of hypercholesterolemia does result in lower cardiovascular risk (Bilheimer, 1988; Expert Panel, 1988). The first evidence linking cholesterol concentration in plasma to human atherosclerosis came from case studies showing that CAD patients had higher concentrations of cholesterol in plasma than did the controls (Dawber et al., 1957). Subsequent longitudinal epidemiologic studies, such as the Framingham Study, found that the concentration of cholesterol predicts the risk of CAD.

Levy (1985) suggested the following facts as support for the lipid hypothesis: (a) An ingredient found in all

atheromata is, almost by definition, cholesterol and its ester; (b) In animal experiments, investigators can induce atherosclerosis by producing hypercholesterolemia; (c) Genetic studies show that patients who inherit elevated levels of LDL, or reduced levels of HDL, often develop premature, severe coronary disease. In contrast, patients who have inherited higher levels of HDL or lower levels of LDL (hyperalpha- or hypobeta-lipoproteinemia) have a decreased incidence of CAD; (d) Metabolic studies show that all the cholesterol in the atheromata comes from circulating lipoproteins and is not made by the vessel itself; and (e) Cross-sectional and cross-cultural epidemiologic studies reveal that the higher the levels of total cholesterol (TC) and LDL-C, the greater risk of CAD.

It seems that LDL-C plays a key role in transformation of arterial smooth muscle cells to proliferate excessively and to accumulate lipids. Evidence suggests that LDL-C may cause direct damage to arterial endothelium, making arterial smooth muscle cells to proliferate and accumulate lipids, and this damage leads to atherosclerotic plaque formation (Miller et al., 1976; Scow et al., 1980). It might be that the arterial smooth muscle cells have receptor for apoprotein B present in LDL, initiating the process of accumulating LDL-C in the arterial wall (Benditt & Gown, 1980).

Ross (1986) suggested two pathways that may lead to formation of intimal smooth-muscle proliferative lesions. One pathway, demonstrated in hypercholesterolemia, involves monocyte and possibly interactions which may stimulate fibrous-plaque formation by growth factor release from the different cells. The second pathway involves direct stimulation of endothelium, which may release growth factors that can induce smooth-muscle migration and proliferation, and possibly autogenous growth factor release by the stimulated smooth muscle cells.

One method of testing the atherogenicity of lipoprotein cholesterol is to examine its effect on cultures of vascular-wall cells in vitro (Mahley, 1983). Slotte, Chait, and Bierman (1988) found that incubation of cultured arterial smooth muscle cells derived from monkey with large concentrations of LDL-C resulted in a net increase in cell cholesterol and cholesteryl ester mass that was dependent on LDL concentration and time of incubation. In their study, about 40% of the accumulated cholesterol mass was derived from surface transfer of LDL free cholesterol. The authors suggested that mechanisms other than the LDL-C receptor pathway are likely to play important roles in cholesterol accumulation and foam cell formation in arterial smooth muscle cells.

In human plasma the most effective cholesterol removing substance is HDL (Goldstein & Brown, 1982). The removal of

cholesterol from the peripheral cells by HDL was first suggested by Glomset (1970) and demonstrated in vitro by Fielding et al. (1983). Glomset hypothesized that HDL served as the substrate for the enzyme LCAT in the esterification of free cholesterol. When this reaction occurs, free cholesterol is esterified by LCAT, and bound to the HDL particle. The lipid content of the HDL particle increases and it becomes HDL_2. Hepatic lipase consequently takes up the HDL_2 particle and either catabolizes it completely or removes part of its cholesterol and releases the lipoprotein in the circulation as HDL_3 (Nikkilä et al., 1982). This causes the net elimination of cholesterol from peripheral cells. It has been suggested that the protective action of HDL against CAD may rest in the concentration of HDL_2 particle (Gotto, Pownall, & Havel, 1986; Miller, 1980).

Gwynne (1989) conceptually divided the reverse cholesterol transport into five steps: cell cholesterol efflux, cholesterol trapping by esterification, cholesterol transfer, hepatic uptake, and biliary excretion. According Gwynne, cholesterol is removed from macrophages and other cells within the arterial wall by an HDL-mediated process. Evidence indicated that apolipoprotein A-I only or pre-β-migrating HDL particles are particularly effective in the removal process. Cholesterol taken up by HDL is prevented from re-entering the cell by esterification.

Two mechanisms by which HDL-C may reduce or retard development of atherosclerosis have been proposed. The first is inhibition of LDL-C uptake by cells of the artery wall (Carew et al., 1976) and the second is facilitated reverse cholesterol transport from cells of the artery walls (Gwynne, 1989).

The exact cellular and molecular processes by which lipoprotein cholesterols are linked to atherogenesis have not yet been identified. However, according to McGill (1988), the evidence is sufficient to conclude that lipoprotein cholesterols are major intervening variables involved in the chain of causation of atherosclerosis.

Mechanisms Involved in Exercise-Related Blood Lipids and Lipoprotein Changes

Due to the link between TG catabolism and formation of HDL-C subfraction mediated by the enzyme, LPL, the response of this enzyme to exercise has been studied. Increases in LPL activity have proven to be the primary mechanism involved in exercise-related blood lipid changes (Haskell, 1984). Low plasma LPL activity has been correlated with increased CAD (Breier et al., 1985). Exercise has been found to cause an increase the activity of LPL, the enzyme which metabolizes TG to free fatty acids (Goldberg & Elliot, 1987). Higher levels of LPL activity have been found after a

prolonged training session (Thompson et al., 1988; Kantor et al., 1984). LPL activity may be associated with the increased utilization of TG as fuel for exercise (Vessby et al., 1985). Moderate-intensity exercise training for 15 weeks produced a significant rise in adipose tissue and post-heparin LPL activity in previously sedentary men, 56% and 33% respectively (Peltonen et al., 1981). Prolonged exercise bouts have resulted in increased LPL activity, such that it nearly doubled in trained men after a 42 km race (Kantor et al., 1984). Sady et al. (1986) found that 46% of post-heparin LPL activity was increased after marathon running with elevated HDL-C, in which the elevated HDL-C was primarily due to an increase in the HDL_2 subfraction. Using biopsy techniques of fat utilization, Hurley et al. (1986) reported that trained individuals have increased lipolysis of muscle TG when compared to untrained subjects during exercise of comparable intensity.

The idea that exercise training increases HDL-C concentration by means of an increase in LPL activity, however, needs more studies to be proven. It may be that the higher LPL activity and the higher HDL-C values in endurance athletes are due, at least in part, to higher proportions of slow-twitch skeletal muscle fibers, which contain a greater density of LPL (Jacobs, Lithell & Karlsson, 1982). Different levels of LPL activity have been found in the different types of muscle fibers existing in heart muscle. Slow-twitch

red skeletal muscle fibers had the largest activity of LPL; fast-twitch white muscle fibers had the least activity, while the fast-twitch red fibers had moderate activity (Kotlar & Borensztajn, 1977).

The biochemical mechanisms that control the skeletal muscle LPL response to exercise are not fully understood. Many of the physiologic adaptations that occur in response to exercise are associated with changes in neuroendocrine control of specific cell and tissue functions (Williams, 1985). Epinephrine excretion rates have been reported to account for about 70% of the change in muscle LPL activity in response to vigorous exercise (Lithell et al., 1981). The use of beta-adrenergic blocking drugs reduces lipid clearance and increases plasma TG concentration, suggesting a catecholamine-mediated influence on LPL (Day, Metcalfe & Simpson, 1982).

In addition to LPL, HL and LCAT changes may contribute to an increase in HDL-C concentration with exercise. HL activity appears to play a role in HDL-C catabolism by promoting plasma clearance of HDL-C (Eisenberg, 1984), and LCAT has an important role in formation of mature HDL-C (Goldberg & Elliot, 1987). A decrease in HL activity and an increase in LCAT may have a role in exercise-related HDL-C concentration. HL activity has been found to be lower in more active men, with a significant negative correlation resulting between HDL-C concentration and HL in highly

trained runners (Herbert et al., 1984; Williams et al., 1986). The role of HL is not well understood, but it may be involved in cholesterol removal from HDL and LDL as they pass through the liver (Haskel, 1984).

The enzyme LCAT catalyzes the transfer of fatty acids in plasma from lecithin to cholesterol in the production of HDL (Haskell, 1984). An increase in LCAT found in moderate training of young man athletes (Marniemi et al., 1982) might be the cause of a corresponding increase in the concentration of plasma HDL-C. Marniemi et al. reported that the increase in LCAT activity after training was correlated with increases in both plasma HDL-C, and TG concentrations. An increase in LCAT activity was found in 19 healthy men after a 15-week aerobic training program (Peltonen et al., 1981).

There is speculation that the regulation of HDL-C levels may be related on the phosphorylation and dephosphorylation of enzymes involved in HDL synthesis or degradation. Hooper and Scallen (1984) stated that the high HDL-C levels are a reflection of an increased catabolic state during which phosphorylations of HDL-related enzymes occur. However, additional research is needed to clarify the enzymatic activity changes induced by exercise that may provide favorable lipid and lipoprotein profile alteration.

Effects of Aerobic Exercise on Lipid-Lipoprotein Profiles

Aerobic exercises such as swimming, walking, jogging, and cycling involve large muscle groups in dynamic and oxygen dependent movements (Goldberg, 1989). Aerobic training directly affects the functional capacity of the cardiovascular, endocrine, and musculoskeletal system by an increase in $\dot{V}O_2$ max, decrease in myocardial oxygen demand, and increase skeletal muscle oxidative enzyme activity and capillarization to augment peripheral oxygen extraction, substrate metabolism, and circulatory function (Goldberg, 1988; Lakatta et al., 1987). Improvements in glucose tolerance, insulin sensitivity, and lipid-lipoprotein profiles and reductions in blood pressure usually accompany the physiological adaptations to aerobic exercise (Goldberg, 1989).

It has been suggested that regular aerobic type of physical activity increases the concentration of HDL-C (Farrell, Maksud, & Pollock, 1982; Schnabel & Kindermann, 1982; Wood & Haskell, 1979).

Several cross-sectional studies have demonstrated that endurance-trained individuals are characterized by a less atherogenic lipid profile than their sedentary counterparts. These studies dealt with elite long distance runners (Martin, Haskell, & Wood, 1977; Wood et al., 1976), competitive cross-country skiers (Enger et al., 1977), and

middle-aged male joggers (Penny et al., 1982). They showed that highly active individuals exhibit higher concentrations of HDL-C than their matched sedentary counterparts.

Penny et al. (1982) found that marathon runners and joggers have significantly higher values of serum HDL-C and HDL-C/TC ratios, compared to inactive subjects. No significant differences were found between the marathon runners and joggers. The authors concluded that middle-age men who take part in a long-term regular running program maintain HDL-C levels and HDL-C/TC ratios that are associated with lower risk of CAD.

Macek et al. (1989) studied the difference in coronary heart disease risk factors in trained and untrained adolescents. The level of TC was the same in both groups, but the level of HDL-C and its lipoprotein subfractions apolipoprotein (Apo-A) were higher, and LDL-C, apolipoprotein (Apo-B) and TG were significantly lower in the trained experimental group. The experimental group was comprised of 29 male and 26 female high school students who trained 5 hours per day in swimming and running. The intensity of training was controlled by monitoring heart rate, which was maintained between 140 and 200 beats per minute. Macek et al. stated that the experimental groups had a more beneficial lipoprotein profile, and a lower level of behavioral and physical coronary risk factors, than the control group.

Clarkson et al. (1981) studied the differences of serum TC, and HDL-C in young adult weight lifters, runners, and untrained subjects. The lipid profile of the weight trained athletes did not differ from controls. However, runners had significantly lower TC, and lower TC/HDL-C ratios. Based on these findings, Clarkson et al. concluded that persons involved in aerobic exercise demonstrate beneficially lower TC/HDL-C levels than those involved in anaerobic exercise programs.

The results from longitudinal training studies dealing with lipids and lipoproteins have been inconsistent. Whereas some investigators have reported that endurance exercise training significantly increased HDL-C concentrations (Peltonen et al., 1981; Farrell & Barboriak, 1980; Heath et al., 1983; Hespel et al., 1988), others have failed to show significant changes in HDL-C concentrations (Allison et al., 1981; Lipson et al., 1980; Raz, Rosenblit, & Kark, 1988; Williford et al., 1988).

Lopez et al. (1974) reported a significant increase in the HDL-C and a significant decrease in LDL-C concentrations in 13 male medical students following 7 weeks of aerobic exercise training. Myhre et al. (1981) reported that increases in plasma HDL-C concentrations of male subjects involved in cross-country skiing were related to the intensity and the duration of their training. Both high intensity-short duration, and low intensity-long duration

skiing induced favorable changes in plasma HDL-C concentrations, but the effect of the low intensity-long duration training was more favorable.

Blumenthal et al. (1988) studied the effects of the exercise training in the subjects with recent acute myocardial infarction. Forty-five patients were randomly assigned to either a high-intensity (65-75% $\dot{V}O_2$ max) or low-intensity (<45% $\dot{V}O_2$ max) exercise group. After aerobic training, three sessions a week for 12 weeks, both groups experienced a significant increase in HDL-C. However, there were no significant changes in serum TC or TG.

A study employing stationary bicycle exercise training showed favorable changes in sedentary men (Thompson et al. 1988). Subjects rode stationary bicycles 1 hour daily, 5 days a week for 14 weeks, and 4 days a week thereafter for a total of 32-48 week of training. HDL-C increased 5 ± 3 mg/dl, and TG decreased 19 ± 23 mg/dl after 14 weeks, but there were no additional changes with continued training. TC, LDL-C, and apolipoprotein A-I and A-II concentrations at the end of study were not different from the initial values.

Raz, Rosenblit, and Kark (1988) conducted a 9-week submaximal aerobic training study with 55 healthy sedentary nonsmoking and nonobese male subjects who had low plasma HDL-C concentration (<40 mg/dl). The researchers reported no significant difference between the exercise group and nonexercising (control) group in TC, HDL-C, calculated

LDL-C, or in the HDL_2 and HDL_3 subfractions. TG levels were lower by 19 mg/dl in the exercise group compared to the control group.

No changes in HDL-C concentrations were reported in young males (Linder, Durant, & Mahoney, 1983), young adults and middle-aged males (Sedgwick et al., 1980) or untrained young adult females (Williford et al., 1988) following aerobic exercise programs lasting from 8 to 18 weeks.

The acute effects of a single prolonged exercise session on the serum concentrations of TC, TG, HDL-C, and apolipoprotein A-I were investigated by Thompson et al. (1980). They studied 12 trained male runners participating in a 42 km footrace. Measurements were made 24 hours and 1 hour before the race, and 5 minutes, 1 hour, and 4, 18, 42, and 66 hours after the race. HDL-C concentration at 5 minutes and 18 hours following exercise was greater than prerace levels, but was not different from prerace levels at other time points. TC concentration did not change immediately after exercise, but significant reductions of 6-10% were found at 4-66 hours. The authors suggested that acute prolonged exercise lowers TC, but has little effect on serum HDL-C levels whether estimated as HDL-C concentration or apolipoprotein A-I concentration. They stated that the subjects in the study were well-trained men with initially high HDL-C levels (62 ± 13 mg/dl), and that the finding did

not imply that untrained individuals would respond similarly.

Lennon et al. (1983) studied the TC and HDL-C changes in 28 subjects (14 males, 14 females) during bicycle exercise for 40 min at the intensity of 55% of maximal oxygen uptake. Total and HDL-C levels were measured (and LDL-C calculated) at rest, 10, 20, 30, and 40 min of exercise, and 15 min post exercise. They reported a significant increase in HDL-C levels at 10 min of exercise for all subjects. This increase persisted at all time points throughout the exercise session, but declined by 15 min post exercise. On the other hand, there was a small, insignificant decline in LDL-C levels. It was concluded that apparent favorable changes in lipoprotein patterns occurred acutely, and were sustained during short-term moderate intensity, aerobic exercise, where the energy production to major extent is primarily a result of fatty acid oxidation.

Hicks, MacDougall, and Muckle (1987) reported significant acute increases in HDL-C and HDL Apoprotein A with 9-12 km running exercise on a treadmill at two intensities (60% and 90% of the subject's $\dot{V}O_2$ max). The greater increase was with the high-intensity exercise. Plasma-free fatty acids and TG did not differ between conditions, but lactate concentrations rose significantly during the high-intensity exercise. The researchers concluded that increases in HDL-C and HDL Apoprotein A can

occur with a relatively moderate exercise session and that the magnitudes of these increases are directly related to the exercise intensity.

Wirth et al. (1983) studied the effect of prolonged exercise on serum lipids and lipoproteins. The researchers monitored 26 men who played soccer continuously for 64 hours to establish a world's record, for acute changes in lipid metabolism. Serum TG levels increased at the beginning of exercise period, then declined. TC levels decreased uniformly during the exercise and LDL-C levels followed a similar pattern and decreased from 123 ± 18 mg/dl to 88 ± 10 mg/dl. In contrast, HDL-C increased 19% (from 40.3 ± 8 to 48.0 ± 8 mg/dl). Wirth et al. concluded that repeated acute prolonged exercise considerably lowers serum TG, TC, and LDL-C, but increases the HDL-C. These changes in lipid metabolism occur despite increased food intake and only minor changes in body weight and plasma volume.

The acute effects of prolonged walking and dietary changes on the plasma lipoprotein concentrations and HDL subfractions in six healthy male subjects were studied by Griffin, Skinner, and Maughan (1988). The plasma concentrations of lipoproteins were evaluated during a 37 km walk on each of four successive days. Statistical analysis revealed that diet can strongly influence changes in plasma lipoprotein concentrations during prolonged low-intensity exercise. With a high carbohydrate diet (85% of the calories

as carbohydrates), there were an increase in the concentration of VLDL-C and decrease in the concentration of HDL-C, due mainly to a decrease in HDL_3 cholesterol. In the absence of exercise, a high fat diet (75% of the calories as fat) produced a decrease in the concentration of VLDL-C and a small increase in HDL-C concentrations that arose largely from an increase in HDL_2 cholesterol. The mean plasma LCAT activity decreased with the high fat diet, but not with the high carbohydrate diet.

Cohn, McNamara, and Schaefer (1988) studied the plasma lipoprotein concentrations of 22 healthy subjects (9 men and 13 women) in fed and fasted states. Plasma lipoprotein cholesterol concentrations measured in the fed subjects differed significantly from those measured in the fasted subjects. The results of this study clearly reinforce the concept of analyzing plasma obtained after at least 12 hours fasting, for accurate assessment of an individual's risk for coronary heart disease.

The evidence obtained from studies concerning the effect of aerobic exercise on lipids and lipoproteins generally supports the premise that regular aerobic exercise may lead to the favorable alteration of plasma lipids and lipoproteins. However, investigators have not always controlled other factors that may influence the lipid and lipoprotein levels such as diet, changes in body weight,

seasonal variation, use of medication, plasma volume changes, and intensity and duration of exercise.

Circuit Weight Training as a Method of Exercise in High Risk Individuals

Conventional resistive training has long been considered to increase strength, power, and muscular endurance (Stewart, 1989). This type of resistive training employs a medium to heavy resistance with short bursts of exertion, followed by long rest interval with no requirements for a continuous high energy state, is categorized as an anaerobic physical activity. It contributes little or no improvement in the cardiovascular system (Hickson et al., 1980; Hurley et al., 1988). Circuit weight training was therefore developed in an attempt to bring a significant improvement in the function of cardiorespiratory system (O'Shea, 1976).

Circuit weight training (CWT) technique involves high repetitions using a moderate intensity exercise performed in a continuous fashion while moving from one station to another with minimal rest between stations (Gettman, 1979). The trainee exercises in short, all-out bursts of 45-60 sec in duration, then rests for 1 min or less between stations. A circuit may consist of 10-15 different exercises. The trainee may complete 2-3 circuits, depending on the exercise

prescription and objective for improvement (Stewart, 1989). The total number of repetitions executed during 45-60 sec should be set at minimum of 15 and a maximum of 20. The exercises are arranged in an alternating order between upper body and lower body (O'Shea, 1976).

CWT has been used extensively for athletes and healthy individuals. According to the guidelines of CWT for healthy adults reported by Gettman and Pollock (1981), the circuit should consist of 10-15 repetitions, using 40-50% of maximum strength, with 15-30 sec rest periods between sets. The circuit should contain 10-15 different exercises, be performed 2-3 times per session, take 20-30 min to complete, and be performed three times per week.

Cardiac patients have generally been excluded from heavy resistive exercise due to concern that increased exertional blood pressure response can lead to a markedly elevate rate pressure product and myocardial ischemia (Stewart, 1989). Mullins and Blomqvist (1973) reported large increases in left ventricular end-diastolic pressure and occurrence of complex ventricular arrhythmias with isometric handgrip exercise.

However, Butler et al. (1987) and Vander et al. (1986) have documented the safety of acute CWT sessions at 40-60% of maximal voluntary contraction in cardiac patients. No chest pain, ST-segment alteration, excessive dyspnea, and significant ectopy were reported in these studies. Left

ventricular wall motion abnormalities tended to improve with CWT exercise while they tended to worsen with treadmill exercise at 85% of maximum heart rate for 35 min (Butler et al., 1987). They concluded that CWT appeared to be as safe or safer than treadmill or bicycle exercise by all measures.

Featherston, Holly, and Amsterdam (1987) reported the heart rate and blood pressure responses to weight lifting at 40, 60, 80, and 100% of 1-RM. Peak heart rates in all lifts were lower than in a graded treadmill test. Peak systolic blood pressures were similar in the treadmill test and weight lifting while peak diastolic blood pressures were greater with lifts at 40, 60, and 80% of 1-RM than in the treadmill test. Peak rate pressure product (RPP = HR x SBP) was greater in the treadmill test than all lifts. From these results, they concluded that myocardial supply and demand balance appear more favorable with weight lifting than the treadmill test.

Ghilarduccii, Holly, and Amsterdam (1989) studied the effects of high resistance training in stable, aerobically trained, male cardiac patients. No signs or symptoms of ischemia or abnormal heart rate or blood pressure responses were observed during the 10 weeks of strength training program comprised lifting 80% of maximum voluntary contractions at 5 stations; quadriceps extension, bench press, standing biceps curl, hamstring curl and military press and performing 80% of the maximum number of sit-ups in

1 min. They suggested that resistive training at 80% of maximum voluntary contraction appears to be both safe and efficacious in stable, aerobically trained cardiac patients.

There is growing evidence that using moderate resistance with frequent repetitions is safe and beneficial (Kelemen,1989). Overall, there appears to be considerable benefit and minimal risk with CWT in cardiac and coronary prone individuals. The efficacy of CWT for improving strength and cardiovascular endurance should enable the individual to better perform both occupational and leisure tasks while decreasing the risk of injury (Stewart, 1989).

Effects of Resistive Exercise on Lipid-Lipoprotein Profiles

Paffenbarger and Hale (1975) provided the first evidence that periodic intense muscular activity may be associated with a protective effect against CAD. They reported that cargo handlers had 34% lower incidence of CAD than workers engaged in less physically demanding jobs.

Several subsequent cross-sectional or longitudinal studies comparing the lipid and lipoprotein profiles of resistive training athletes to those of endurance training athletes and to sedentary controls (Clarkson et al., 1981; Cuppers et al., 1982; Farrell, Maksud, & Pollock, 1982; Morgan et al., 1986), did not reach the same conclusion as Paffenbarger and Hale. The studies reported either low

levels of HDL-C or high levels of HDL-C among resistive training athletes.

In a cross-sectional study, Cuppers et al. (1982) compared the lipid and lipoprotein profiles among weight lifters, track and field athletes, and age-matched sedentary controls. They reported the track and field athletes had a trend for lower serum TC than weight lifters and sedentary men and that weight lifters had the highest concentrations of HDL-C when compared with the control group and with the track and field athletes.

Several cross-sectional studies reported that the lipid and lipoprotein profiles in resistive training athletes were less favorable (i.e., either low levels of HDL-C or high TC/HDL-C ratio) than those of endurance training athletes (Berg et al., 1980; Farrell, Maksud, & Pollock, 1982; Morgan et al., 1986). Berg et al. (1980) showed that HDL-C was significantly lower for weight lifters, shot putters, and discus throwers than for sedentary individuals. Farrell, Maksud, and Pollock (1982) found that HDL-C concentration was significantly higher in speed skaters, and the TC/HDL-C ratio was significantly higher in weight-lifters. Similar findings of low levels of HDL-C or high TC/HDL-C ratio have been observed in middle-aged powerlifters (Hurley et al., 1987), and female weight lifters (Morgan et al., 1986).

To further study lipid and lipoprotein profiles in different resistive training athletes, Hurley, Seals et al.

(1984) compared the lipid and lipoprotein profiles among bodybuilders, powerlifters, and runners. The researchers reported that the bodybuilders who trained with moderate resistance high repetitions (10-20) and took short rest intervals between exercise bouts had lipid and lipoprotein profiles which could be protective (i.e., high HDL-C and low LDL-C) against CAD. On the other hand, power lifters who trained primarily using heavy resistance with few repetitions (1-3) and long rest intervals between exercise bouts did not appear to have favorable lipid and lipoprotein profiles. Androgen use by 8 bodybuilders and 4 powerlifters lowered the values of both HDL-C and HDL_2-C and raised the value of LDL-C. Androgen use by strength-trained athletes may increase their risk for coronary heart disease. Based on these results, the researchers suggested that the training regimen of bodybuilders is associated with a more favorable lipid profile than the training regimen used by powerlifters. Bodybuilders used more dynamic forms of resistive exercise (e.g., circuit weight training) with moderate resistance, high repetitions, and short rest intervals. These more dynamic forms of resistive training are fueled by oxygen, and the continuous movement and isotonic contraction of muscle promotes the utilization of glucose and fat by muscles. This effect improves insulin sensitivity, lipid-lipoprotein profiles, and raises HDL-C

(Fleck & Lean, 1987; Harris & Holly, 1987; Hurley et al., 1988).

Longitudinal studies concerning the effects of resistive training on important risk factors for CAD have been conducted by several investigators (Blessing et al., 1988; Goldberg et al., 1984; Hurley et al., 1988; Johnson et al., 1983; Kokkinos et al., 1988; Kokkinos et al., 1989; Stone et al., 1982).

Stone et al. (1982) were the first to examine the effects of weight training on lipid and lipoprotein profiles. They reported decreases in LDL-C concentration, TC/HDL-C ratios, and body fat following 12 weeks of weight training. Johnson et al. (1983) studied 24 male volunteers, ranging in age from 24 to 69 years, who were divided into two groups, a weight training group and a control group. After 12 weeks of training, 3 days per week, 45-60 min at midday, the weight training group showed a significant increase in serum HDL-C and a significant decrease in TC and LDL-C concentrations. It was concluded that the weight training regimen produced alterations in the serum lipid profiles that might be beneficial in terms of risk for CAD.

Goldberg et al. (1984) trained previously sedentary females (8) and males (6) 3 times per week for 16 weeks of progressive resistance weight training, using machine gym equipment (Universal). The researchers reported increases in HDL-C level, decreases in LDL-C level and TC/HDL-C ratio in

male subjects following weight training. The women in the study demonstrated reduction in TC, LDL-C, and TG levels. However, the increase in HDL-C in women was not significant.

Hurley et al. (1988) studied the effect of 16 weeks of high-intensity resistive training on the risk factors for CAD. The training program resulted in a 13% increase in HDL-C, a 43% increase in HDL_2-C, a 5% reduction in LDL, and an 8% decrease in the TC/HDL-C ratio, despite no changes in $\dot{V}O_2$ max, body weight, or percent body fat. The supine diastolic blood pressure was reduced as a result of the training program. The authors suggested that resistive training could lower risk factors for CAD independent of changes in $\dot{V}O_2$ max, body weight, or body composition.

The mechanism by which weight training may improve lipid and lipoprotein profiles has not been fully understood. Alteration of body habitus among subjects engaged in resistive training, with decreased body fat and increased lean body mass, may be in part responsible for favorable lipid and lipoprotein profiles (Goldberg & Elliot, 1985). Possible correlation to hepatic lipase activity has been implied by Hurley, Hagberg et al. (1984).

Several training studies have indicated less favorable effect of resistive training on lipoprotein profiles. Blessing et al. (1988) reported no significant changes of serum lipid and lipoprotein levels of 13 healthy females (mean age 21 years) with 10 weeks of weight training. TG,

TC, HDL-C, LDL-C, and TC/HDL-C ratio did not change significantly. Body composition did not change with the training.

Kokkinos et al. (1988) compared the effects of 10 weeks of low and high repetition resistive training on lipid and lipoprotein profiles. They equated the total weight lifted by the two experimental groups to keep the total workload constant in both the high repetition, low resistance training group (14 to 16 repetitions maximum) and the low repetition, heavy resistance training group (4 to 5 repetitions maximum). No significant changes in plasma concentrations of TG, TC, HDL-C, HDL_2-C were observed in either group. They stated that the results of this study may be related to the age, low fat of the subjects, and their low initial cholesterol levels. The authors concluded that resistive training of low or high repetitions do not alter lipid and lipoprotein profiles when initial total blood cholesterol levels are low.

Kokkinos et al. (1989) studied the effect of 18 weeks of strength training on lipoprotein profiles and post-heparin lipase activities. There were no significant changes in $\dot{V}O_2$ max and percentage fat from training. Plasma concentration of TG, HDL-C, and LDL-C were unchanged. There were no changes in the activities of post-heparin lipoprotein lipase and hepatic lipase as a result of the training program. The researchers concluded

that strength training does not improve lipoprotein profiles or alter the lipases which regulate HDL-C and TG metabolism in individuals who are at risk for CAD.

Since study results have conflicted, it is not possible to make overall conclusions on the effect of resistive exercise on lipid and lipoprotein profiles. Hurley, Seals et al. (1984) suggested that some studies may have been confounded by the heterogeneity of the strength trained athletes examined whose training regimens varied in resistance, number of sets, number of repetitions, and duration of rest intervals. Adequate control for age, body fat, recent androgen administration, and diet was not established in these investigations. Other factors that have not been adequately controlled in studies on this topic include day-to-day variations of lipid-lipoprotein profiles and weight training-induced changes in plasma volume. Studies of the acute versus long-term effect of weight training need to be performed to provide a better understanding of the effects of resistive training on lipid-lipoprotein profiles (Hurley & Kokkinos, 1987). Few studies have monitored changes in lipid-lipoprotein profiles during resistive training exercise.

CHAPTER 3

METHODS AND PROCEDURES

The purpose of this study was to investigate the acute effects of moderate intensity CWT on lipid-lipoprotein profiles. This chapter describes subjects, circuit weight training, blood sampling procedures, analytical methods, experimental design, and statistical analysis.

Subjects

Seventeen healthy nonsmoking males, student volunteers, between the ages of 18 and 25 years, who were enrolled in weight lifting classes at the Oregon State University during the Winter term of 1990, were the subjects for this investigation. All subjects were familiar with the concept of CWT, had previous practice in going through the circuit, and had been participating in a weight lifting program at intermediate or advanced weight lifting classes. Volunteers who were on medications or other drugs such as niacin and gemfibrozil that may affect blood lipid-lipoprotein profiles were not accepted for participation.

The descriptive statistics for subject age, height, weight, and body mass index are presented in Table 1. Raw data for these variables are listed in Appendix A.

Table 1

Description of Subjects

	Range	Mean	SD
Age (yrs)	18 - 25	20.1	2.4
Height (cm)	172.7 - 185.4	181.2	3.8
Weight (kg)	68.9 - 106.6	81.6	10.7
BMI* (kg/m^2)	21.8 - 31.0	24.8	2.9

*Body Mass Index

The use of human subjects for this study was approved by the Committee for Protection of Human Subjects at Oregon State University. An informed consent form (Appendix B) and a medical history and health form (Appendix C) were completed by each subject after a group meeting to explain the testing procedures involved in the data collection for the study.

Circuit Weight Training

Subjects reported at the pre-set time to the weight training room in Langton Hall at Oregon State University between 6:30-10:30 a.m. Subjects were told to fast overnight (at least 12 hours) and not to do any exercise prior to reporting to the weight training room. Each subject had one warming-up of 7 repetitions in each lift with 50% of starting loads, and then performed the CWT workout.

Subjects repeated a four-station weight training circuit three times utilizing the following lifts; (1) bench press, (2) parallel squat, (3) leg extension, (4) seated row. These exercises were chosen because they are commonly used in a CWT program.

Bench press

The bench press develops primarily the pectoralis major muscle group in the chest (O'Shea, 1986; Williams, 1990). It also develops the deltoid in the shoulder and the triceps at the back of the arm.

The subject begins the bench press with the barbell at straight arms over the chest with the elbows locked in full extension, using an overhand grip, hands spread shoulder-width apart. The subject then carefully lowers the weight to the chest touching lightly and pushes upward returning the weight to its original starting position. The subjects were told not to arch the back to isolate the movement to the muscles of the shoulders and arms (see Figure 1).

Parallel squat

The squat holds a unique and unparalleled position of eminence in strength training and conditioning. It is unique and it stands supreme in its ability to develop the large muscle group in the body's power zone composed of

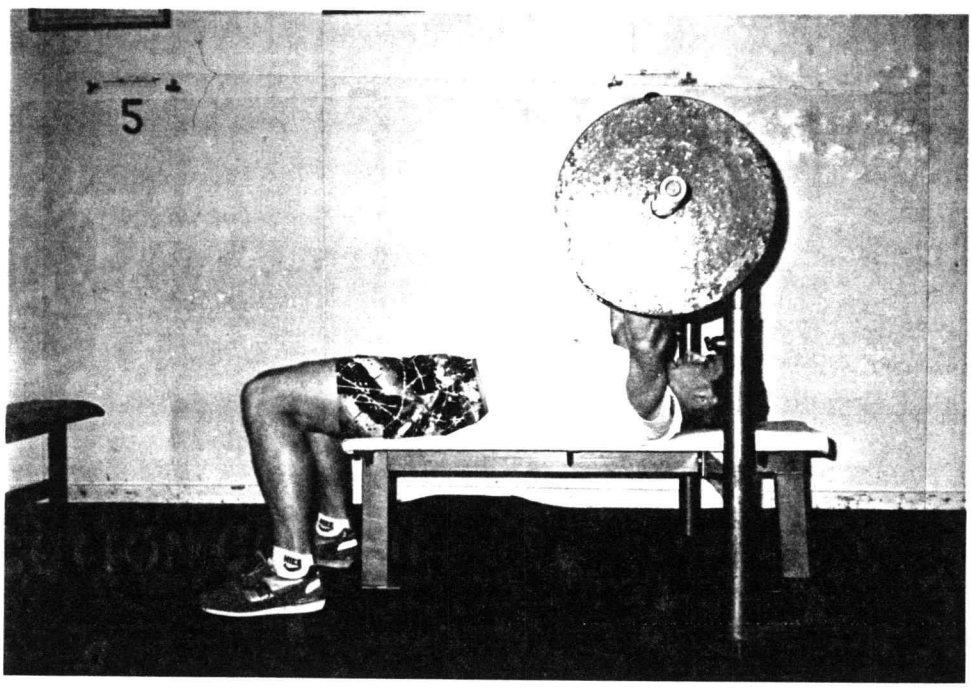

Figure 1
Bench Press

quadriceps, hamstrings, gluteus maximus, and the lower back (O'Shea, 1985).

In a biomechanical analysis of the parallel squat, the parallel squat involves three segments: ready position, descent, and ascent. At ready position, the bar is positioned across the shoulders with the load distributed over the mass of the back. Hands are positioned as close in on the bar as possible. Head is up; the chest is out. Feet are flat on the floor and spaced wider than shoulder width, with the toes turned out at approximately 30 degrees. During the descent, a subject commences to squat down in a slow and controlled manner to a position where the thighs are parallel to ground. The transition from the descent to the ascent commences with a powerful drive to accelerate out of the bottom position utilizing a strong quadricep extension. Once upward acceleration has started, a subject immediately begins to forcefully thrust the hips forward under the bar. A subject should maintain a tight torso throughout the ascent and not relax until knee lock is achieved in the standing position (see Figure 2).

Leg extension

The leg extension is a good exercise for strengthening the muscles of the upper leg, quadriceps femoris group (O'Shea, 1976). A subject sits on the edge of the leg extension machine with the knee flexed to 90 degrees and

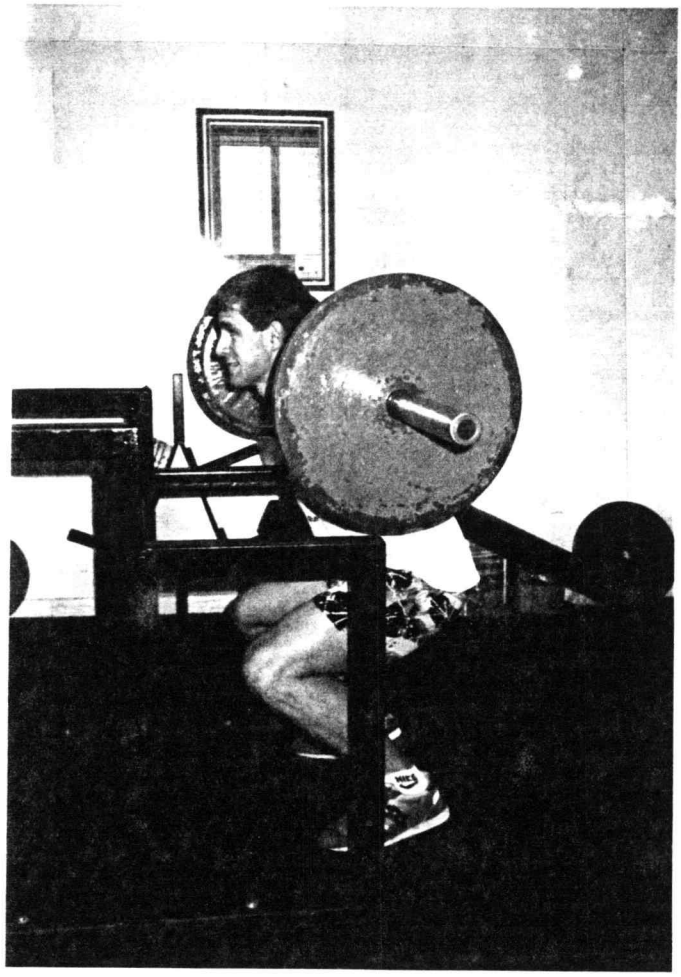

Figure 2

Parallel Squat

extends the leg outward at the knee joint until it locks into full extension, and then returns to the starting position (see Figure 3).

Seated row

The seated row develops deltoids, trapezius, biceps, and the muscles of the forearm. Since these muscles are utilized in many sports (e.g., canoeing, tennis, batting, golf-swing, handball, and swimming), the seated row is frequently included in training programs.

The subject takes a seated position with the legs fully or partially extended and braced against the machine. The subject grasps the bar with arms extended and pulls the bar toward the body until it touches the lower chest and then returns to the starting position (Stiggins & Allsen, 1989). The subject should keep the back straight during the exercise (see Figure 4).

Subjects exercised each lift for 1 min using a resistance determined by their individual 3 repetition maximum (3-RM) values. Subjects' 3-RM of bench press and parallel squat were measured using free weights. The 3-RM of leg extension and seated row were measured using an extension machine and a row machine (Iron Co., San Diego, CA). The resistances of the lifts at the first circuit were 50% of 3-RM for bench press, 60% of 3-RM for squat, 40% of 3-RM for seated row, and 40% of 3-RM for leg extension. The

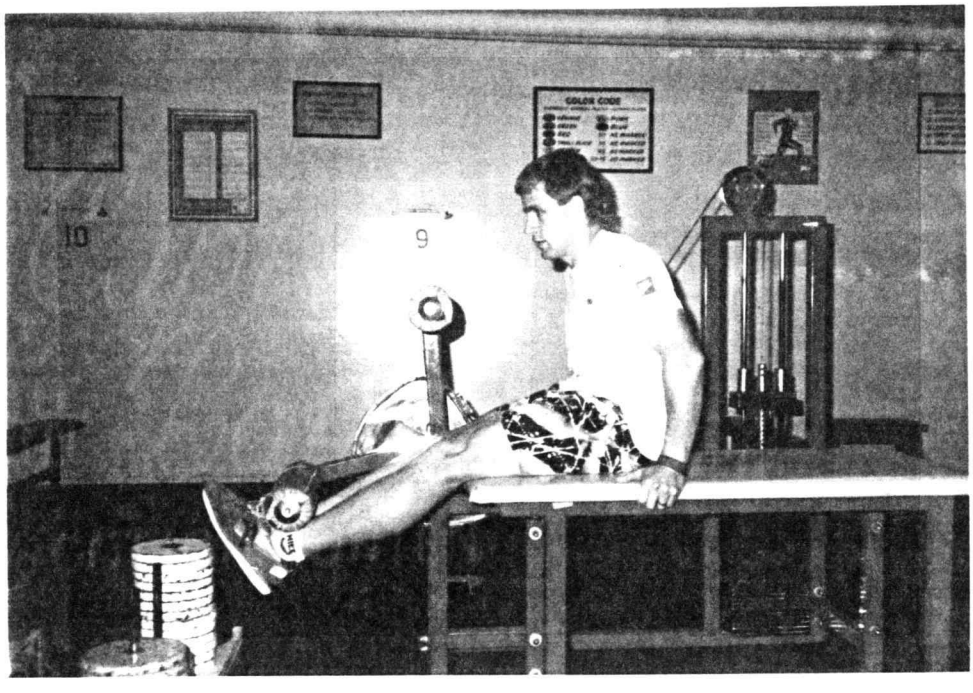

Figure 3

Leg Extension

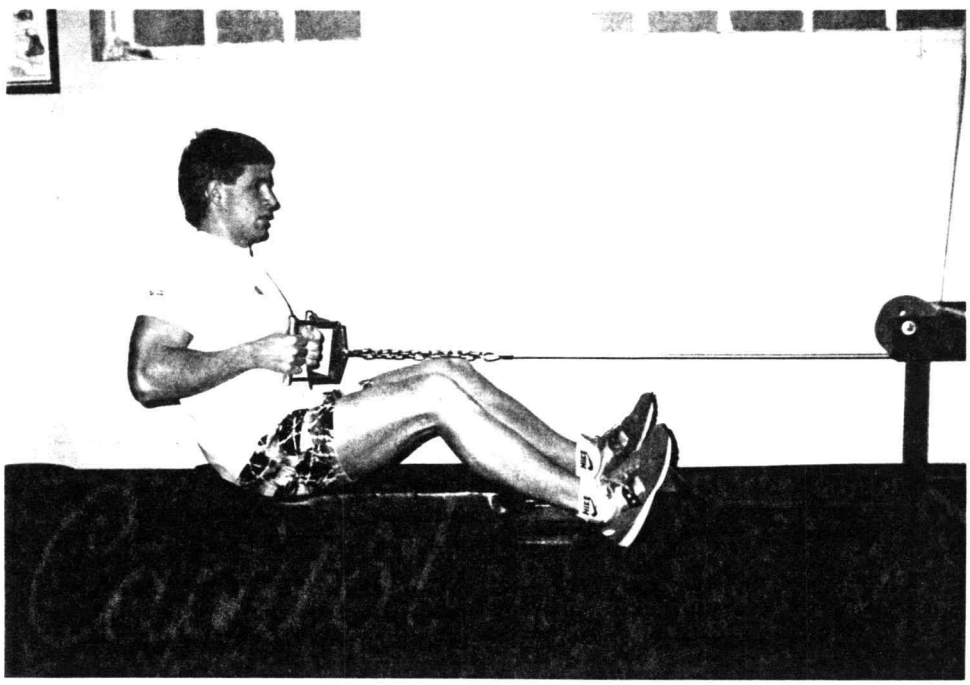

Figure 4

Seated Row

resistances of CWT at the second and third circuits were reduced each time by 10 pounds from the resistance used at the first circuit for bench press and seated row, 15 pounds for leg extension, and 20 pounds for parallel squat.

The average resistance and the 3-RM of each lift used in CWT are given in Table 2. Raw data for the 3-RM of the each lift are provided in Appendix D.

A 30-second rest was allowed between exercise stations. A 2 min rest was used to obtain blood samples between repeats of circuits. Subjects were allowed to walk about during the rest periods between repeats of circuits.

Table 2

The 3-RM and Resistance of Lifts Used in CWT

		Resistance (lbs)		
	3-RM	1st Circuit	2nd Circuit	3rd Circuit
Bench Press	184 ± 32*	85 ± 14	75 ± 14	65 ± 14
Parallel Squat	238 ± 50	132 ± 26	112 ± 26	92 ± 26
Leg Extension	160 ± 24	64 ± 10	49 ± 10	39 ± 10
Seated Row	158 ± 22	58 ± 10	48 ± 10	38 ± 10

* Mean ± SD

Blood Sampling Procedures

Blood samples were drawn before the warm-up period (pre-CWT), after completion of the first and third circuit, and at 15 min post-CWT. Approximately 8 cc of blood was drawn at the each time point from the antecubital vein, using a sterile disposable needle and a Vacutainer sterile evacuated blood collection tube with the anticoagulant EDTA (Becton Dickinson, Rutherford, NJ). A pressure dressing using sterile gauze and elastoplast brand tape (Johnson & Johnson) was applied following blood sampling. Blood samples were transferred within 5 hours to the Clinical Laboratory of the Good Samaritan Hospital (Corvallis, Oregon) for lipid-lipoprotein, hematocrit, and hemoglobin analysis.

Analytical Methods

Triglycerides

The concentration of TG was determined by an enzymatic procedure which uses lipase taken from a microorganism (Pseudomonas Species) to promote rapid and complete hydrolysis of TG to glycerol with subsequent oxidation to dihydroxyacetone phosphate and hydrogen peroxide. In the presence of peroxidase, the peroxide reacts with 4-aminophenazone and 4-chlorophenol to form an quinoneimine dye (Stein, 1986). TG concentration was determined by the intensity of the color formed using Boehringer Mannheim

Diagnostics Hitachi 704 Chemistry Analyzer (Boehringer Mannheim Diagnostics, Indianapolis. IN).

Total cholesterol

TC concentration was determined using the peroxidase and phenol-4-amino-phenazone indicator reaction (Trinder reaction) (Stein, 1986):

$$\text{Cholesterol esters} \xrightarrow{\text{Cholesterol esterase}} \text{Cholesterol} + \text{fatty acid}$$

All cholesterol esters present in plasma are hydrolyzed quantitatively into free cholesterol and fatty acids by microbial cholesterol esterase. In the presence of oxygen, free cholesterol is oxidized by cholesterol oxidase to cholest-4-en-3-one.

$$\text{Cholesterol} + O_2 \xrightarrow{\text{Cholesterol Oxidase}} \text{Cholest-4-en-3-one} + H_2O_2$$

The H_2O_2 reacts in the presence of peroxidase with phenol and 4-aminophenazone to form a quinoneimine dye.

The intensity of the color formed is proportional to the cholesterol concentration and measured using Boehringer Mannheim Diagnostics Hitachi 704 Chemistry Analyzer (Boehringer Mannheim Diagnostics, Indianapolis, IN).

HDL-C

The HDL-C concentration was determined by the phosphotungstate/magnesium with enzymatic quantitation. Chylomicrons, VLDL-C and LDL-C are precipitated by adding phosphotungstic acid and magnesium ions to the sample. Centrifugation (10 min at 4000 rpm) leaves only the HDL-C in the supernatant (McNamara & Schaefer, 1989). The HDL-C concentration is then determined by the method to measure TC (previously cited).

LDL-C

LDL-C concentration was calculated by the method of Friedewald, Levy, and Fredrickson (1972):

LDL-C = TC - (HDL-C + TG/5).

Hematocrit and hemoglobin

Hematocrit was measured in duplicate using a microhematocrit method. The raw hematocrit values were multiplied by the factor (0.96 x 0.91) to correct for trapped plasma and to convert venous hematocrit to whole body hematocrit, respectively (van Beaumont et al., 1973).

Hemoglobin was measured by the cyanomethemoglobin method (Fairbanks & Klee, 1986), using the Technicon Hematology System (Technicon Instruments Co., Tarrytown, NY). Raw data for hematocrit and hemoglobin are provided in Appendix E and F, respectively.

Calculation of the effects of plasma volume change on plasma concentration

The equation derived by Hill (1988) was used to quantify precisely the effects of a plasma volume change on the concentration of lipid and lipoprotein cholesterol in the plasma. The equation employed to calculate the expected concentration that would result mainly from the plasma volume change was:

$$C_X = C_B / (1 + \text{rel PV})$$

where,

C_X= expected concentration.

C_B= concentration measured before CWT.

rel PV= relative change in the plasma volume (expressed in decimal form).

The relative plasma volume change was determined from hematocrit and hemoglobin concentration using the equation of Dill and Costill (1974):

$$\% \text{ PV} = \{([Hb]_c/Hb_T) \times ((100 - Hct_T)/(100-Hct_c))-1\} \times 100$$

where,

Hct= hematocrit.

[Hb]= hemoglobin concentration.

c= refers to control values (before a change in PV occurs).

T= subsequent test values for Hct and [Hb].

The actual change of plasma constituent was calculated by subtracting the expected concentration from the measured concentration (van Beaumont et al., 1972):

$$C_A = C_M - C_X$$

where,

C_A= actual change of plasma constituent.

C_M= measured concentration.

C_X= expected concentration due to plasma volume change

The concentration after the intervention (i.e., during CWT and 15 min post-CWT) was determined by adding the actual change of plasma constituent to the concentration measured before CWT:

$$C_D = C_B + C_A$$

where,

C_D= concentration after the intervention (used in the statistical analysis of data).

C_B= measured concentration before CWT.

C_A= actual change of plasma constituent.

All concentrations of the plasma lipids and lipoprotein cholesterols were corrected according to the method previously cited, for each subject.

Experimental Design

The independent variable was the circuit weight training. The selected dependent variables were the concentrations of TC, TG, HDL-C, LDL-C, and the ratio of TC to HDL-C. The alpha level of 0.05 was used as the acceptable level of error probability for rejection of the null hypothesis.

Statistical Analysis

Statistical analysis of the data was performed using the BMDP Statistical Software Version VM/CMS 1987 (BMDP Statistical Software Inc., Los Angeles, CA) subroutine BMDP2V on the IBM 4381 Model 13 mainframe computer (International Business Machines Corp.) at the Computer Center of Oregon State University. A repeated measures analysis of variance (ANOVA) was used to determine if significant differences existed among mean values of the dependent variables obtained at pre-CWT rest and the specified time points on which the measures of dependent variables were acquired. The Bonferroni multiple comparisons procedure was employed to isolate the significant differences as suggested by Maxwell (1980).

CHAPTER 4

RESULTS AND DISCUSSION

The purpose of this study was to examine the acute effects of CWT on lipid and lipoprotein profiles. Seventeen healthy, nonsmoking male students enrolled in a weight training class at Oregon State University served as the subjects. Subjects repeated three times a four-station weight training circuit using the following lifts; bench press, parallel squat, leg extension, and seated row. Blood was drawn at pre-CWT, after completion of the 1st circuit and 3rd circuit, and 15 min post-CWT.

Results

The results of the study are presented in the following sections: (1) plasma volume change with CWT, (2) triglycerides, (3) total cholesterol, (4) HDL-C, (5) LDL-C, and (6) the ratio of total cholesterol to HDL-C. Raw data for the plasma concentrations of these variables are provided in appendices G, H, I, J, and K, respectively.

Plasma volume change with circuit weight training

The mean plasma volume decreased 11.9 ± 4.1% and 10.3 ± 3.8% at completion of the 1st and 3rd circuit of CWT, respectively. At 15 min post-CWT, mean plasma volume had returned to the pre-CWT level. Descriptive statistics for plasma volume changes during and 15 min post-CWT are presented in Table 3. Mean plasma volume changes during and 15 min post-CWT are presented in Figure 5.

Table 3

Means and Standard Deviations for Plasma Volume Changes

	PV1	PV2	PV3
Mean (%)	-11.9	-10.3	-1.2
SD	4.1	3.8	4.4

PV1= plasma volume change at completion of the 1st circuit
PV2= plasma volume change at completion of the 3rd circuit
PV3= plasma volume change at 15 min post-CWT

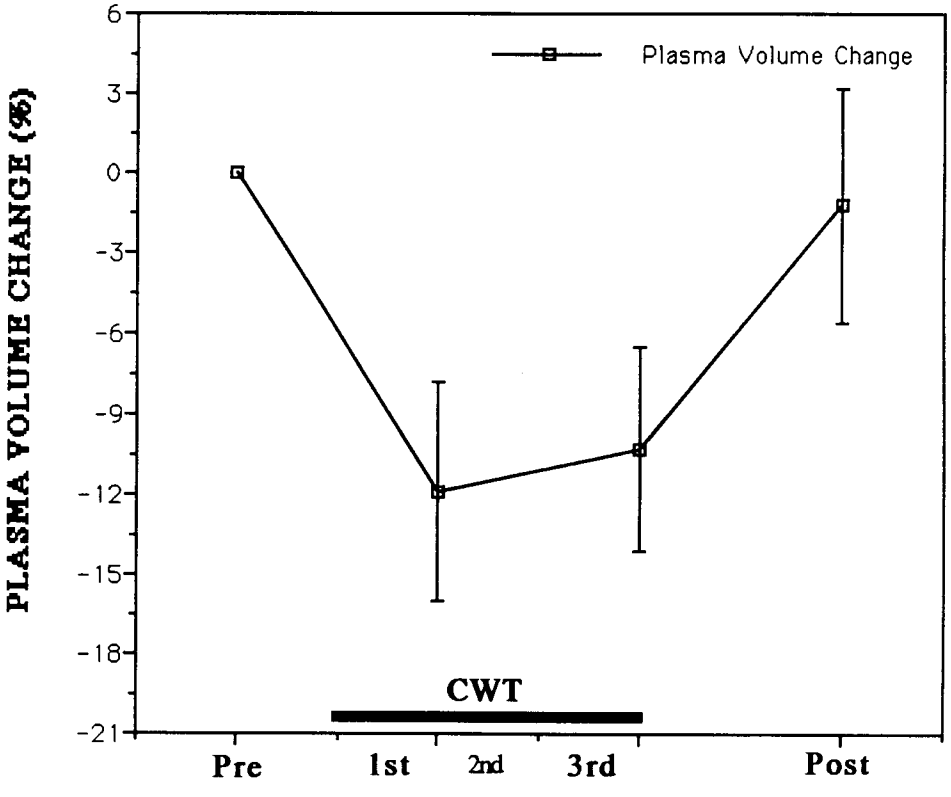

Figure 5

Mean Plasma Volume Changes

Triglycerides

The repeated measures ANOVA performed on the data for triglyceride (TG) levels showed that plasma TG levels did not change significantly during CWT or 15 min post-CWT. Table 4 presents the ANOVA summary table for plasma TG concentrations. Table 5 presents descriptive statistics for plasma TG concentrations measured at pre-CWT, completion of 1st and 3rd circuits, and 15 min post-CWT.

Total cholesterol

The ANOVA for the total cholesterol (TC) data yielded significant F values as shown in Table 6. Anderson's test of sphericity revealed that the sphericity assumption was not met. Greenhouse Geisser Probability (G.G.P.) was used to test the within subjects effects (Dunn & Clark, 1987). Descriptive statistics for TC concentrations are presented in Table 7. Comparison of means for TC concentrations at pre-CWT, completion of 1st and 3rd circuits, and 15 min post-CWT is shown in Figure 6. Figure 6 shows comparisons of means of the TC corrected and not corrected for plasma volume change.

Table 4

Analysis of Variance for Plasma Triglycerides

SOURCE	SS	df	MS	F	p
MEAN	449197.11	1	449197.11	130.03	0.0000
ERROR	55274.36	16	3454.64		
R(1)	40.01	1	40.01	0.36	0.5593
ERROR	1799.89	16	112.49		
R(2)	54.13	1	54.13	2.93	0.1062
ERROR	295.55	16	18.47		
R(3)	25.21	1	25.21	2.71	0.1190
ERROR	148.65	16	9.29		
R	119.35	3	39.78	0.85	0.4730
ERROR	2244.09	48	46.75		

Table 5

Means and Standard Deviations for Plasma Triglycerides

	TG1	TG2	TG3	TG4
Mean (mg/dl)	81.4	79.2	81.5	82.9
SD	29.9	27.2	30.2	32.1

TG1= plasma TG concentration pre-CWT
TG2= plasma TG concentration at completion of the 1st circuit
TG3= plasma TG concentration at completion of the 3rd circuit
TG4= plasma TG concentration at 15 min post-CWT

Table 6

Analysis of Variance for Plasma Total Cholesterol

SOURCE	SS	df	MS	F	p	G.G.P*
MEAN	1690975.97	1	1690975.97	657.67	0.0000	
ERROR	41138.69	16	2571.16			
R(1)	1.04	1	1.04	0.09	0.7743	
ERROR	197.44	16	12.34			
R(2)	98.42	1	98.42	9.85	0.0063	
ERROR	159.80	16	9.98			
R(3)	2.96	1	2.96	0.55	0.4692	
ERROR	86.40	16	5.40			
R	102.44	3	34.14	3.69	0.0180	0.034
ERROR	443.65	48	9.24			

* G.G.P.= Greenhouse Geisser Probability

Table 7

Means and Standard Deviations for Total Cholesterol

	TC1	TC2	TC3	TC4
Mean (mg/dl)	158.8	156.1	156.8	158.9
SD	25.4	25.1	24.9	26.4

TC1= plasma TC concentration pre-CWT
TC2= plasma TC concentration at completion of the 1st circuit
TC3= plasma TC concentration at completion of the 3rd circuit
TC4= plasma TC concentration at 15 min post-CWT

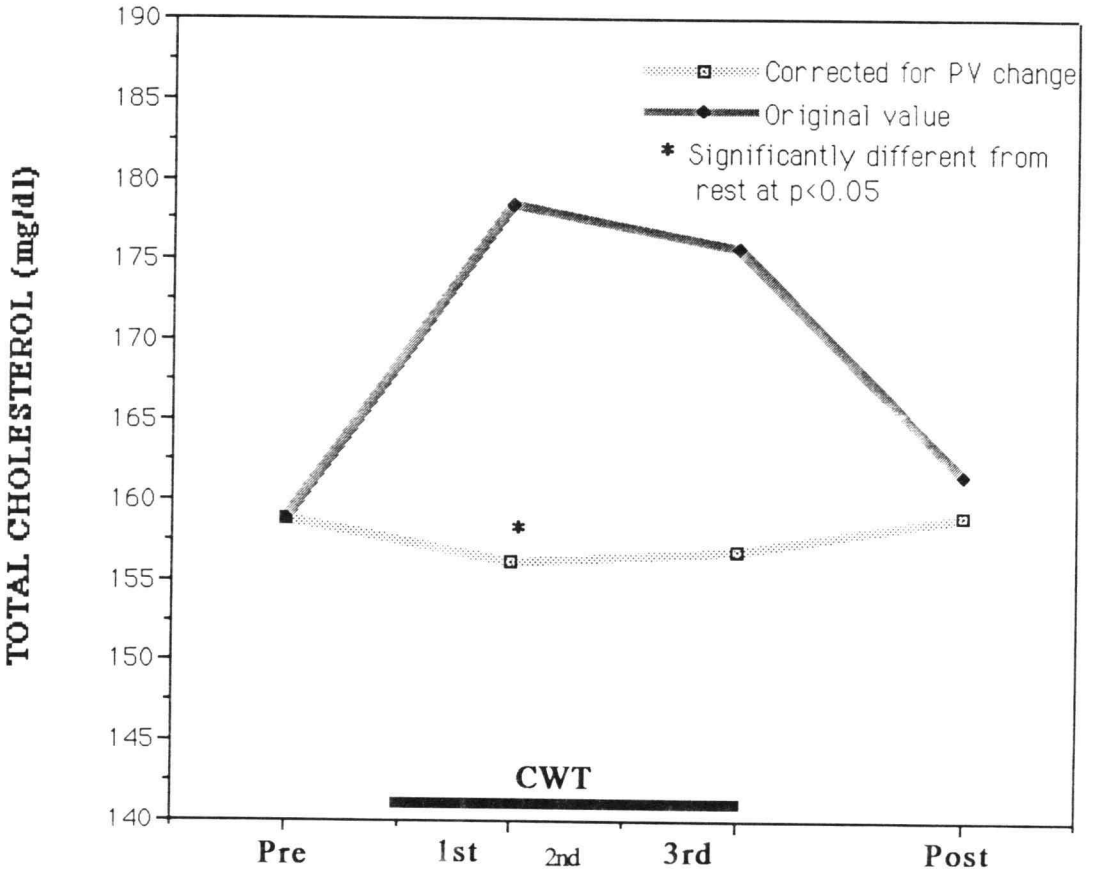

Figure 6

Mean Plasma Total Cholesterol Changes

The Bonferroni procedure was used to make multiple comparisons (Maxwell, 1980). The critical Bonferroni $\underline{t}_{(0.05/2;3,64)}$ value was 2.47. Bonferroni statistics for the plasma TC concentrations are presented in Table 8. The mean plasma TC concentration level at completion of the 1st circuit was significantly lower than the pre-CWT level ($p<0.05$).

Table 8

Bonferroni Statistics for Plasma Total Cholesterol

	TC1	TC2	TC3	TC4
TC1 = 158.8	-----	2.499*	1.922	-0.096
TC2 = 156.2		-----		
TC3 = 156.8			-----	
TC4 = 158.9				-----

* $p<0.05$

TC1= plasma TC concentration pre-CWT
TC2= plasma TC concentration at completion of the 1st circuit
TC3= plasma TC concentration at completion of the 3rd circuit
TC4= plasma TC concentration at 15 min post-CWT

HDL-C

The ANOVA performed on the plasma HDL-C concentration data showed significant changes in HDL-C concentrations during CWT exercise. Anderson's test of sphericity revealed that the sphericity assumption was not met. Greenhouse Geisser Probability was used to test the within subjects effects.

Table 9 and Table 10 present the ANOVA summary table and descriptive statistics, respectively, for plasma HDL-C concentrations measured at the specified time points. Figure 7 presents the means for plasma HDL-C concentrations measured at pre-CWT, completion of 1st and 3rd circuits, and 15 min post-CWT. It also shows the mean plasma HDL-C concentrations without correction for plasma volume changes.

The Bonferroni multiple comparison test was performed to identify whether there were significant differences among the plasma HDL-C concentrations measured at the specified time points. The Bonferroni test indicated that plasma HDL-C concentration at the completion of the 1st circuit (43.1 mg/dl) was significantly different from level at pre-CWT (47.0 mg/dl). Bonferroni statistics for plasma HDL-C concentrations are shown in Table 11.

Table 9

Analysis of Variance for Plasma HDL-C

SOURCE	SS	df	MS	F	p	G.G.P*
MEAN	138921.26	1	138921.26	441.17	0.0000	
ERROR	5038.23	16	314.88			
R(1)	1.08	1	1.08	0.06	0.8030	
ERROR	269.17	16	16.82			
R(2)	253.29	1	253.29	7.23	0.0161	
ERROR	560.19	16	35.01			
R(3)	0.54	1	0.54	0.12	0.7342	
ERROR	73.32	16	4.58			
R	254.92	3	84.97	4.52	0.0072	0.021
ERROR	902.69	48	18.80			

* G.G.P.= Greenhouse Geisser Probability

Table 10

Means and Standard Deviations for Plasma HDL-C

	H1	H2	H3	H4
Mean (mg/dl)	47.0	43.1	43.4	47.3
SD	9.2	9.9	9.9	9.4

H1= plasma HDL-C concentration pre-CWT
H2= plasma HDL-C concentration at completion of the 1st circuit
H3= plasma HDL-C concentration at completion of the 3rd circuit
H4= plasma HDL-C concentration at 15 min post-CWT

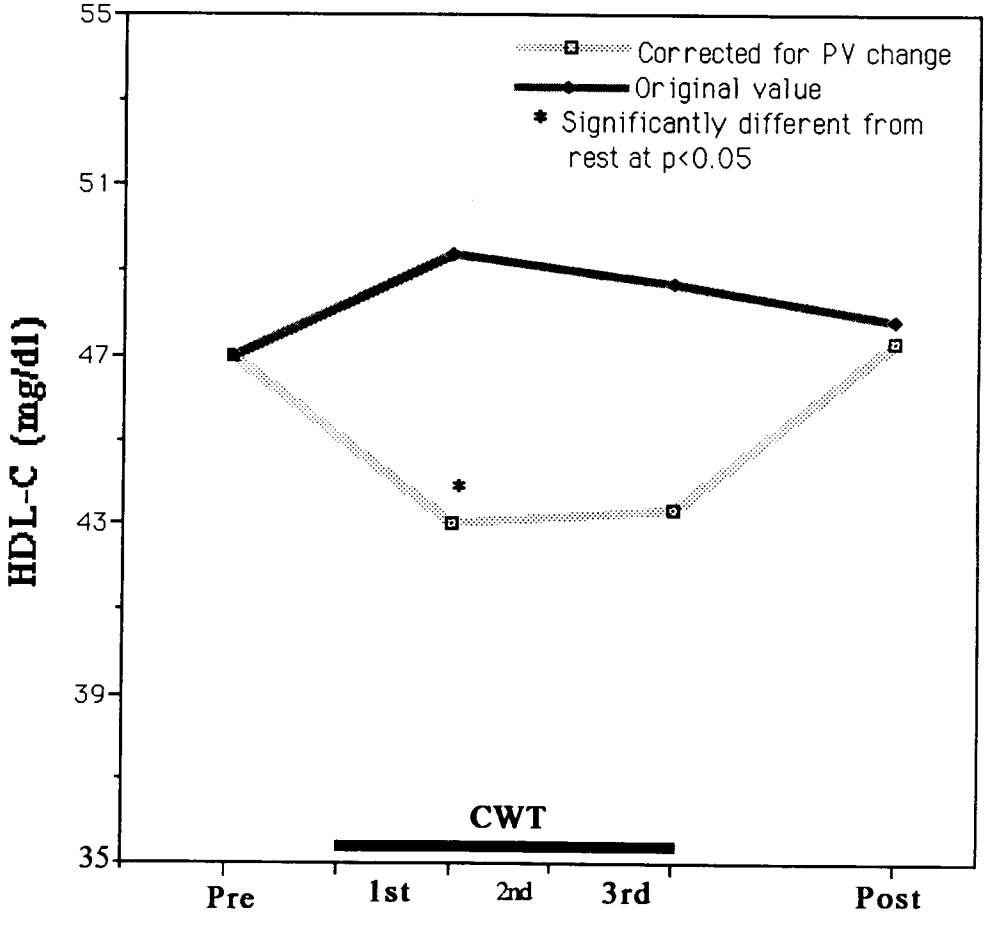

Figure 7

Mean Plasma HDL-C Concentration Changes

Table 11

Bonferroni Statistics for Plasma HDL-C

	H1	H2	H3	H4
H1 = 47.0	-----	2.622*	2.421	-0.202
H2 = 43.1		-----		
H3 = 43.4			-----	
H4 = 47.3				-----

* $p<0.05$
H1= plasma HDL-C concentration pre-CWT
H2= plasma HDL-C concentration at completion of the 1st circuit
H3= plasma HDL-C concentration at completion of the 3rd circuit
H4= plasma HDL-C concentration at 15 min post-CWT

LDL-C

The ANOVA for plasma LDL-C concentration data indicated that plasma LDL-C levels did not change significantly during or after CWT. The ANOVA summary for plasma LDL-C is presented in Table 12. Descriptive statistics for plasma LDL-C concentrations measured at pre-CWT, completion of 1st and 3rd circuits, and 15 min post-CWT are presented in Table 13.

Table 12

Analysis of Variance for Plasma LDL-C

SOURCE	SS	df	MS	F	p
MEAN	630008.13	1	630008.13	308.29	0.0000
ERROR	32697.10	16	2043.56		
R(1)	1.52	1	1.52	0.04	0.8510
ERROR	669.86	16	41.86		
R(2)	57.72	1	57.72	1.65	0.2174
ERROR	560.22	16	35.01		
R(3)	0.02	1	0.02	0.00	0.9553
ERROR	97.26	16	6.08		
R	59.26	3	19.76	0.71	0.5482
ERROR	1327.36	48	27.65		

Table 13

Means and Standard Deviations for Plasma LDL-C

	L1	L2	L3	L4
Mean (mg/dl)	95.5	97.2	97.1	95.1
SD	23.8	22.7	21.9	23.6

L1= plasma LDL-C concentration pre-CWT
L2= plasma LDL-C concentration at completion of the 1st circuit
L3= plasma LDL-C concentration at completion of the 3rd circuit
L4= plasma LDL-C concentration at 15 min post-CWT

Ratio of total cholesterol to HDL-C

The ANOVA performed on the data of the ratio (TC/HDL-C) showed significant differences, as presented in Table 14. Anderson's test of sphericity revealed that the sphericity assumption was not met. Greenhouse Geisser Probability was used to test the within subjects effects. Means and standard deviations for the ratios measured at pre-CWT, completion of 1st and 3rd circuits, and 15 min post-CWT are presented in Table 15. Means for the ratios measured at pre-CWT, completion of 1st and 3rd circuits, and 15 min post-CWT are displayed in Figure 8. Figure 8 shows comparisons of means of the ratios corrected and not corrected for plasma volume change.

Although Greenhouse Geisser Probability indicated significant differences among the data for the ratio measured at the specified time points ($p=0.0465$), the Bonferroni multiple comparison test showed that there was no statistically significant differences between means for the ratios of TC to HDL-C during and after CWT and the ratio at pre-CWT. Bonferroni statistics for the mean ratios of TC to HDL-C are listed in Table 16. Figure 9 shows the changes of plasma volume and all dependent variables corrected for plasma volume changes.

Table 14

Analysis of Variance for Ratio of TC to HDL-C

SOURCE	SS	df	MS	F	p	G.G.P*
MEAN	892.54	1	892.54	378.78	0.0000	
ERROR	37.70	16	2.35			
R(1)	0.02	1	0.02	0.24	0.6318	
ERROR	1.35	16	0.08			
R(2)	1.29	1	1.29	5.58	0.0312	
ERROR	3.72	16	0.23			
R(3)	0.001	1	0.001	0.02	0.8924	
ERROR	1.085	16	0.067			
R	1.32	3	0.44	3.43	0.0243	0.0465
ERROR	6.16	48	0.12			

* G.G.P.= Greenhouse Geisser Probability

Table 15

Means and Standard Deviations for Ratio of TC to HDL-C

	R1	R2	R3	R4
Mean	3.50	3.77	3.75	3.46
SD	0.81	0.89	0.82	0.77

R1= ratio (TC/HDL-C) pre-CWT
R2= ratio (TC/HDL-C) at completion of the 1st circuit
R3= ratio (TC/HDL-C) at completion of the 3rd circuit
R4= ratio (TC/HDL-C) at 15 min post-CWT

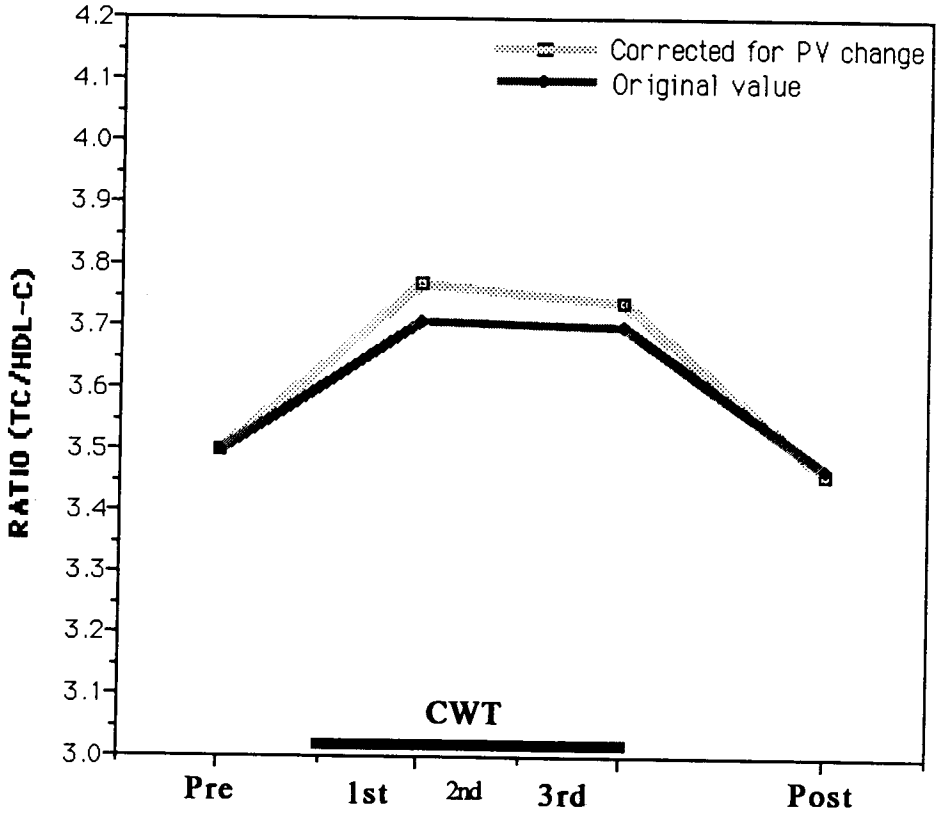

Figure 8

Mean Ratio (TC/HDL-C) Changes

Table 16

Bonferroni Statistics for Ratio of TC to HDL-C

	R1	R2	R3	R4
R1 = 3.50	-----	-2.200	-2.037	0.326
R2 = 3.77		-----		
R3 = 3.75			-----	
R4 = 3.46				-----

R1= ratio (TC/HDL-C) pre-CWT
R2= ratio (TC/HDL-C) at completion of the 1st circuit
R3= ratio (TC/HDL-C) at completion of the 3rd circuit
R4= ratio (TC/HDL-C) at 15 min post-CWT

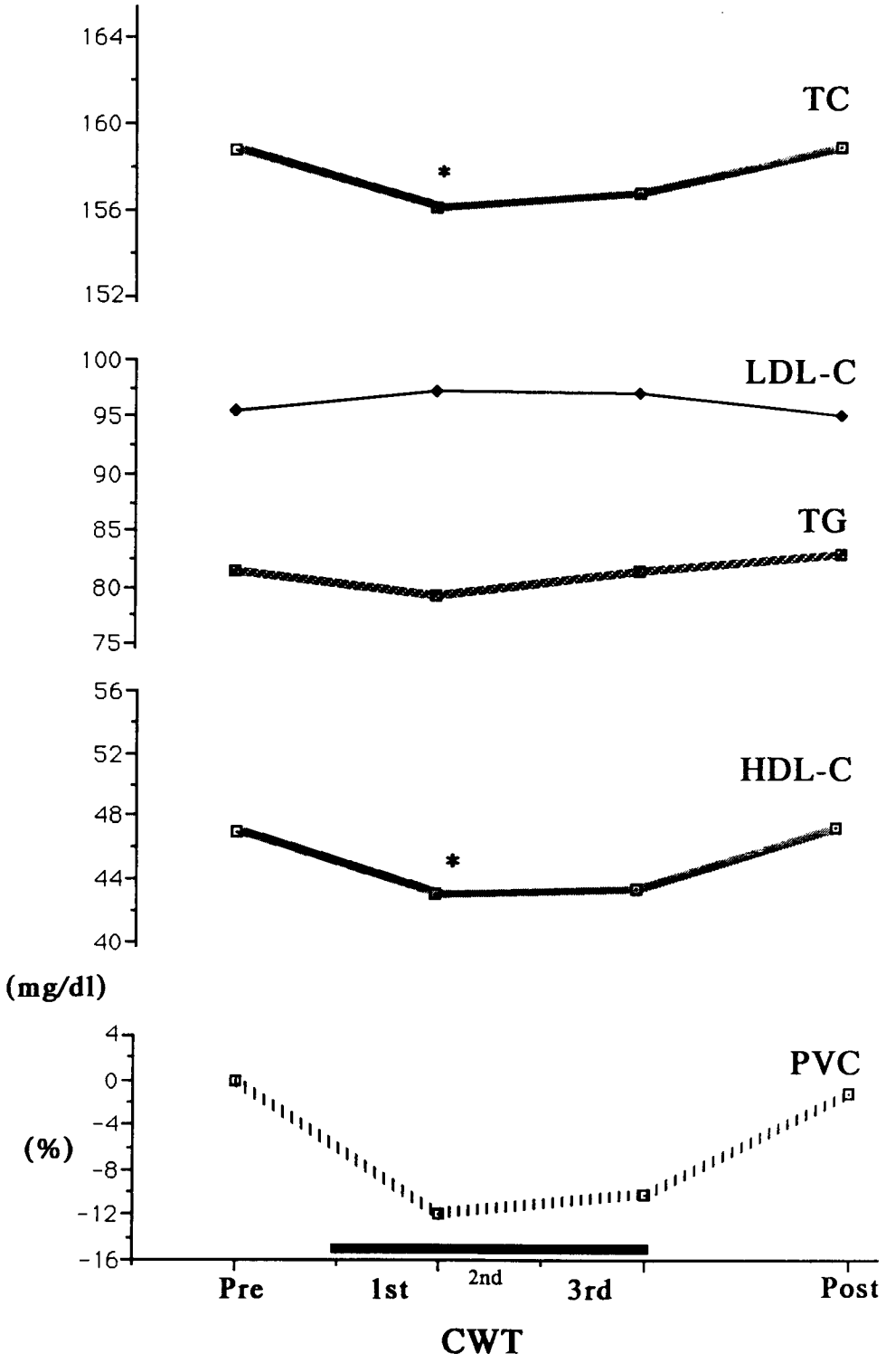

Figure 9

The Changes of Mean TC, LDL-C, TG, HDL-C and PV

Discussion

The discussion of the results is presented under the following headings: (1) plasma volume changes with circuit weight training, (2) triglycerides, (3) total cholesterol and LDL-C, (4) the ratio of total cholesterol to HDL-C, and (5) HDL-C.

Plasma volume changes with circuit weight training

The decreases of mean plasma volume at completion of 1st and 3rd circuits of CWT (-11.9 ± 4.1%, -10.3 ± 3.8%) were within the range reported in other studies involving resistance exercise.

Several studies have reported plasma volume responses due to weight lifting exercise. Collins et al. (1986) reported a 14.3% decrease in plasma volume following weight lifting at 70% of 1-RM using 3 sets of 4 exercises, each performed to exhaustion. Knowlton et al. (1987) studied the plasma volume response following squat exercise. Each subject completed 8 sets of 8 repetitions at 55% of 1-RM with 3 min of rest between sets. Plasma volume, determined by using hematocrit volume, was decreased 17.9%.

Collins et al. (1989) studied the relation of plasma volume change to the intensity of weight lifting at four different intensities (40, 50, 60 and 70% of 1-RM). The weight lifting exercise consisted of 3 circuits of 4

exercises (bench press, bent-over row, arm curl, and parallel squat), with 10 repetitions of each exercise performed over a 30 sec period followed by 30 sec of rest. Plasma volume decreased linearly in relation to intensity of weight lifting, with mean responses of -7.7, -10.7, -12.1, and -13.9% at 40, 50, 60, and 70% of 1-RM, respectively. They concluded that plasma volume decreases linearly in relation to intensity of weight lifting and that the relationship is similar to that reported for dynamic, low resistance exercise such as cycling and running.

Plasma volume shifts can be attributed to higher arterial pressures and larger transcapillary osmotic gradients due to greater accumulation of muscle metabolites (Miles et al., 1983). Increased arterial pressure promotes filtration of plasma into the interstitial space. Knowlton et al. (1987) reported that elevation in arterial pressure is highly correlated ($r = -0.98$) with changes in plasma volume during weight lifting. Since mean arterial pressure appears to be higher during weight lifting than during dynamic, low resistance exercise, a larger plasma volume change might be expected (Collins et al., 1989).

During weight lifting the breakdown of glycogen, along with the accumulation of lactate and other metabolites in the active muscle increases intracellular osmolarity which may result in fluid fluxes from interstitial to

intracellular and from vascular to interstitial spaces (Lundvall, 1972; Collins et al., 1989).

Sweat loss can contribute to decrease in plasma volume. Although sweat loss was not measured in this study, it was probably negligible because the duration of CWT was short (21 min). Mean plasma volume returned to the pre-CWT level at 15 min post-CWT. Plasma volume change during CWT probably reflected body fluid redistribution rather than loss.

Triglycerides

Mean triglyceride (TG) level at pre-CWT (81.4 mg/dl) was slightly above the 50th percentile (78 mg/dl) for white male subjects of similar age in North America (Lipid Research Clinics Program Epidemiology Committee, 1979).

The results of ANOVA for TG concentrations showed that there were no statistically significant differences between pre-CWT levels and levels during CWT or 15 min post-CWT. Therefore, the null hypothesis of hypothesis 2 was retained.

The magnitude to which plasma TG concentrations have been shown to change due to vigorous exercise is highly dependent on the pre-exercise values, and the degree and duration of exercise performed (Haskell, 1984). When plasma TG values are relatively low (as seen in trained endurance athletes), vigorous exercise for as long as 2 hours usually does not result in a significant lowering of plasma TG (Cullinane, Sinconolfi et al., 1982; Hurter et al., 1972).

However, exercise for a longer duration on a single day (Enger, Strömme, & Refsum, 1980; Thompson et al., 1980) or exercise repeated on consecutive days (Dressendorfer et al., 1982; Wirth et al., 1983) lowers TG levels for up to several days.

Several authors have reported that plasma TG concentration decreases with prolonged exercise and remains low for 24 hours or longer (Enger, Strömme, & Refsum, 1980; Sady et al., 1986; Thompson et al., 1980). Thompson et al. (1980) found TG to be lower among trained men after a 42 km race. This decrement persisted for at least 2 days after the race. Sady et al. (1986) reported a decrease in TG concentration of well-trained male subjects after a 42 km race. However, most studies on the acute effects on TG levels of exercise for shorter periods have failed to demonstrate an immediate effect of exercise on TG concentration (Cullinane, Lazarus et al., 1981; Cullinane, Sinconolfi et al., 1982; Hicks, MacDougall, & Muckle, 1987).

In this study, the lack of significant changes of TG level during or following CWT can be attributed to the single session and short duration (21 min) of exercise.

The concentration of plasma TG reflects the relative rates of synthesis and clearance. TG synthesis is likely to be increased early in moderate exercise due to the increased availability of fatty acid substrate (Berger & Griffiths, 1987). With more prolonged and severe physical activity, TG

synthesis may actually decrease as a result of a high glucagon:insulin ratio and a depletion of carbohydrate energy sources (Ahlborg & Felig, 1982). In this study, it is possible that TG synthesis and clearance rates were comparable during and after CWT, with little net change of TG levels in plasma.

Total cholesterol and LDL-C

Total Cholesterol (TC).

The mean plasma TC level for subjects at pre-CWT was 158.8 mg/dl. This value is comparable to the 50th percentile (159 mg/dl) for white male subjects of similar age in North America (Lipid Research Clinics Program Epidemiology Committee, 1979).

The results of ANOVA for TC concentrations (Table 6) showed that plasma TC levels were changed significantly during CWT and at 15 min post-CWT. Therefore, the null hypothesis of hypothesis 1 was rejected.

The acute effects of aerobic-type exercise on TC concentrations have been studied, with exercise duration ranging from 13 min (Naughton & Balke, 1964) to 64 hours (Wirth et al., 1983); intensity ranging from moderate (Sannerstedt, Sanbar, & Conway, 1970) to maximum (Enger, Strömme, & Refsum, 1980); and frequencies ranging from one session on one day (Thompson et al., 1980) to repeated

sessions for up to 20 days (Dressendorfer et al., 1982). When TC is measured during or immediately after exercise, either no change (Cullinane et al., 1981; Hurter et al., 1972) or an increase (Lennon et al.,1983; Hicks, MacDougall, & Muckle, 1987) have been reported. Others have observed no significant changes immediately following acute activity, but significant reductions in TC several hours later (Thompson et al., 1980).

In this study, the mean for TC concentration measured at the completion of the 1st circuit was slightly lower than the mean pre-CWT level (3 mg/dl, 2% of the pre-CWT value). Although relatively small, the difference was statistically significant (G.G.P.=0.034). Mean for TC concentrations at 15 min post-CWT was almost same the mean pre-CWT level.

The reason for the exercise-induced reduction of TC levels is not known (Wirth et al., 1983). The reduction may be due to enhanced uptake and oxidation of free fatty acids during periods of exertion reduce the availability of free fatty acids for VLDL-C formation (Grundy et al., 1979; Wirth et al., 1981).

Another factor that may affect change in TC concentration with CWT is a shift in plasma volume. Sink et al. (1989) suggested that plasma volume shifts may account for the disparity of the research findings regarding exercise effects on lipoproteins. In this study, without correction of TC concentrations for plasma volume changes,

TC concentration actually increased during and immediately following CWT, as shown in Figure 6. This pattern of change in TC concentrations is consistent with patterns reported in other studies (Lennon et al., 1983; Hicks et al., 1987).

LDL-C.

The mean plasma LDL-C concentration of subjects at pre-CWT was 95.5 mg/dl. This is slightly below the 50th percentile (101 mg/dl) for white male subjects of similar age in North America (Lipid Research Clinics Program Epidemiology Committee, 1979).

The results of ANOVA for LDL-C concentrations showed that LDL-C levels were not changed significantly during or after CWT. Therefore, the null hypothesis of hypothesis 4 was retained. In the present study, it appeared that CWT exercise produced little change in the plasma LDL-C concentration during CWT and 15 min post-CWT.

Most studies have reported no acute responses of LDL-C concentrations to either short or long duration aerobic-type exercise (Berger et al., 1981; Cullinane et al., 1982; Lennon et al., 1983).

Ratio of total cholesterol to HDL-C

The means for ratios of TC to HDL-C at the completion of 1st circuit (3.77) and at the completion of 3rd circuit (3.75) were slightly higher than the mean pre-CWT level (3.50), but the mean ratio of TC to HDL-C at 15 min post-CWT (3.46) was lower than the mean pre-CWT level.

The results of ANOVA for ratio of TC to HDL-C (Table 14) showed that ratios of TC to HDL-C were changed significantly during CWT and recovery period. Therefore, the null hypothesis of hypothesis 5 was rejected. However, the Bonferroni multiple comparison test showed that the ratios of TC to HDL-C at the completion of the 1st circuit and 3rd circuit, and at 15 min post CWT were not significantly different from the pre-CWT ratio. The significant results of the ANOVA may be due to the difference between the ratio of TC to HDL-C at the completion of the 1st circuit and the ratio of TC to HDL-C at 15 min post-CWT.

Changes in the ratio of TC to HDL-C during and following CWT reflected decrease in HDL-C concentration during CWT and slight increase in HDL-C concentration at 15 min post-CWT.

HDL-C

Average HDL-C level at resting (47 mg/dl) was slightly higher than the 50th percentile (45 mg/dl) for white male subjects of similar age in North America (Lipid Research Clinics Program Epidemiology Committee, 1979).

The results of ANOVA for HDL-C concentrations (Table 9) showed that plasma HDL-C levels were changed significantly during and after CWT. Therefore, the null hypothesis of hypothesis 3 was rejected.

The acute effects of exercise on plasma HDL-C concentrations have been studied for a wide diversity of aerobic-type exercise conditions. Findings have shown either no change or an increase of 5-25%. However, no study has reported on the acute effects of resistance exercise on plasma HDL-C concentrations. It is difficult to compare the results of this study with other studies of acute effects of endurance aerobic-type exercise since the energy source, intensity, and duration of effort are different. Table 16 shows a summary of results of related studies concerning the acute effects of exercise on lipid and lipoprotein profiles.

Lennon et al. (1983) reported an increase above pre-exercise resting levels of plasma HDL-C. The increase persisted at all time points (10, 20, 30, and 40 min of cycling at 55% of $\dot{V}O_2$ max), but declined to pre-exercise level by 15 min post-exercise. Hicks et al. (1987) observed statistically significant acute increases in HDL-C and HDL

Table 17

Summary of Studies on Acute Effects of Exercise

Researchers	Subjects (Age)	Exercise	PV Change (corrected)	Results
Thompson et al. (1980)	12 trained male runners (24-50)	42 km	-	↓ TG, TC
Lennon et al. (1983)	14 male 14 female U. students (29.7±2.6)	bicycle for 40 min 55% of $\dot{V}O_2$ max	-	↑ HDL-C
Wirth et al. (1983)	26 volunteers (22-28)	soccer 64 hours	-	↑ HDL-C ↓ TC,LDL-C
Skinner et al. (1985)	56 male runners (21-62)	42 km	-	↑ HDL-C
Sady et al. (1986)	10 male runner (35.1±3.7)	42 km	-	↑ HDL-C (HDL_2)
Hicks et al. (1987)	12 male Volunteers (19-41)	9-12 km run on treadmill at 60, 90% of $\dot{V}O_2$ max	Hct	↑ TC,HDL-C
Study	17 male U. Students (18-25)	CWT (21 min)	Hct & Hb	↓ TC,HDL-C

apoprotein A with 9-12 km running on a treadmill at two different intensities (60% and 90% of the subject's $\dot{V}O_2$ max). The greater increases in HDL-C and HDL apoprotein A were found with the higher intensity exercise (90% of the subject's $\dot{V}O_2$ max). Skinner, Black, and Maughan (1985) reported an increase of mean concentration of HDL-C for 56 male subjects after a 42.2 km marathon race.

Cullinane et al. (1982) evaluated the acute effects of exercise on lipoprotein of both sedentary and trained cyclists at their respective anaerobic threshold, after 1 and 2 hours of cycling. No significant change in HDL-C was observed among the subjects after either cycling period.

In this study, the HDL-C concentrations during and immediately following CWT were lower than the pre-CWT level. However, the Bonferroni multiple comparison test (Table 11) showed that only the HDL-C concentration at the completion of the 1st circuit was statistically different from the pre-CWT level. The HDL-C concentration returned to the pre-CWT level (47 mg/dl) at 15 min post-CWT exercise (47.3 mg/dl).

The decrease in HDL-C concentrations with CWT is not consistent with results of other studies concerning the acute effects of aerobic-type exercise.

The mechanisms responsible for the changes of HDL-C concentrations during exercise are not fully understood. However, the observed decrease in the HDL-C concentration in

this study may indicate a reduced rate of lipolysis during CWT exercise. The decrease may be due to the subjects using more anaerobic energy source during CWT exercise. Resistance exercise is more associated with anaerobic energy sources (ATP-PC and lactic acid sources) than it is with aerobic metabolism (Fleck & Kraemer, 1987). Griffin, Skinner, and Maughan (1988) suggested a reduced rate of lipolysis and a decrease in skeletal muscle lipoprotein lipase (LPL) to explain the decreased in HDL-C concentration of the subjects when they were consuming more carbohydrate calories with exercise. The decrease LPL activity after carbohydrate diet has been reported by others (Jacobs, Lithell, & Karlsson, 1982; Lithell et al., 1982). An increase in LPL activity accelerates the catabolic rate of TG-rich lipoproteins, which results in the transfer of cholesterol, phospholipids, and apoproteins to nascent HDL particles, thus increasing HDL mass (Haskell, 1984). The fast twitch glycolytic muscle involves the lowest level of LPL activity (Borensztajn et al., 1975).

There may be an association between changes in HDL-C concentration and energy metabolism. During CWT exercise, the energy sources are provided by ATP-PC and lactic acid system, reducing the rate of lipolysis in muscles. Following CWT exercise, anaerobic energy sources are replenished by aerobic energy sources produced by the increased rate of lipolysis. This explanation is supported by the findings in

this study that LDL-C concentration was slightly higher at the completion of the 1st circuit of CWT, and that HDL-C concentration was slightly higher at 15 min post-CWT, and by findings of Kiens et al. (1989). Kiens et al. conducted a study to determine the effects of insulin and exercise on muscle LPL activity. They reported that no change in muscle LPL activity was observed immediately after one-legged knee extension exercise, but 4 hours after exercise muscle LPL activity was higher in the exercised thigh compared with the contralateral non-exercised thigh. They suggested that muscle contractions cause a local, delayed, and transient increase in muscle LPL activity.

Another possible explanation for the decreased HDL-C concentration is that there is either a decreased rate of HDL synthesis or an increased rate of HDL uptake, by a receptor-mediated process (Fidge, 1986). Fidge demonstrated that an HDL-binding protein present in rat liver and kidney membranes is involved in the metabolism of plasma HDL-C. Ruys et al. (1989) studied the peripheral production of HDL-C and of the subclasses HDL_2 and HDL_3 assessed by measurement of the arteriovenous fluxes across the human forearm. They reported that exercise (20 min isometric contraction) had no significant effect on the net arteriovenous flux (i.e., production or removal) of HDL_2 and HDL_3 in the fasted state. After fat loading, exercise increased the arteriovenous flux for HDL_3. Based on these

results, they suggested that formation of HDL_3 during lipolysis by LPL in the muscle capillary bed is influenced by the supply of chylomicrons and other lipoprotein substrates.

In this study, the fasted state of subjects (12-14 hours overnight fast) may have contributed to decreased peripheral HDL-C production. However, LPL activity and the subfractions of HDL-C were not measured.

The significant changes of HDL-C levels during CWT was not in the anticipated direction, but there were slight increase in HDL-C and decrease in LDL-C at 15 min post CWT. This response may be due to a local, delayed, and transient increase of lipolysis in muscles.

It appears that CWT may improve lipid and lipoprotein profiles as part of the adaptation of total conditioning process, although more studies are needed to prove this relationship. Decreased body fat and increased lean body mass (the long-term effect of CWT), and increased transient lipolysis in muscles (the acute or short-term effect of CWT) may be responsible for favorable lipid-lipoprotein changes.

CHAPTER 5

SUMMARY, CONCLUSIONS, AND RECOMMENDATIONS

Summary

The purpose of this study was to determine the acute response of total cholesterol, triglycerides, HDL-C, LDL-C, and the ratio of total cholesterol to HDL-C to a single session of CWT exercise. The subjects for this study were seventeen nonsmoking male students with a mean age 20.1 ± 2.4 years; height 181.2 ± 3.8 cm; weight 81.6 ± 10.7 kg. All subjects were familiar with the concept of CWT and had been participating in weight lifting classes.

Subjects repeated a four-station CWT three times. The CWT stations consisted of bench press, parallel squat, leg extension, and seated row. The resistances of the lifts at the first circuit were 50% of 3-RM for bench press, 60% of 3-RM for squat, 40% of 3-RM for seated row, and 40% of 3-RM for leg extension. The resistances of the lifts at the second and third circuits were reduced each time by 10 pounds for the bench press and seated row, 15 pounds for the leg extension, and 20 pounds for the parallel squat. Subjects exercised each lift for 1 min with a 30-sec rest between exercise stations. A 2-min rest was used to obtain blood samples between repeats of circuits. Blood samples

were drawn pre-CWT, after completion of the 1st circuit and 3rd circuit, and 15 min post-CWT. All concentrations for each subject were corrected for plasma volume change.

A repeated measures ANOVA was used to determine if significant differences existed among mean values for TG, TC, HDL-C, LDL-C and ratio of TC to HDL-C measured at pre-CWT, completion of the 1st and 3rd circuit, and 15 min post-CWT.

Mean plasma concentrations of TG and LDL-C were not changed significantly during and 15 min post-CWT. Mean plasma concentrations of TC and HDL-C were changed significantly during and 15 min post-CWT. The mean plasma levels of TC and HDL-C at the completion of the 1st circuit were lower than the levels at pre-CWT ($p<0.05$). Mean ratios of TC to HDL-C were changed significantly during and 15 min post-CWT ($p<0.05$).

Conclusions

Within the limitations and design of this study, the following conclusions can be drawn:

1. Plasma TG and LDL-C levels were not changed significantly by short duration moderate intensity CWT exercise.

2. Plasma TC and HDL-C levels were temporarily effected by short duration moderate intensity CWT exercise. In this

study, plasma TC and HDL-C were lower at the completion of the 1st circuit of CWT, but returned to the pre-CWT level in 15 min post-CWT.

3. The ratios of TC to HDL-C were changed significantly by short duration moderate intensity CWT. In this study, HDL-C was lower at the completion of the 1st circuit, and slightly higher at 15 min post-CWT, than at pre-CWT.

Recommendations

The following recommendations are suggested for future investigation:

1. A replication of the present study with measurement of the muscle lipoprotein lipase activity and plasma LCAT activity. The study should extend the blood sampling period after CWT (i.e., 2 and 4 hours post-CWT).

2. A replication of the present study with measurement of plasma free fatty acid levels and HDL-C subfractions (HDL_2 and HDL_3).

3. A replication of the present study with direct measurement of VLDL-C and LDL-C levels.

4. A study to compare the acute response of lipid and lipoprotein to CWT between strength-type trained subjects and sedentary subjects.

5. A study to investigate the acute effects of different intensity CWT in trained subjects (heavy

resistance with a low repetition vs low resistance with a high repetition). The total weight lifted by two groups should be the same (Total weight lifted = Total number of repetitions performed x Total resistance).

REFERENCES

Ahlborg, G., & Felig, P. (1982). Lactate and glucose exchange across the forearm, legs and splanchnic bed during and after prolonged leg exercise. Journal of Clinical Investigation, 69, 45-54.

Allison, T. G., Iammarino, R. M., Metz, K. F., Skrinar, G. S., Kuller, L. H., & Robertson, R. J. (1981). Failure of exercise to increase high density lipoprotein cholesterol. Journal of Cardiac Rehabilitation, 1, 257.

Aro, A., Soimakallio, S., Voutailainen, E., Ehnholm, C., & Wiljasalo, M. (1986). Lipoprotein lipid levels as indicators of severity of angiographically assessed coronary artery disease. Atherosclerosis, 62, 219-275.

Benditt, E. P., & Gown, A. M. (1980). Atheroma: The artery wall and the environmemt. In G. W. Richter & M. A. Epstein (Eds.), International review of experimental pathology (pp. 56-109). New York: Academic Press.

Berg, A., Johns, J., Baumstark, M., & Keul, J. (1981). HDL-cholesterol changes during and after intense long-lasting exercise. International Journal of Sports Medicine, 2, 121-123.

Berg, A. G., Ringwald, G., & Keul, J. (1980). Lipoprotein cholesterol in well-trained athletes: A preliminary communication: reduced HDL-cholesterol in power athletes. International Journal of Sports Medicine, 1, 137-138.

Berger, G. M. B., & Griffiths, M. P. (1987). Acute effects of moderate exercise on plasma lipoprotein parameters. International Journal of Sports Medicine, 8, 336-341.

Bilheimer, D. W. (1988). Therapeutic control of hyperlipidemia in the prevention of coronary atherosclerosis: A review of results from recent clinical trials. The American Journal of Cardiology, 62, 1J-9J.

Blankenhorn, D. H., Nessim, S. A., Johnson, R. L., Sanmarco, M. E., Azen, S. P., & Cashin-Hemphill, L. (1987). Beneficial effects of combined cholestipol-niacin therapy on coronary atherosclerosis and coronary venous bypass grafts. Journal of the American Medical Association, 257, 3233-3240.

Blessing, D. L., Williford, H. N., Barksdale, J. M., & Smith, F. H. (1988). Alterations in lipid and cardiorespiratory function after weight training. Journal of Human Movement Studies, 14, 75-83.

Blumenthal, J. A., Rejeski, W. J., Walsh-Riddle, M., Emery, C. F., Miller, H., Roark, S., Ribisl, P. M., Morris, P. B., Brubaker, P., & Williams, S. (1988). Comparison of high and low intensity exercise training early after acute myocardial infarction. American Journal of Cardiology, 61, 26-30.

Borensztajn, J., Rone, M. S., Babirak, S. P., McGarr, J. A., & Oscai, L. B. (1975). Effect of exercise on lipoprotin lipase activity in rat heart and skeletal muscle. American Journal of Physiology, 229(2), 394-397.

Breier, CH., Drexel, H., Lisch, H. J., Mülhberger, V., Herold, M., & Knapp, E. (1985). Essential role of post-heparin lipoprotein lipase activity and of plasma testosterone in coronary artery disease. Lancet, 1, 1242-1244.

Brown, M. S., & Goldstein, J. L. (1983). Lipoprotein receptors in the liver: Control signals for plasma cholesterol traffic. Journal of Clinical Investigation, 72, 743-747.

Brown, M. S., & Goldstein, J. L. (1984). How LDL receptors influence cholesterol and atherosclerosis. Scientific America, 251(5), 58-66.

Butler, R. M., Beierwaltes, W. H., & Rogers, F. J. (1987). The cardiovasular response to circuit weight training in patients with cardiac disease. Journal of Cardiopulmonary Rehabilitation, 7, 402-409.

Carew, T. E., Koschinsky, T., Hayes, S. B., & Steinberg, D. (1976). A mechanism by which high density lipoproteins may slow the atherogenic process. Lancet, 1315-1317.

Cassel, J., Heyden, S., Bartel, A. G., Kaplan, B. H., Tyroler, H. A., Cornoni, J. C., & Hames, C. G. (1971). Occupation and physical activity and coronary heart disease. Archives of Internal Medicine, 128, 920-928.

Castelli, W. P., Garrison, R. J., Wilson, P. W., Abbott, R. D., Kalousdian, S., & Kannel, W. B. (1986). Incidence of coronary heart disease and lipoprotein cholesterol levels: The Framingham Study. Journal of the American Medical Association, 256(20), 2835-2838.

Chapman, J. M., Goerke, L. S., Dixon, W., Loveland, D. B., & Philips, E. (1957). Measuring the risk of coronary heart disease in adult population groups: The clinical status of a population group in Los Angeles under observation for two to three years. American Journal of Public Health, 47(2), 33-42.

Clarkson, P. M., Hintermister, R., Fillyaw, M., & Stylos, L. (1981). High density lipoprotein cholesterol in young adult weight lifters, runners, and untrained subjects. Human Biology, 53(2), 251-257.

Cohn, J. S., McNamara, J. R., & Schaefer, E. J. (1988). Lipoprotein cholesterol concentrations in the plasma of human subjects as measured in the fed and fasted states. Clinical Chemistry, 34(12), 2456-2459.

Collins, M. A., Cureton, K. J., Hill, D. W., & Ray, C. A. (1989). Relation of plasma volume change to intensity of weight lifting. Medicine and Science in Sports and Exercise, 21, 178-185.

Collins, M. A., Hill, D. W., Cureton, K. J., & DeMello, J. J. (1986). Plasma volume change during heavy reisitamce weight lifting. European Journal of Applied Physiology, 55, 44-48.

Cullinane, E., Lazarus, B., Thompson, P. D., Saratelli, A., & Herbert, P. N. (1981). Acute effects of a simple exercise session on serum lipids. Clinica Chimica Acta, 109, 341-344.

Cullinane, E., Sinconolfi, S., Saratelli, A., & Thompson, P. D. (1982). Acute decrease in serum triglycerides with exercise: Is there a threshold for an exercise effect? Metabolism, 31, 844-847.

Cuppers, H. J., Erdmann, D., Schubert, H., Berchtold, P., & Berger, M. (1982). Glucose tolerance, serum insulin, and serum lipids in athletes. In M. Berger, C. Christacopoulus, & J. Wahren (Eds.), Diabetes and exercise (pp. 115-165). Amsterdam: Elsevier.

Dawber, T. R., Moore, F. E., & Mann, G. V. (1957). Coronary Heart Disease in the Framingham Study. American Journal of Public Health, 47(2), 4-24.

Day, J. L., Metcalfe, J., & Simpson, C. N. (1982). Adrenergic mechanisms in control of plasma lipid concentrations. British Medical Journal, 284, 1145-1148.

Dill, D. B., & Costill, D. L. (1974). Calculation of percentage changes in volumes of blood, plasma, and red cells in dehydration. Journal of Allpied Physiology, 37(2), 247-248.

Dressendorfer, R. H., Wade, C. E., Hornick, C., & Timmis, G. C. (1982). High-density lipoprotein-cholesterol in marathon runners during a 20 day road race. Journal of the American Medical Association, 247, 1715-1717.

Dufaux, B., Assman, G., & Hollman, W. (1982). Plasma lipoproteins and physical activity: A review. International Journal of Sports Medicine, 3, 123-136.

Dunn, O. J. & Clark, V. A. (1987). Applied statistics: Analysis of variance and regression (2nd ed.). New York: John Wiley & Sons

Eisenberg, S. (1984). High density lipoprotein metabolism. Journal of Lipid Research, 25, 1017-1058.

Eisenberg, S., Chajek, T., & Deckelbaum, R. J. (1982). The plasma origin of low density and high density lipoproteins. In L. A. Carlson, & B. Pernow (Eds.), Metabolic risk factors in ischemic cardiovascular disease (pp. 59-65). New York: Raven Press.

Enger, S. C., Herbjornsen, K., Eriksen, J., & Fretland, A. (1977). High density lipoproteins and physical activity: The influence of physical exercise, age and smoking on HDL cholesterol and HDL total cholesterol ratio. Scandinavia Journal of Clinical Laboratory Investigations, 37, 251-255.

Enger, S. C., Strömme, S. B., & Refsum, H.E. (1980). High-density lipoprotein cholestrol, total cholestrol and triglycerides in serum after a single exposure to prolonged heavy exercise. Scandinavia Journal of Clinical Laboratory Investigations, 40, 341-345.

Expert Panel. (1988). Report of the national cholesterol education program expert panel on detection, evaluation, and treatment, of high blood cholesterol in adults. Archives of Internal Medicine, 148, 36-69.

Fairbanks, V. F., & Klee, G. G. (1986). Biochemical aspects of hematology. In N. W. Tiets (Ed.), Textbook of clinical chemistry (pp. 1532-1541). Philadelphia: W.B. Saunders.

Farrell, P. A., & Barboriak, J. (1980). Time course of alterations in plasma lipid and lipoprotein concentration during eight weeks of endurance training. Atherosclerosis, 37, 231.

Farrell, P. A., Maksud, M. L., & Pollock, M. L. (1982). A comparison of plasma cholesterol, triglycerides, and high density lipoprotein cholesterol in speed skaters, weightlifters and nonathletes. European Journal of Applied Physiology, 48, 77-82.

Featherston, J. F., Holly, R. G., & Amsterdam, E. A. (1987). Physiological responses to weight lifting in cardiac patients. Medicine and Science in Sports and Exercise, 19(abstract), S93.

Fidge, N. H. (1986). Partial purification of a high density lipoprotein-binding protein from rat liver and kidney membranes. Federation of European Biochemical Societies, 199, 265-268.

Fielding, P. E., Fielding, C. J., Havel, R. J., Kane, J. P., & Tun, P. (1983). Cholesterol net transprt esterification, and transfer in human hyperlipidemic plasma. Journal of Clinical Inverstigation, 71, 449-460.

Fleck, S. J., & Kraemer, W. J. (1988). Resistance training: Physiological responses and adaptation (part 2 of 4). Physician and Sportsmedicine, 16, 108-124.

Fleck, S. J. & Lean, L. S. (1987). Resistive training experience and the pressor response during resistive training. Journal of Applied Physiology, 63, 116-120.

Friedewald, W. T., Levy, R. I., & Fredrickson, D. S. (1972). Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. Clinical Chemistry, 18, 499-502.

Gettman, L. R., Ayers, J. J, & Pollock, M. L. (1979). Physiologic effects on adult men of circuit strength training and jogging. Archives of Physical Medicine and Rehabilitation, 60, 114-120.

Gettman, L. R., & Pollock, M. L. (1981). Circuit weight training: A critical review of its physiological benefits. Physician and Sportsmedicine, 9, 44-60.

Ghilarducci, L. E., Holly, R. G., & Amsterdam, E. A. (1989). Effects of high resistance training in coronary artery disease. American Journal of Cardiology, 64, 866-870.

Glomset, J. A. (1970). Physiological role of lecithin-cholesterol Acyltransferase. American Journal of Clinical Nutrition, 23, 1120-1136.

Gofman, J. W., Young, W., & Tandy, R. (1966). Ischemic heart disease, atherosclerosis and longevity. Circulation, 34, 679-697.

Goldberg, A. P. (1988). Health promotion and aging physical exercise. In Surgeon General's workshop, health promotion and aging (pp. C1-C19). Washington, D.C.

Goldberg, A. P. (1989). Aerobic and resistive exercise modify risk factors for coronary heart disease. Medicine and Science in Sports and Exercise, 20(6), 669-674.

Goldberg, L., & Elliot, D. L. (1985). The effect of physical activity on lipid and lipoprotein levels. Medical Clinics of North America, 69(1), 41-55.

Goldberg, L., & Elliot, D. L. (1987). The effect of exercise on lipid metabolism in men and women. Sports Medicine, 4, 307-321.

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

Goldstein, J. L., & Brown, M. S. (1982). Insights into the pathogenesis of atherosclerosis derived from studies of familial hypercholesterolemia. In L. A. Carlson & B. Pernow (Eds.), Metabolic risk factors in ischemic cardiovascular disease (pp. 17-34). New York: Raven Press.

Goldstein, J. L., & Brown, M. S. (1983). Defective lipoprotein receptors and atherosclerosis. New England Journal of Medicine, 309(5), 289-296.

Gotto, A. M., Pownall, H. J., & Havel, R. J. (1986). Introduction to the plasma lipoproteins. In J. P. Segrest & J. J. Albers (Eds.), Methods in enzymology. Vol. 128 plasma lipoproteins part A (pp. 3-41). Orlando, FL: Academic Press.

Griffin, B. A., Skinner, E. R., & Maughan, R. J. (1988). The Acute effect of prolonged walking and dietary changes on Plasma lipoprotein concentrations and HDL subfractions. Metabolism, 37, 535-541.

Grundy, S. M., Mok, H. Y., Zech, L., Steiberg, D., & Berman, M. (1979). Transport of very low density lipoprotein triglycerides in varying degrees of obesity and hypertriglyceridemia. Journal of Clinical Investigation, 63, 1274-1283.

Guyton. A. (1986). Textbook of medical physiology. Philadelphia: W.B. Saunders.

Gwynne, J. T. (1989). High density lipoprotein cholesterol levels as a maker of reverse cholesterol transport. American Journal of Cardiology, 64, 10G-17G.

Harris, K. A., & Holly, R. G. (1987). Physiological response to circuit weight training in boderline hypertensive subjects. Medicine and Science in Sports and Exercise, 19, 246-252.

Hartung, G. H., Foreyt, J. P., Mitchell, R. E., Vlasek, I., & Gotto, A. M. (1980). Relation of diet to HDL-C in middle-aged marathon runners, joggers, and inactive men. New England Journal of Medicine, 302, 357-361.

Haskell, W. L. (1984). The influence of exercise on the concentrations of triglyceride and cholesterol in human plasma. Exercise and Sports Science Review, 12, 205-244.

Heath, G. W., Ehsani, A. A., Hagberg, J. M., Hinderliter, J. M., & Goldberg, A. P. (1983). Exercise training improves lipiprotein lipid profiles in patients with coronary artery disease. American Heart Journal, 105(6), 889-895.

Herbert, P. N., Bernier, D. N., Cullinane, E. M., Edelstein, L., Kantor, M. A., & Thompson, P. D. (1984). High density lipoprotein metabolism in runners and sedentary men. Journal of the American Medical Association, 252, 1034-1037.

Hespel, P., Lijnen, P., Fagard, R., Van Hoof, R., Rosseneu, M., & Amery, A. (1988). Changes in plasma lipids and apoproteins associated with physical training in middle-aged sedentary men. American Heart Journal, 115(4), 786-792.

Hicks, A. L., MacDougall, J. D., & Muckle, T. J. (1987). Acute changes in HDL cholesterol with exercise of different intensities. Journal of Applied Physiology, 63(5), 1956-1960.

Hickson, R. C., Rosenkoetter, M. A., & Brown, M. M. (1980). Strength training effects on aerobic power and short-term endurance. Medicine and Science in Sports and Exercise, 12, 336-339.

Hill, D.W. (1988). Equations to calculate the effects of plasma volume change on blood and plasma concentrations. Research Quarterly for Exercise and Sport, 59(2), 169-172.

Hooper, P. L., & Scallen, T. J. (1984). Modulation of high density lipoprotein: The importance of protein phosphorylation/dephosphorylation. American Heart Journal, 108, 1393-1398.

Hurley, B. F., Hagberg, J. M., & Goldberg, A. P. (1988). Resistive training can reduce coronary risk factors without altering VO_2 max or percent body fat. Medicine and Science in Sports and Exercise, 20, 150-154.

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

Hurley, B. F., Hagberg, J. M., Seals, D. R., Florman, R., & Goldberg, A. P. (1984). Hepatic triglyceride lipase modulates high density lipoprotein cholesterol levels in weightlifters and runners. Abstract. Clinical Research, 32, 398A.

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

Hurley, B. F., Nemeth, P. M., Martin III, W. H., Hagberg, J. M., Dalsky, G. P., & Holloszy, J. O. (1986). Muscle triglyceride utilization during exercise: Effect of training. Journal of Applied Physiology, 60, 562-567.

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

Hurter, R., Swale, J., Peyman, M. A., & Barnett, C. W. H. (1972). Some immediate and long-term effects of exercise on the plasma lipids. Lancet, 2, 671-675.

Huttunen, J. K., Lansimies, E., Voutilainen, E., Ehnholm, C., Hietanen, E., Penttila, I., Sitonen, O., & Rauramaa, R. (1979). Effect of moderate physical exercise on serum lipoproteins. Circulation, 60, 1220-1229.

Illingworth, D. R. & Connor, W. E. (1985). Hyperlipidemia and coronary heart disease. In W. E. Connor, & J. D. Bristo (Eds.), Coronary heart disease: Prevention, complications, and treatment (pp. 21-42). Philadelphia: J.B. Lippincott.

Jacobs, I., Lithell, H., & Karlsson, J. (1982). Dietary effects on glycogen and lipoprotein lipase activity in skeletal muscle of men. Acta Physiologia Scandinavia, 104, 117-121.

Johnson, C. C., Stone, M. H., Byrd, R. J., & Lopez, A. (1983). The response of serum lipids and plasma androgens to weight training exercise in sedentary males. Journal of Sports Medicine, 23, 39-44.

Kannel, W. B. (1983). High density lipoproteins: Epidemiologic profile and risks of coronary artery disease. American Journal of Cardiology, 52, 9B-12B.

Kantor, M. A., Cullinane, E. M., Herbert, P. N., & Thompson, P. D. (1984). Acute increase in lipoprotein lipase following prolonged exercise. Metabolism, 33, 454-457.

Katch, F. I., & McArdle, W. D. (1988). Nutrion, weight control, and exercise. Philadelphia: Lea & Febiger.

Kelemen, M. H. (1989). Resistive training safety and assessment guidelines for cardiac and coronary prone patients. Medicine and Science in Sports and Exercise, 21(6), 675-677.

Keys, A., Kimuira, N., Kusukawa, A., Bronte-Stewart, B., Larsen, N., & Keys, M. H. (1958). Lessons from serum cholesterol studies in Japan, Hawaii, and Los Angeles. Annals of Internal Medicine, 48, 83-94.

Keys, A., Taylor, H. L., Blackburn, H., Brozek, J., Anderson, J. T., & Simonson, E. (1963). Coronary heart disease among Minnesota business and professional men followed fifteen years. Circulation, 28, 381-395.

Kiens, B., Lithell, H., Mikines, K. J., & Richter, E. A. (1989). Effects of insulin and exercise on muscle lipoprotein lipase activity in man and its realtion to insulin action. Journal of Clinical Investigation, 84, 1124-1129.

Knowlton, R. G., Hetzler, R. K., Kaminsky, L. A., & Morrison, J. J. (1987). Plasma volume changes and cardiovascular responses associated with weight lifting. Medicine and Science in Sports and Exercise, 19, 464-468.

Kokkinos, P., Hurley, B., Smutok, C., Farmer, C., Reece, C., Shulman, R., & Goldberg, A. (1989). Lipoprotein-lipid profiles and post-heparin lipase activities are unaltered from strength training. Medicine and Science in Sports and Exercise, 21(Suppl.), s116.

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

Kotlar, T. J., & Borensztajn, J. (1977). Oscillatory changes in muscle lipoprotein lipase activity of fed and starved rats. American Journal of Physiology, 233, E316-E319.

Lakatta, E. G., Goldberg, A. P., Fleg, J. L., Fortney, S. M., & Drinkwater, D.T. (1987). Reduced cardiovasular and metabolic reserve in older persons: Disuse, disease or aging in nutrition and aging? In D. A. Lipschitz & R. Chernoff (Eds.), Nutrition and aging II: Health promotion and disease prevention in the elderly (pp. 75-78). New York: Raven Press.

Lennon, D. L., Stratman, F. W., Shrago, E., Nagle, F. J., Hanson, P. G., Madden, M., & Spennetta, T. (1983). Total cholesterol and HDL cholesterol changes during acute, moderate-intensity exercise in men and women. Metabolism, 32(3), 244-249.

Levy, R. L. (1985). Primary prevention of coronary heart disease by lowering lipids: Results and implications. American Heart Journal, 110(5), 1116-1122.

Linder, C. W., DuRant, R. H., & Mahoney, O. M. (1983). The effect of physical conditioning on serum lipids and lipoproteins in white male adolescents. Medicine and Science in Sports and Exercise, 15(3), 232-236.

Lipid Research Clinics Program Epidemiology Committee. (1979). Plasma lipid distributions in selected North American population. The lipid research clinics program prevalence study. Circulation, 60, 427-439.

Lipson, L. C., Bonow, R. O., Schaefer, E. J., Brewer, H. B., & Lindgren, F. T. (1980). Effect of exercise conditioning on plasma HDL and other lipoproteins. Atherosclerosis, 37, 529.

Lithell, H., Cedermark, M., Froberg, J., Tesch, P., & Karlsson, J. (1981). Increase of lipoprotein lipase activity in skeletal muscle during heavy exercise. Relation to epinephrine. Metabolism, 30, 1130-1134.

Lithell, H. Jacobs, I., Vessby, B., Hellsing, & Karsson, J. (1982). Decrease of lipoprotein lipase activity in skeletal muscle in man during a short-trem carbohydrate-rich dietary regime. With special reference to HDL-cholesterol, apolipoprotein and insulin concentrations. Metabolism, 31, 994-998.

Lopez, A., Vial, R., Ballart, L., & Arroyave, G. (1974). Effect of exercise and physical fitness on serum lipids and lipoproteins. Atherosclerosis, 20, 1-9.

Lundvall, J. (1972). Tissue hyperosmolarity as a mediator of vasodilation and transcapillary fluid flux in exercising skeletal muscle. Acta Physiologica Scandinavica, 379(Suppl.), 1-142.

Macek, M., Bell, D., Rutenfranz, J., Vavra, J., Masopust, J., Jeidhart, B., & Schmidt, K. H. (1989). A comparison of coronary risk factors in groups of trained and untrained adolescents. European Journal of Applied Physiology, 58, 577-582.

Mahley, R.W. (1983). Development of accelerated atherosclerosis. Concepts derived from cell biology and animal model studies. Archives of Pathology and Laboratory Medicine, 107, 393-399.

Marniemi, J., Dahlstrom, S., Kvist, M., Seppanen, A., & Hietanen, E. (1982). Dependence of serum lipid and lecithin: cholesterol acyltransferase levels on physical training of young men. European Journal of Applied Physiology, 49, 25-35.

Martin, R. P., Haskell, W. L., & Wood, P. D. (1977). Blood chemistry and lipid profiles of elite distance runners. New York Academy of Science, 301, 346-360.

Maxwell, S. E. (1980). Pairwise multiple comparisons in repeated measures designs. Journal of Educational Statistics, 5(3), 269-287.

McGill, Jr H. C. (1988). The pathogenesis of atherosclerosis. Clinical Chemistry, 34(8B), B33-B39.

McNamara, J. R., & Schaefer, E. J. (1987). Automated enzymatic standardized lipid analyses for plasma and lipoprotein fractions. Clinica Chimica Acta, 166, 1-8.

Miles, D. S., Sawka, M. N., Glasen, R. M., & Petrofsky, J. S. (1983). Plasma volume shifts during progressive arm and leg exercise. Journal of Applied Physiology, 54, 491-495.

Miller, J. P., & Gotto, A. M. (1982). The plasma lipoproteins their formation and metabolism. Comprehensive Biochemistry, 19B, 419-506.

Miller, N.E. (1980). HDL cholesterol, tissue cholesterol and coronary atherosclerosis: Epidemiological correlations. In A. M. Gotto & B. Allen (Eds.), Atherosclerosis (pp. 500-503). New York: Springer-Verlag.

Miller, N. E., Nestel, P. J., & Clifton-Bligh, P., (1976). Relation between plasma lipoprotein cholesterol concentrations and the pool size and metabolism of cholesterol in man. Atherosclerosis, 23, 535-547.

Morgan, D. W., Cruise, R. J., Giradin, B. W., Lutz-Schneider, V., Morgan, D. H., & Qi, W. M. (1986). HDL-C concentrations in weight trained, endurance trained, and sedentary females. Physician and Sportsmedicine, 14(3), 166-181.

Mullins, C. B., & Blomqvist, G. (1973). Isometric exercise and the cardiac patient. Texas Medicine, 69, 53-58.

Myhre, K., Mjös, O. D., Björsvik, G., & Strömme, S. B. (1981). Relationship of HDL cholesterol concentration to the duration and intensity of endurance training. Scandinavia Journal of Clinical Laboratory Investigations, 41(3), 303-309.

Naughton, J., & Balke, B. (1964). Physical working capacity in medical personnel and the response of serum cholesterol to acute exercise and to training. American Journal of Medicine Science, 247, 286-292.

Natio, H. K., & Galen, R. S. (1983). Apolipoproteins: Biochemistry, physiology, and pathophysiology, in American society for clinical pathology. Clinical Chemistry, 29, 1-13.

Nikkilä, E. A., Kuusi, T., & Taskinen, M. (1982). Role of lipoprotein lipase and hepatic endothelial lipase in the metabolism of high density lipoproteins: A novel concept on cholesterol transport in HDL cycle. In L. A. Carlson & B. Pernow (Eds.), Metabolic risk factors in ischemic cardiovascular disease (pp. 205-216). New York: Raven Press.

Noma, A., Yokosuka, T., & Kitamura, K., (1983). Plasma lipids and apolipoproteins as discriminators for presence and severity of angiographically defined coronary artery disease. Atherosclerosis, 49, 1-7.

O'Shea, J. P. (1976). Scientific principles and methods of strength fitness. Menlo Park: Addison-Wesley Publishing.

O'Shea, J. P. (1985). The parallel squat. National Strength and Conditioning Association Journal, 7, 4-6, 78.

O'Shea, J. P. (1986). The push press: An alternative to the bench press. National Strength and Conditioning Association Journal, 8(5), 28-31.

O'Shea, J. P., Simmons, J., & O'Connor, B. (1989). Weight training today. St. Paul: West Publishing.

Paffenbarger, R. S., & Hale, W. E. (1975). Work capacity and coronary heart mortality. New England Journal of Medicine, 292, 546-550.

Paul, O., Lepper, M. H., Phelan, W. H., Dupertuis, G. W., MacMilian, A., McKean, H., & Park, H. (1963). A longitudinal study of coronary heart disease. Circulation, 28, 20-31.

Peltonen, P., Marniemi, J., Heitanen, E., Vuori, I., & Ehnholm, C. (1981). Changes in serum lipids, lipoprotein and heparin releasable lipolytic enzymes during moderate physical training in men: A longitudinal study. Metabolism, 30, 518.

Penny, G. D., Shaver, L. G., Carlton, J., & Kendall, D. W. (1982). Comparison of serum HDL-C and HDL-total cholesterol ratio in middle age active and inactive males. Journal of Sports Medicine, 22, 432-439.

Raz, I., Rosenblit, H., & Kark, J. D. (1988). Effect of moderate exercise on serum lipids in young men with low HDL cholesterol. Arteriosclerosis, 8, 245-251.

Rifkind, B. M. (1984). Lipid research clinics coronary primary prevention trial: Results and implications. American Journal of Cardiology, 54, 30c-34c.

Ross, R. (1986). The pathogenesis of atherosclerosis: An update. New England Journal of Medicine, 314(8), 488-500.

Ruys, T., Shaikh, M., Nordestgaard, B. G., Sturgess, I., Watts, G. F., & Lewis, B. (1989). Effects of exercise and fat ingestion on high density lipoprotein production by peripheral tissues. Lancet, 8672, 1119-1122.

Sady, S. P., Thompson P. D., Cullinane, E. M., Kantor, M. A., Domagala, E., & Herbert, P. N. (1986). Prolonged exercise augments plasma triglyceride clearance. Journal of the American Medical Association, 256(18), 2552-2555.

Sannerstedt, R., Sanbar, S. S., & Conway, J. (1970). Metabolic effects of exercise in patients with Type IV hyperlipoproteinemia. American Journal of Cardiology, 25, 642-648.

Schnabel, A., & Kindermann, W. (1982). Effect of maximal oxygen uptake and different forms of physical training on serum lipoproteins. European Journal of Applied Physiology, 48, 263-277.

Schwartz, C. J., Kelley, J. L., Nerem, R. M., Sprague, E. A., Rozek, M. M., Valente, A. J., Edwards, E. H., Prasad, A. R. S., Kerbacher, J. J, & Logan, S. A. (1989). Pathophysiology of the atherogenic process. American Journal of Cardiology, 64, 23G-30G.

Scow, R. O., Blanchette-Mackie, E. J., & Smith, L. C. (1980). Transport of lipid across capillary endothelium. Federation Proceedings, 39, 2610.

Sedgwick, A. W., Brotherhood, J. R., Harris-Davidson, A., Taplin, R. E., & Thomas, D. W. (1980). Long term effects of physical training programme on risk factors for coronary heart disease in otherwise sedentary men. British Medical Journal, 281(6232), 7-10.

Shephard, R. J. (1986). Exercise on coronary heart disease. Sports Medicine, 3, 26-49.

Sherwin, R. (1988). Cardiovascular system: Blood lipids and lipoproteins. In D. M. Paige (Ed.), Clinical nutrition (pp. 205-209). St. Louis: C.V. Mosby.

Sink, K. R., Thomas, T. R., Araujo, J., & Hill, S. F. (1989). Fat energy use and plasma lipid changes associated with exercise intencity and temperature. European Journal of Applied Physiology, 58, 508-513.

Skinner, E. R., Black, D., & Maughan, J. (1985). Variability in the response of different male subjects to the effect of marathon running on the increase in plasma high density lipiprotein. European Journal of Applied Physiology, 54, 488-493.

Slotte, J. P., Chait, A., & Bierman, E. L. (1988). Cholesterol accumulation in aortic smooth muscle cells exposed to low density lipoproteins: Contribution of free cholesterol transfer. Arteriosclerosis, 8, 750-758.

Stein, E. A. (1986). Lipids, lipoproteins, and apolipoproteins. In N. W. Tietz (Ed.), Textbook of clinical chemistry (pp. 879-885). Philadelphia: W.B. Saunders.

Stewart, K. J. (1989). Resistive training effects on strength and cardiovascular endurance in cardiac and coronary prone patients. Medicine and Science in Sports and Exercise, 21(6), 678-682.

Stiggins, C., & Allsen, P. (1989). Exercise methods notebook #43 Seated rowing. National Strength & Conditioning Association Journal. 11(4), 82.

Stone, M., Blessing, D., Byrd, R., Tew, J., & Boatwright, D. (1982). Physiological effects of a short term resistive training program on middle-aged untrained men. National Strength and Conditioning Association Journal, 4, 16-20.

Taylor, E. J. (1988). Dorland's illustrated medical dictionary (27th ed.). Philadelphia: W. B. Saunders.

Thomas, C. L. (1985). Taber's cyclopedic medical dictionary. Philadelphia: F.A. Davis.

Thompson, P. D., Cullinane, E., Henderson, L. O., & Herbert, P. N. (1980). Acute effects of prolonged exercise on serum lipids. Metabolism, 29, 662-665.

Thompson, P. D., Cullinane, E., Sady, S., Flynn, M., Bernier, D., Kantor, M., Saritelli, A., & Herbert, P. (1988). Modest changes in HDL concentration and metabolism with prolonged exercise training. Circulation, 78, 25-34.

Tsopanakis, C., Kotsarellis, D., & Tsopanakis, A. (1988). Plasma lecithin:cholesterol acyltransferase activity in elite athletes from selected sports. European Journal of Applied Physiology, 58, 262-265.

Tyroler, H. A. (1984). Cholesterol and cardiovascular disease. American Journal of Cardiology, 54, 14c-19c.

van Beaumont, W., Greenleaf, J. E., Juhos, L. (1972). Disproportional changes in hematocrit, plasma volume, and proteins during exercise and bed rest. Journal of Applied Physiology, 33, 55-61.

van Beaumont, W., Strand, J. C., Petrofsky, J. S., Hipskind, S. G., & Greenleaf, J. E. (1973). Changes in total plasma content of electrolytes and proteins with maximal exercise. Journal of Applied Physiology, 34(1), 102-106.

Vander, L. B., Franklin, B. A., Wrisley, D., & Rubenfire, M. (1986). Acute cardiovascular response to circuit weight training in patients with cardiac disease. Annals of Sports Medicine, 2, 165-169.

Vessby, B., Selinus, I., & Lithell, H. (1985). Serum lipoprotein and lipoprotein lipase in overweight Type II diabetics during and after supplemental fasting. Atherosclerosis, 5, 93-100.

Voutilainen, E., & Hietanen, E. (1982a). Lipids as a Risk Factor of Coronary Heart Disease. In E. Hietanen (Ed.), Regulation of serum lipids by physical exercise (pp. 19-33). Florida: CRC Press.

Voutilainen, E., & Hietanen, E. (1982b). Synthesis and catabolism. In E. Hietanen (Ed.), Regulation of serum lipids by physical exercise (pp. 1-9). Florida: CRC Press.

Williams, M. H. (1990). Lifetime fitness and wellness (2nd Ed.). Dubuque: Wm. C. Brown Publishers.

Williams, P. T., Krauss, R. M., Wood, P. D., Lindgren, F. T., Giotas, C., & Vranizan, K. M. (1986). Lipoprotein subfractions of runners and sedentary men. Metabolism, 35, 45-52.

Williams, P. T., Wood, P. D., Krauss, R. M., Haskell, W. L., Vranizan, K. M., Blair, S. N., Terry, R., & Farquhar, J. W. (1983). Does weight loss cause the exercise-induced increase in plasma high-density lipoproteins? Atherosclerosis, 47, 173-185.

Williams, R. S. (1985). Role of receptor mechanism in the adaptive response to habitual exercise. American Journal of Cardiology, 55, 68D-73D.

Williford, H. N., Blessing, D. L., Barksdale, J. M., & Smith, F. H. (1988). The effects of aerobic dance training on serum lipids, lipoproteins and cardiopulmonary function. Journal of Sports Medicine and Physical Fitness, 28(2), 151-157.

Wilson, P. W., Abbott, R. D., & Castelli, W. P. (1988). High density lipoprotein cholesterol and mortality: The Framingham Heart Study. Arteriosclerosis, 8, 737-741.

Wirth, A., Diehm, C., Kohlmeier, M., Heuck, C. C., & Vogel, I. (1983). Effect of prolonged exercise on serum lipids and lipoproteins. Metabolism, 32, 669-672.

Wirth, A., Holm, G., Lindstedt, G., Lundberg, P. A., & Björntorp, P. (1981). Thyroid hormones and lipolysis in physically trained rats. Metabolism, 30, 237-241.

Wood, P. D., & Haskell, W. L. (1979). The effect of exercise on plasma high density lipoproteins. Lipids, 14, 417-427.

Wood, P. D., Haskell, W. L., Stern, M. P., Lewis, S., & Farquhar, J. W. (1976). The distribution of plasma lipoproteins in middle-aged male runners. Metabolism, 25(11), 1249-1257.

Wood, P. D., & Stefanick, M. L. (1990). Exercise, fitness, and atherosclerosis. In C. Bouchard, R. J. Shephard, T. Stephens, J. R. Sutton, & B.D. McPherson (Eds.), Exercise, fitness, and health (pp. 409-424). Champaign: Human Kinetics Book.

Appendices

APPENDIX A

Personal Data

Subjects	Height (cm)	Weight (kg)	Age (yrs)	BMI*
1	180.3	70.8	24	21.7
2	185.4	106.6	18	31.0
3	180.3	72.1	19	22.1
4	172.7	70.3	25	23.5
5	185.4	81.6	20	23.7
6	182.9	86.2	19	25.7
7	182.9	81.2	21	24.2
8	177.8	86.2	20	27.2
9	175.3	70.3	18	22.8
10	185.4	88.5	19	25.7
11	182.9	81.6	22	24.3
12	180.4	72,6	19	22.3
13	180.3	97.9	25	30.1
14	177.8	68.9	18	21.7
15	185.4	82.6	18	24.0
16	180.2	92.9	18	28.6
17	185.4	77.1	19	22.4

BMI= Body Mass Index

APPENDIX B

Informed Consent Form

Oregon State University

Subject's Name (Please Print):_____

Current Address: _____

_____

Phone Number: _____

Project Title: "The acute effects of moderate intensity circuit weight training on lipid-lipoprotein profiles."

The purpose of this study is to determine the acute response of triglyceride, HDL-C, LDL-C, total cholesterol, and the ratio of total cholesterol:HDL-C to a single period of moderate intensity circuit weight training exercise.

Project Director: Dr. John P. O'Shea (X 7-3719)

Researcher's Name: Young-Soo Lee (X 7-3221)

Testing Procedures:

Circuit Weight Training Exercise and Blood Sampling

Subjects will report to the weight training room in Langton Hall at the preset time. Subjects will be required to fast overnight (at least 12 hours) prior to reporting to the weight training room.

Subjects will repeat a four station weight training circuit three times utilizing the following lifts: (1) bench press, (2) parallel squat, (3) seated row, and (4) leg extension.

Subjects will exercise for 1 min at each station using a resistance determined by their individual 3 repetition maximum (3-RM) values. The resistance to be used are, 50% of 3-RM for bench press, 60% of 3-RM for parallel squat, 40% of 3-RM for seated row, and 40% of 3-RM for leg extension. A 30-second rest will be given between exercise stations. A 2 min rest will be used to obtain heart rate and blood samples between the 1st and 2nd circuit. A 2 min rest will be given between the 2nd and 3rd circuit.

Blood samples will be drawn before CWT exercise, after the first circuit of the four CWT exercise stations,

immediately after the 3rd circuit of the CWT exercise stations, and after 15 min of the third circuit of the CWT exercise. Blood samples are to be drawn from the antecubital vein, using a Vacutainer 100 x 13 mm sterile evacuated blood collection tube with EDTA. Pressure dressing using sterile gauze and Elastoplast brand tape (Johnson & Johnson) will be applied following blood sampling.

Risks and Discomforts: Certain hazards and discomforts are associated with the procedures previously described. They include:
1. A minimal amount of discomfort associated with blood drawing. If fainting occurs, testing will be discontinued.
2. The exertion for the submaximal contractions in circuit weight training exercise will be intense and momentarily exhausting.

Benefits: Each participant will learn (a) more about his body's response to increasing exercise stress and (b) his plasma lipoprotein level, which is one of the important indicators of coronary heart disease.

Confidentiality: Data collected from testing and the personal questionnaire of this project are strictly for research purposes only and kept confidential. No names will be used in the reports.

Freedom of Consent: Participation in the study is voluntary. Subjects are free to withdraw consent and discontinue at any time without penalty. The researcher will answer questions about the testing procedures or any concerns that subjects may have.

Persons at increased risk for Hepatitis B or HIV (AIDS) infections should not donate blood nor body fluids.

I have read this form and understand the testing procedures and agree to participate in this study under the direction of the researcher named above.

---------- ----------

Date Signature of Subject

APPENDIX C

Medical History and Health Form

HUMAN PERFORMANCE LABORATORY OREGON STATE UNIVERSITY

SELF-ADMINISTERED PRE-EXERCISE MEDICAL HISTORY & HEALTH FORM

Name _____ **Age**____ **Ht.**_____ **Wt.**_____ **Date**_____

Occupation _____ **Phone: Home** _____ **Time** _____

Work _____

(Check if yes)

PAST HISTORY
(Have you ever had?)

Rheumatic Fever	( )
Heart Murmur	( )
High Blood Pressure	( )
Any Heart Trouble	( )
Disease of Arteries	( )
Varicose Veins	( )
Lung Disease	( )
Operations	( )
Injuries to back, etc.	( )
Epilepsy	( )
Musculoskeletal problem	( )

FAMILY HISTORY
(Have any of your relatives had?)

Heart Attacks	( )
High Blood Pressure	( )
Too much cholesterol	( )
Diabetes	( )
Congenital Heart Diseases	( )
Heart Operations	( )
Other	( )

Date of Last Medical Exam_____

Physician_____

PRESENT SYMPTOMS REVIEW
(Have you recently had?)

Chest pain	( )
Shortness of Breath	( )
Heart Palpitations	( )
Cough on Exertion	( )
Coughing Blood	( )
Back Pain	( )
Swollen, Stiff or Painful Joints	( )
Do you awaken at night to urinate	( )

Other _____

Are you taking any medication?

_____

_____

HEALTH AND FITNESS HABITS

1. Smoking

	Yes	No	
Do you smoke	( )	( )	
Cigarettes	( )	( )	How many?____How many years?___
Cigar	( )	( )	How many?____How many years?___
Pipe	( )	( )	How many times a day?_____

How old were you when you started? _____

In case you have stopped, when did you? _____
Why?_____

2. **Diet**

What is your weight now?_____1 year ago?____At age 21?___
Are you presently on a diet?____If so, has the diet been suggested by your physician?_____Describe the diet:_____
_____
_____
Where are your meals eaten?_____
Who prepares your meals?_____
How often do you eat in restaurants?_____
Does your eating pattern vary on weekends?_____
_____
Do you consume alcohol on a regular basis?___If so, in what form is the drink?_____How much would you consume in an average day?_____
Do you take any protein, vitamin, or mineral supplements?
_____Amount_____
Kind_____

3. **Exercise**

Do you engage in sports? _____
What?_____How often?_____
How far do you think you walk each day?_____
Is your occupation: Sedentary ( ) Active ( )
Inactive ( ) Heavy work ( )
Do you have discomfort, shortness of breath, or pain with moderate exercise?_____
Specify_____
Did you participate in youth sports?_____Age at start____
Specify teams and positions_____
_____

How would you rate the amount of physical activity you perform during your leisure time?
very little/little/moderate/active/very active

Are you presently performing any standard physical fitness program? Describe (intensity, frequency, duration)_____
_____

How physically fit do you feel at the present?
very little/little/moderate/active/very active

Do you have any exercise equipment or device at home?
Yes ( ) No ( )
If yes, please specify_____

Specify other fitness and recreation activities you participate in _____
_____

APPENDIX D

Three Repetition Maximum of Weight Lifts

Subjects	BP (lbs)	PS (lbs)	SR (lbs)	LE (lbs)
1	135	195	160	100
2	220	285	170	200
3	135	165	160	140
4	160	190	130	140
5	165	165	140	170
6	185	245	160	170
7	205	295	190	180
8	205	305	140	150
9	185	185	120	150
10	225	270	190	150
11	225	225	180	150
12	185	240	160	150
13	235	350	200	190
14	135	225	150	180
15	175	225	130	180
16	185	235	160	160
17	165	245	150	170

BP= Bench Press
PS= Parallel Squat
SR= Seated Row
LE= Leg Extension

APPENDIX E

Hematocrit

Subjects	Pre (%)	1st Cir. (%)	2nd Cir. (%)	Post (%)
1	49.0	52.8	52.2	48.3
2	42.9	46.2	45.4	42.4
3	42.2	50.2	49.0	45.3
4	45.8	53.7	52.6	49.1
5	46.1	50.6	50.0	47.1
6	46.1	49.9	50.1	47.1
7	47.1	50.5	51.8	47.9
8	48.5	50.8	50.9	47.8
9	43.9	46.3	45.6	42 5
10	42.3	45.1	44.6	42.1
11	43.3	48.6	48.0	44.0
12	47.7	51.8	51.3	47.7
13	46.3	53.2	51.6	49.8
14	47.7	52.7	52.2	46.6
15	47.3	52.3	52.0	46.0
16	47.8	50.0	49.3	46.6
17	44.5	51.8	49.5	46.5

Pre= Measured before CWT
1st Cir= Measured after the completion of 1st circuit
3rd Cir= Measured after the completion of 3rd circuit
Post= Measured 15 min post CWT

APPENDIX F

Hemoglobin

Subjects	Pre (g/dl)	1st Cir. (g/dl)	2nd Cir. (g/dl)	Post (g/dl)
1	16.4	17.3	17.2	16.3
2	14.5	15.6	15.3	14.8
3	15.1	16.2	16.1	15.2
4	15.1	17.1	16.9	16.0
5	15.4	16.6	16.4	15.7
6	15.6	16.4	16.2	15.6
7	15.6	16.5	16.7	16.1
8	15.8	16.5	16.4	15.7
9	14.5	15.1	15.0	14.4
10	14.6	15.2	15.2	14.4
11	14.0	15.0	15.0	14.3
12	16.1	17.0	17.0	16.1
13	15.6	16.8	16.6	16.6
14	16.7	17.7	17.5	16.2
15	15.7	16.7	16.3	15.6
16	15.9	16.2	15.9	15.6
17	15.1	16.3	15.9	15.3

Pre= Measured before CWT
1st Cir= Measured after the completion of 1st circuit
3rd Cir= Measured after the completion of 3rd circuit
Post= Measured 15 min post CWT

APPENDIX G

Triglycerides

Subjects	Pre (mg/dl)	1st Cir. (mg/dl)		2nd Cir. (mg/dl)		Post (mg/dl)	
1	55	71	(64.4)+	75	(69.3)	71	(71.9)
2	93	87	(75.1)	82	(73.3)	76	(74.7)
3	108	123	(106.1)	119	(105.1)	109	(108.1)
4	101	124	(95.7)	135	(110.5)	132	(120.5)
5	57	59	(50.2)	55	(47.6)	44	(42.0)
6	50	54	(48.3)	56	(50.8)	49	(48.2)
7	65	77	(69.6)	82	(72.2)	77	(74.1)
8	55	60	(55.4)	62	(57.7)	53	(53.9)
9	54	62	(57.7)	53	(49.7)	45	(46.4)
10	55	56	(51.4)	50	(45.8)	47	(47.9)
11	69	72	(61.1)	71	(60.8)	65	(62.8)
12	128	127	(111.0)	118	(103.1)	104	(104.0)
13	137	157	(129.8)	166	(144.9)	156	(139.3)
14	112	127	(110.6)	124	(110.1)	108	(113.1)
15	116	136	(118.6)	138	(124.5)	133	(135.9)
16	77	89	(84.8)	97	(95.2)	91	(93.7)
17	52	67	(56.3)	72	(65.0)	75	(72.7)

+ Corrected value for plasma volume change

Pre= Measured before CWT
1st Cir= Measured after the completion of 1st circuit
3rd Cir= Measured after the completion of 3rd circuit
Post= Measured 15 min post CWT

APPENDIX H

Total Cholesterol

Subjects	Pre (mg/dl)	1st Cir. (mg/dl)	3rd Cir. (mg/dl)	Post (mg/dl)
1	164	183 (163.3)*	181 (164.1)	162 (164.7)
2	139	152 (134.2)	149 (136.0)	141 (139.1)
3	174	195 (167.8)	194 (171.7)	173 (171.5)
4	159	193 (148.5)	190 (151.4)	171 (153.0)
5	149	171 (148.0)	166 (146.7)	154 (148.8)
6	151	166 (148.9)	162 (146.4)	148 (145.7)
7	147	163 (146.2)	166 (143.8)	152 (145.4)
8	134	150 (139.0)	151 (140.6)	138 (140.2)
9	154	172 (159.9)	172 (162.7)	157 (161.0)
10	155	170 (157.1)	167 (155.3)	157 (159.5)
11	235	268 (230.9)	263 (228.4)	245 (237.5)
12	146	156 (137.7)	154 (137.0)	143 (143.0)
13	185	211 (174.3)	209 (180.5)	198 (175.4)
14	146	162 (140.7)	160 (141.9)	143 (149.6)
15	189	220 (191.7)	213 (191.0)	195 (199.7)
16	131	139 (131.9)	136 (132.9)	128 (132.7)
17	142	163 (133.8)	154 (134.9)	141 (134.8)

* Corrected value for plasma volume change

Pre= Measured before CWT
1st Cir= Measured after the completion of 1st circuit
3rd Cir= Measured after the completion of 3rd circuit
Post= Measured 15 min post CWT

APPENDIX I

HDL-Cholesterol

Subjects	Pre (mg/dl)	1st Cir. (mg/dl)	3rd Cir. (mg/dl)	Post (mg/dl)
1	52	46 (39.7)[+]	51 (45.6)	59 (59.8)
2	59	56 (48.4)	44 (38.4)	49 (48.2)
3	48	44 (36.5)	39 (32.8)	44 (43.6)
4	44	41 (28.7)	50 (39.3)	56 (51.0)
5	57	51 (42.2)	42 (34.6)	51 (49.0)
6	51	55 (49.2)	56 (50.7)	50 (49.2)
7	42	47 (42.2)	48 (41.6)	44 (42.1)
8	50	57 (52.9)	59 (55.1)	53 (53.8)
9	65	72 (66.9)	72 (68.1)	67 (68.7)
10	45	49 (45.2)	49 (45.6)	46 (46.7)
11	56	64 (55.1)	64 (55.7)	59 (57.2)
12	36	39 (34.5)	39 (34.8)	36 (36.0)
13	35	39 (32.0)	40 (34.6)	39 (34.7)
14	30	33 (28.6)	33 (29.2)	30 (31.3)
15	46	55 (48.1)	53 (47.6)	46 (47.1)
16	39	42 (39.9)	41 (40.1)	39 (40.4)
17	44	51 (41.9)	49 (43.1)	46 (44.1)

[+] Collected values for plasma volume change

Pre= Measured before CWT
1st Cir= Measured after the completion of 1st circuit
3rd Cir= Measured after the completion of 3rd circuit
Post= Measured 15 min post CWT

APPENDIX J

LDL-Cholesterol

Subjects	Pre (mg/dl)	1st Cir. (mg/dl)		3rd Cir. (mg/dl)		Post (mg/dl)	
1	101.0	122.8	(110.6)+	115.0	(104.6)	88.8	(90.4)
2	61.4	78.6	(70.7)	88.6	(82.8)	76.8	(75.9)
3	104.4	126.4	(110.0)	131.2	(117.8)	107.2	(106.3)
4	94.8	127.2	(100.6)	113.0	(90.0)	88.6	(77.8)
5	80.6	108.2	(95.8)	113.1	(102.5)	94.2	(91.4)
6	90.0	100.2	(90.0)	94.8	(85.5)	88.2	(86.8)
7	92.0	100.6	(90.1)	101.6	(87.7)	92.6	(88.5)
8	73.0	81.0	(75.0)	79.6	(73.9)	74.4	(75.6)
9	78.2	87.6	(81.5)	89.4	(84.7)	81.0	(83.1)
10	99.0	109.8	(101.5)	106.0	(100.5)	101.6	(103.2)
11	165.2	189.6	(163.5)	184.8	(160.4)	173.0	(167.7)
12	84.4	91.6	(81.0)	91.4	(81.6)	86.2	(86.2)
13	122.6	140.6	(116.3)	135.8	(116.9)	127.8	(112.8)
14	93.6	103.6	(89.9)	102.2	(90.6)	91.4	(95.6)
15	119.8	137.8	(119.9)	132.4	(118.4)	120.4	(125.4)
16	76.6	79.2	(75.1)	75.6	(73.8)	70.8	(73.5)
17	87.6	98.6	(80.6)	90.6	(78.8)	80.0	(76.2)

+ Collected values for plasma volume change

Pre= Measured before CWT
1st Cir= Measured after the completion of 1st circuit
3rd Cir= Measured after the completion of 3rd circuit
Post= Measured 15 min post CWT

APPENDIX K

Ratio of Total Cholesterol to HDL-Cholesterol

Subjects	Pre	1st Cir.	3rd Cir.	Post
1	3.2	4.0 (4.11)[+]	3.5 (3.60)	2.7 (2.75)
2	2.4	2.7 (2.77)	3.4 (3.53)	2.9 (2.89)
3	3.6	4.4 (4.60)	5.0 (5.23)	3.9 (3.93)
4	3.6	4.7 (5.18)	3.8 (3.85)	3.1 (2.99)
5	2.6	3.4 (3.51)	4.0 (4.24)	3.0 (3.04)
6	3.0	3.0 (3.02)	2.9 (2.89)	3.0 (2.96)
7	3.5	3.5 (3.46)	3.5 (3.45)	3.5 (3.45)
8	2.7	2.6 (2.63)	2.6 (2.55)	2.6 (2.60)
9	2.4	2.4 (2.39)	2.4 (2.39)	2.3 (2.34)
10	3.4	3.5 (3.47)	3.4 (3.41)	3.4 (3.41)
11	4.2	4.2 (4.19)	4.1 (4.10)	4.2 (4.15)
12	4.1	4.0 (3.99)	3.9 (3.94)	4.0 (3.97)
13	5.3	5.4 (5.44)	5.2 (5.22)	5.1 (5.05)
14	4.9	4.9 (4.92)	4.8 (4.85)	4.8 (4.77)
15	4.1	4.0 (3.98)	4.0 (4.01)	4.1 (4.24)
16	3.4	3.3 (3.31)	3.3 (3.32)	3.3 (3.28)
17	3.2	3.2 (3.20)	3.1 (3.13)	3.1 (3.06)

[+] Collected values for plasma volume change

Pre= Measured before CWT
1st Cir= Measured after the completion of 1st circuit
3rd Cir= Measured after the completion of 3rd circuit
Post= Measured 15 min post CWT